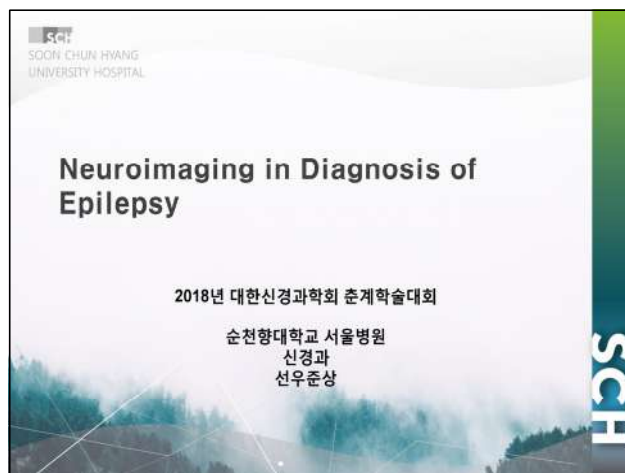


# Neuroimaging in Diagnosis of Epilepsy



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### Introduction

- The etiologies of epilepsies are varied and multifactorial in most cases. Neuroimaging study, in particular **MRI**, are crucial for determining the possible etiology of epilepsy
- CT has low sensitivity for detecting small cortical lesions, orbitofrontal and medial temporal lesions (low as approximately 30% in focal epilepsies)

뇌전증 역학조사 보고서, 2012

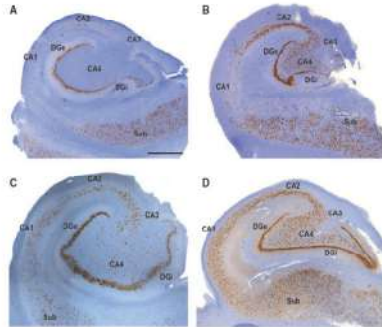
### Brain MRI

- ❖ **When to perform Brain MRI**
  - Newly diagnosed epilepsy
  - Longstanding epilepsy
    - Drug resistant epilepsy
      - candidates for surgery
    - Focal epilepsy of unknown etiology
      - surgically treatable lesions, such as low-grade tumors
  - Urgent assessment of patients with new-onset seizures
  - Presurgical evaluation

### Mesial Temporal Lobe Epilepsy

- ❖ **MTLE**
  - The most common surgically remediable epileptic syndrome.
  - HS (hippocampal sclerosis)** is the most common pathologic substrate
    - 40% of temporal lobe surgical specimen
    - Neuronal cell loss, gliosis, and granule cell dispersion
    - Pathology in other mesial temporal lobe structures (amygdala and entorhinal cortex)
    - Febrile seizures or other cerebral insults early in life
    - Good surgical candidates: seizure freedom in the 60% to 80%

### Histopathologic subtypes of HS



A. ILAE HS type 1  
B. ILAE HS type 2  
C. ILAE HS type 3  
D. No HS

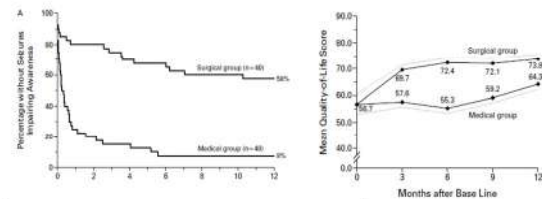
NeuN immunohistochemistry with hematoxylin counterstaining

Epilepsia, 54(7):1315-1329, 2013

### Temporal lobe resection

#### A RANDOMIZED, CONTROLLED TRIAL OF SURGERY FOR TEMPORAL-LOBE EPILEPSY

SAMUEL WEIR, M.D., WARREN T. BLUME, M.D., JOHN P. GREEN, M.D., Ph.D., AND MICHAEL ELIASZOW, Ph.D., FOR THE EFFICACY AND EFFICIENCY OF SURGERY FOR TEMPORAL LOBE EPILEPSY STUDY GROUP\*



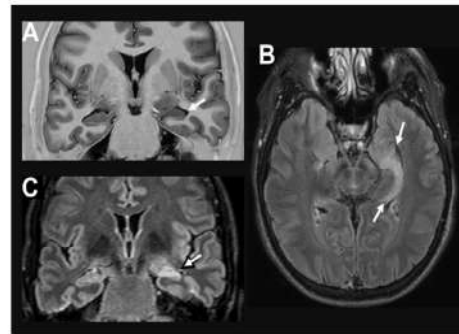
HS in MRI: 72.5% (medical) and 70.0% (surgical)

