

# Epilepsy surgery preoperative work-up



# When to consider epilepsy surgery ?



- Persistent Seizures despite adequate pharmacological treatment

Drug resistant epilepsy may be defined as failure of adequate trials of two tolerated and appropriately chosen and used AED schedules (whether as monotherapies or in combination) to achieve sustained seizure freedom)

Definition of drug resistant epilepsy: Consensus proposal by the ad hoc Task Force of the ILAE Commission on Therapeutic Strategies :Patrick Kwan, yAlexis Arzimanoglou, zAnne T. Berg, xMartin J. Brodie, {W. Allen Hauser, #2Gary Mathern, \*\*Solomon L. Moshe´ , yyEmilio Perucca, zzSamuel Wiebe, and xx2Jacqueline French:Epilepsia, \*(\*)1–9, 2009

- Significant impairment of quality of life

# The goal of the presurgical evaluation



To generate a “surgical diagram” depicting

- Epileptogenic region (ER)

Location

Extent

- Critical cortex ( CC)
- Surgical resection = ER - CC

The most common location of seizure onset in adults is the temporal lobe, especially the medial temporal lobe (hippocampus). This is also the seizure location most amenable to surgical cure.



- 90 % of adult patients with seizure disorder have partial or localization related epilepsy.
- 80 % of the patients of partial seizure experience temporal lobe seizures
- Nearly 90 % of the partial seizures of temporal lobe origin emanate from the medial temporal lobe
- The majority of extratemporal epilepsy patients have seizures of frontal lobe origin

(Chabolla DR ,Casino GD .The treatment of epilepsy ;Principles and Practice,1996:264-279)

# Presurgical tests



No single test adequately defines the epileptogenic region

- Mandatory noninvasive – “universal access”
  - Clinical semiology and Exam
  - Scalp EEG
  - Imaging MRI



- Optional Noninvasive – “Limited Access “

Video EEG

Magnetoencephalography (MEG),

EEG-fMRI,

Nuclear imaging (PET,SPECT )

Neuropsychological testing

Wada (intracarotid amobarbital) test

- Invasive EEG monitoring

# Clinical semiology and Exam



- TLE may have an epigastric aura, followed by a quiet period of unresponsiveness with staring, lipsmacking (oral automatisms).
- Picking at sheets or clothes (manual automatisms), contralateral dystonic posturing, and postictal confusion and lethargy.
- If from the dominant hemisphere, there is usually delayed recovery of language, often with transient aphasia on testing.



- A typical frontal lobe seizure will occur from sleep with no warning, may show restlessness and/or prominent bilateral limb movements (such as bicycling or asymmetric tonic posturing), and will end quickly with immediate recovery; this may recur several times in one night.



- Occipital lobe seizures often have a visual aura, and may progress (due to electrical spread) into a temporal lobe or frontal lobe type of seizure.
- Parietal lobe seizures are the least common, may have a sensory aura, and tend to mimic frontal lobe seizures.

# SEMIOLGY OF FRONTAL VERSUS TEMPORAL LOBE



<u>FEATURES</u>	<u>FRONTAL LOBE</u>	<u>TEMPORAL LOBE</u>
<b>SEIZURE FREQUENCY</b>	FREQUENT, OFTEN DAILY	LESS FREQUENT
<b>SLEEP ACTIVATION</b>	CHARACTERSTIC	LESS COMMON
<b>SEIZURE ONSET</b>	ABURT, EXPLOSIVE	SLOWER
<b>PROGRESSION</b>	RAPID	SLOWER
<b>INITIAL MOTIONLSS STARTING</b>	LESS COMMON	COMMON
<b>AUTOMATISM</b>	LESS COMMON	MORE COMMON AND LONGER
<b>BIPEDAL AUTOMATISM</b>	CHARACTERSTIC	RARE
<b>COMPLEX POSTURE</b>	EARLY, FREQUENT, AND PROMINENT	LATE, LESS FREQUENT AND LESS PROMINENT
<b>HYPERKINETIC MOTOR SIGNS</b>	COMMON	RARE
<b>SOMATOSENSORY SYMPTOMS</b>	COMMON	RARE
<b>SPEECH</b>	LOUD VOCALISATION(GRUNTING, SCREAMING, MOAMING)	VERBALIZATION SPEECH IN NON DOMINENT SEIZURE
<b>SEIZURE DURATION</b>	BRIEF	LONGER
<b>SECONDARY GENERALISATION</b>	COMMON	LESS COMMON
<b>POST ICTAL CONFUSION</b>	LESS PROMINENT OR SHORT	MORE PROMINENT AND LONGER
<b>POST ICTAL DYSPHASIA</b>	RARE, UNLESS IT SPREADS TO THE DOMINENT TEMPORAL LOBE	COMMAN IN DOMINENT TEMPORAL LOBE SEIZURE

# LANGUAGE ABNORMALITIES



<b>ICTAL SPEECH AREST</b>	TEMPORAL LOBE SEIZURES, USALLY DOMINANT HEMISPHERE
<b>ICTAL SPEECH PRESERVATION</b>	TEMPORAL LOBE SEIZURES, USUALLY NON-DOMINANT HEMISPHERE
<b>POST ICTAL DYSPHASIA</b>	DOMINANT HEMISPHERE INVOLVEMENT

# **AUTONOMIC FEATURES**



<b>ICTUS EMETICUS</b>	RIGHT TEMPORAL SEIZURES
<b>ICTAL URINARY URGE</b>	RIGHT TEMPORAL SEIZURES
<b>PILOERECTIOM(GOOSE BUMPS)</b>	LEFT TEMPORAL SEIZURES