N Engl J Med. 2001;345(5): 311-318

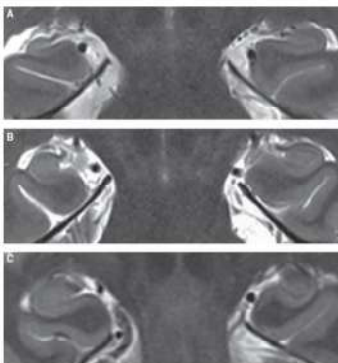
### MRI findings of HS

- ❖ **Hippocampal atrophy**  
The most specific and reliable feature  
Determined by comparing the size on each side on coronal slices (small asymmetries can be normally present)  
Shape of the hippocampus: oval (normal) → flattened (HS)
- ❖ **Increased T2/FLAIR signal**  
May be insufficient to diagnose HS in isolation
- ❖ **Loss of internal hippocampal structure**  
Consequence of neuronal loss and gliosis
- ❖ **Other features**  
Asymmetry of the temporal horn of the lateral ventricles  
Atrophy of the anterior temporal lobe  
Atrophy of the fornix and mammillary body

### MRI findings of HS



### MRI findings of HS



Degrees of hippocampal atrophy

A. ILAE HS type 1: severe  
B. ILAE HS type 2: moderate  
C. ILAE HS type 3: not detectable

Epilepsia, 54(7):1315-1329, 2013

### Neocortical epilepsy

- ❖ The most common lesions causing neocortical epilepsies:  
Low-grade tumors, malformations of cortical development, posttraumatic and postischemic lesions, inflammatory infectious scars, cavernous malformations, and arteriovenous malformations

## Focal cortical dysplasia

### ❖ FCD

Approximately one-fourth of focal epilepsies  
Highly epileptogenic, leading to intractable epilepsy in 75%  
Characterized by disorganization of the cortical lamination associated with bizarre (dysplastic) neurons or balloon cells  
General MRI findings

- cortical thickening
- loss of the interface between white and gray matter
- focal atrophy
- hyperintense signal in T2/FLAIR sequences

## FCD classification

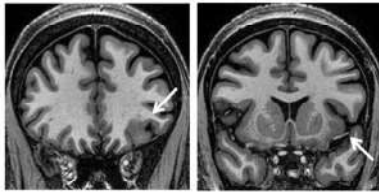
Table 1. The three-tiered ILAE classification system of focal cortical dysplasia (FCD) distinguishes isolated forms (FCD Types I and II) from those associated with another principal lesion (FCD Type III).

FCD Type I (isolated)	Focal cortical dysplasia with abnormal radial cortical lamination (FCD Type Ia)	Focal cortical dysplasia with abnormal tangential cortical lamination (FCD Type Ib)	Focal cortical dysplasia with abnormal radial and tangential cortical lamination (FCD Type Ic)
FCD Type II (isolated)	Focal cortical dysplasia with dysplastic neurons (FCD Type IIa)	Focal cortical dysplasia with dysplastic neurons and balloon cells (FCD Type IIb)	
FCD Type III (associated with principal lesion)	Cortical lamination abnormalities in the temporal lobe associated with hippocampal sclerosis (FCD Type IIIa)	Cortical lamination abnormalities adjacent to a glial or glioneuronal tumor (FCD Type IIIb)	Cortical lamination abnormalities adjacent to vascular malformation (FCD Type IIIc)
			Cortical lamination abnormalities adjacent to any other lesion acquired during early life, e.g., trauma, ischemic injury, encephalitis (FCD Type IIId)

FCD Type II (not otherwise specified, NOS): if directly radiologically suspected principal lesion is not available for macroscopic inspection. Please note that the rare association between FCD Types IIa and IIb with hippocampal sclerosis, tumor, or vascular malformations should not be classified as FCD Type III variant.

Epilepsia, 52(1):158-174, 2011

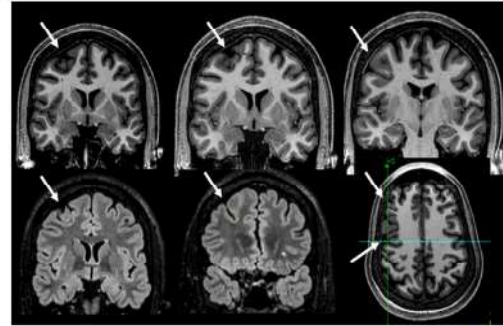
## FCD type I



Thickened cortex and blurred cortical-subcortical transition in the left lateral-basal frontal lobe.

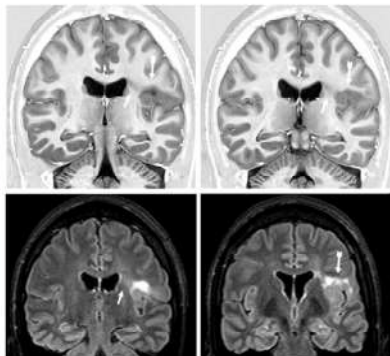
Also be associated with focal volume loss and thin cortex  
In many patients MRI does not show abnormalities in the white matter

## FCD type II



Thickened cortex associated with abnormal gyri and cortical dimple (T1)  
slightly blurred cortical-subcortical transition (FLAIR)  
Pathology: FCD Type IIA

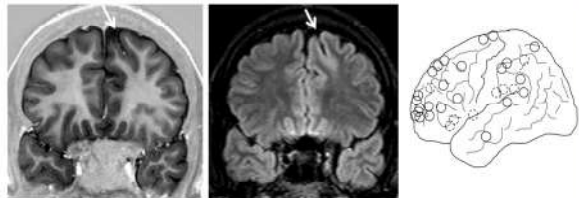
## FCD type II



### FCD Type IIB

Cortical thickening  
Loss of the cortical-subcortical transition  
"Transmantle" sign  
hyperintense T2 FLAIR signal in the subcortical white matter with a wedge shape that extends to the ventricle

## FCD type II



### FCD Type IIB

"Bottom-of-sulcus" dysplasia

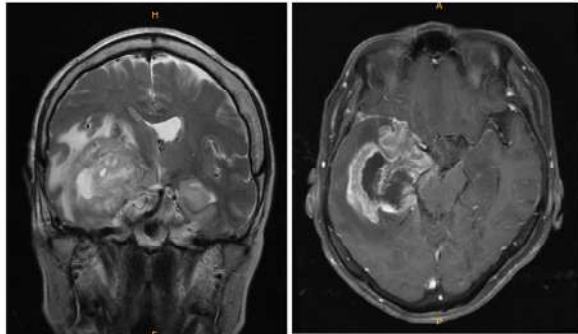
Thickened cortex and hyperintense FLAIR signal in the bottom of deep sulcus  
Dysplastic features are maximal at the sulcal depth, tapering to a relatively normal gyral crown, with or without transmantle sign  
The most common location: frontal, parietal, and insula cortex  
Motor/premotor/ prefrontal seizure semiology, the nocturnal occurrence  
Surgically remediable (seizure free rate: 88%, 28/32)

Neurology 2015;84:2021-2028





## Epilepsy-associated tumors



SCH

## Presurgical Evaluation

## Presurgical evaluation of epilepsy

### ❖ Objective of resective epilepsy surgery

Complete resection or complete disconnection of the **epileptogenic zone**, which is defined as the area of cortex indispensable for the generation of clinical seizures.

Table 1 Descriptions of zones and lesions of the cortex (adapted from Lüders and Awad, 1992)

Epileptogenic zone	Region of cortex that can generate epileptic seizures. By definition, total removal or disconnection of the epileptogenic zone is necessary and sufficient for seizure-freedom.
Irritative zone	Region of cortex that generates interictal epileptiform discharges in the EEG or MEG
Seizure onset zone	Region where the clinical seizures originate
Epileptogenic lesion	Structural lesion that is causally related to the epilepsy
Ictal symptomatic zone	Region of cortex that generates the initial seizure symptoms
Functional deficit zone	Region of cortex that in the interictal period is functionally abnormal, as indicated by neurological examination, neuropsychological testing and functional imaging or non-epileptiform EEG or MEG abnormalities
Eloquent cortex	Region of cortex that is indispensable for defined cortical functions

Brain. 2001;124(Pt 9):1683-700.

## Lesional and Non-lesional epilepsy

### ❖ Lesional epilepsy

Epilepsy with an unequivocal MRI abnormality responsible for seizures  
2.5-times greater likelihood of a seizure-free outcome

Categories (in of studies)	Non-lesional			Lesional		
	Total N patients	Seizure-free %	95%CI	Total N patients	Seizure-free %	95%CI
<b>Temporal and extratemporal</b>						
Overall (n=35)	697	43	39-46	2860	68	66-70
Using MRI (n=19)	398	56	45-51	965	70	68-73
Using histopathology (n=17)	302	39	34-44	1953	67	65-69
<b>Temporal lobe</b>						
Overall (n=20)	398	45	40-49	1657	69	66-70
Using MRI (n=12)	226	51	45-57	514	75	71-79
Using histopathology (n=8)	172	36	29-43	1179	65	63-68
<b>Extratemporal</b>						
Overall (n=13)	136	34	27-41	350	66	61-70
Using MRI (n=9)	124	35	27-42	225	60	54-66
Using histopathology (n=4)	35	32	18-47	125	74	67-82

Similar outcomes in children, adults, and studies that used MRI or histopathology to identify lesions.

Epilepsy Research (2010) 89, 310-318

## Lesional and Non-lesional epilepsy

### ❖ Non-lesional epilepsy

Location	No. of Patients	Engel Class			
		I	II	III	IV
Frontal	35	15	1	12	7
Neocortical temporal	31	17	3	5	6
Parietal	11	5	1	3	4
Occipital	11	7	1	3	0
Multifocal	1	0	0	0	1
Total	99	42 (44.2%)	6 (6.7%)	23 (25.8%)	18 (20.2%)

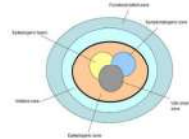
Localization by FDG-PET and interictal EEG was correlated with a seizure-free outcome

Concordance with two or more presurgical evaluations

was significantly related to a seizure-free outcome

Surgical treatment can benefit selected patients with nonlesional neocortical epilepsy

Careful interpretation of presurgical evaluations and the presence of concordant results



Ann Neurol 2005;58:525-532

## Brief history

여자 33세.

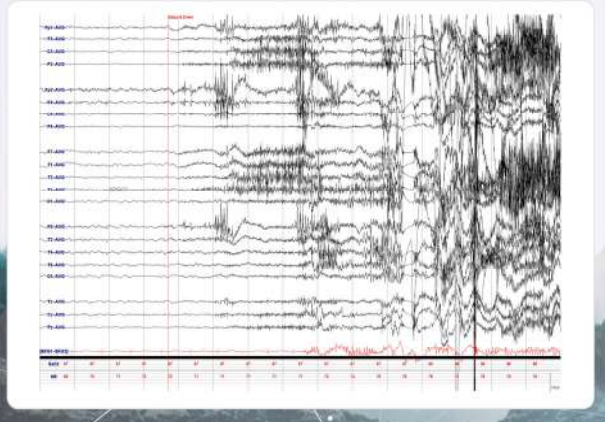
중 2때부터 Sz 시작.

2004년 Rt anterior temporal lobectomy 타병원

수술 이후에도 증상 반복, 2014년부터 빈도 증가 (한 달에 2-3회)

CBM2S 200mg bid, TPM25 bid 복용

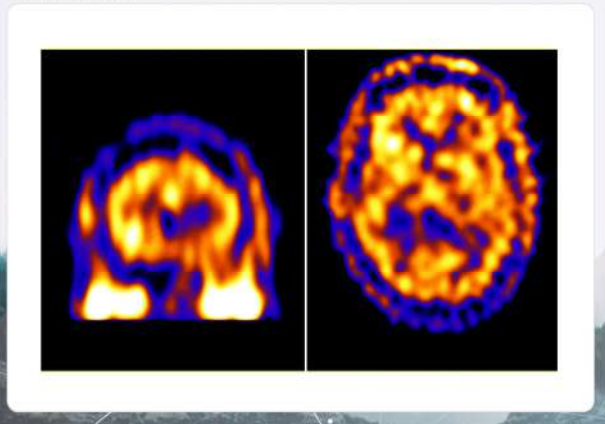
Ictal EEG



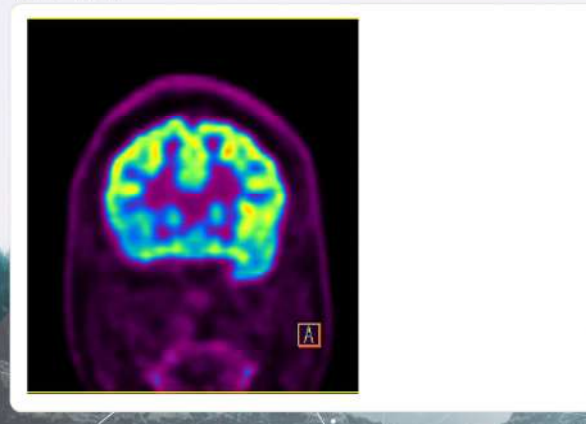
Brain MRI



Ictal SPECT



FDG-PET



Brief history

41세 남자  
중학교 1학년부터 GTC 양상의 seizure 발생. 여러 대학병원에서 AED 복용했으나 seizure 지속되어 내원.

#### Semiology

주로 수면 중에 팔다리 경직되면서 뺨치는 양상으로 발생  
EBD(+), drooling (+), urinary incontinence (-); 매번 일정하지 않다.  
Frequency: variable, 많을 때는 하루 4-5번, 적을 때는 일주일 이상 sz free  
Duration: 30초-1분 미만  
Aura (+): 소름 끼치는 느낌이 아래에서 부터 위로 올라오는 느낌

Ictal EEG