# MOTOR ABNORMALITIES



<b>EARLY NONFORCED HEAD TURN</b>	IPSILATERAL OF SEIZURE ORIGIN
<b>LATE FORCED HEAD TURNED</b>	CONTRLATERAL OF SEIZURE ORIGIN
<b>EYE DEVIATION</b>	CONTRLATERAL OF SEIZURE ORIGIN
<b>FOCAL CLONIC JERKING</b>	CONTRLATERAL OF SEIZURE ORIGIN, PERI-ROLANDIC
<b>ASYMMETRIC CLONIC ENDING</b>	CONTRLATERAL OF SEIZURE ORIGIN
<b>DYSTONIC LIMB POSTURING</b>	CONTRLATERAL OF SEIZURE ORIGIN
<b>FENCING POSTURE</b>	CONTRLATERAL FRONTAL LOBE (SUPPLEMENTARY MOTOR) SEIZURES
<b>FIGURE OF 4 SIGN</b>	CONTRALATERAL TO EXTENDED LIMB, USUALLY TEMPORAL LOBE
<b>UNILATERAL ICTAL PARESIS</b>	CONTRLATERAL OF SEIZURE ORIGIN
<b>POSTR ICTAL TODD'S PARESIS</b>	CONTRLATERAL OF SEIZURE ORIGIN

# **EXAMINATION OF PATIENT DURING THE SEIZURE OF SEMILOGIC CHARACTERISTICS**



<b><u>EXAMINATION</u></b>	<b><u>SIGNIFICANCE</u></b>
<b>RESPONSE TO COMMUNICATION</b>	LEVEL OF AWARENESS
<b>SPEECH (NAMING, READING)</b>	DOMINANT HEMISPHERIC INVOLVEMENT
<b>MEMORY (PRESENTING WORDS OR PHRASES FOR LATER RECALL)</b>	TEMPORAL LOBE INVOLVEMENT
<b>DISTRACTIBILITY</b>	FRONTAL LOBE INVOLVEMENT
<b>RESPONSE TO PASSIVE EYE OPENING</b>	TO EXCLUDE PSEUDOSEIZURE (TIGHT CLOSURE )
<b>RESPONSE TO PHYSICAL STIMULATION</b>	ATTENTION, MOTOR DYSFUNCTION
<b>WEAKNESS OR LACK OF MOTOR CONTROL</b>	CONTRALATERAL SEIZURE ORIGIN
<b>PLANTER EXTENSOR RESPONSE</b>	POST ICTAL PARESIS

# TEMPORAL LOBE SEIZURES



- The seizures are typically complex partial, with or without simple partial onset.
- Jackson described the so-called “dreamy state” in seizure Semiology in the 19th century.
- The Montreal school described these psychic phenomena as interpretative illusions or experiential hallucinations.
- Oroalimentary automatisms are common, occurring in approximately 70% of cases of limbic (hippocampal) seizures compared to 10% of patients with extra-limbic seizures.



- Unilateral dystonic posturing of an arm is classical of contralateral temporal lobe epilepsy.
- Ipsilateral upper arm automatism.
- Postictal coughing.
- Olfactory auras - poorly recognizable, always unpleasant, foul smell - “uncal” or “uncinate” fits.
- Fear is another limbic aura considered to be amygdaloid in origin.



- Patients with left temporal lobe seizures cannot read normally post-ictally.
- Post-ictal dysphasia
- Behavioral aggression , sexual features, fetishes, and hypergraphia.
- Temporal Lobe seizures can be separated into
  - Limbic -amygdala ( 80 % )
  - Antero infero mesial - entorhinal, hippocampal area
  - Neocortical - neocortex,
- The auditory illusory auras may originate from either side of temporal neocortex.

# FRONTAL LOBE SEIZURES



- Briefer
- Associated with less post-ictal confusion
- More motor in characteristics
- Are more likely to be involved with secondary generalization
- Are less likely to demonstrate psychic/emotional/affective phenomena
- Are more likely to exhibit a rapid onset and offset, and are more likely to occur nocturnally than other partial seizures
- The automatisms of frontal lobe are of a “forced nature”
- These collectively stand in particular contrast to temporal lobe seizures.

## *Fronto-polar:*



- Origin are often from scars following head injuries.
- They have the greatest likelihood of simply being characterized by what appear clinically to be primary generalized seizures,
- they may have some of the general characteristics of FLE, especially contralateral head deviation.

# *Orbito-frontal*



- May have semiology that mimics temporal lobe seizures in which case the origin is usually attributed to the posterior part of the orbital cortex.
- Once again, however, it may involve, in various combinations, other frontal lobe semiology, especially contralateral head and eye deviation.

## *Premotor:*



- Head and eye deviation with varying bilateral tonic posturing is the commonest seizure.
- When the deviation occurs at the onset of the seizure when consciousness is intact it has significant localizing value to the contra lateral frontal cortex.
- Those seizures arising in the mid part of the frontal lobe have a prominent bilateral tonic posturing. Bipedal automatisms may take the form of symmetric bicycling or kicking movements.

- Prominent leg movements favor involvement of the supplementary motor area.



- The so-called “fencing posture” is classically associated with the contralateral FL, particularly the SMA.
- This complex posture is characterized by abduction, external rotation, and partial flexion of the contralateral arm at the shoulder, contralateral deviation of the head and eyes such that they “look at” the contralateral arm, extended ipsilateral arm downwards and backwards, and with the feet apart so as to support the partially contralaterally rotated trunk. Occasionally, the upper limb is also flexed at the elbow with the hand raised to the face that has turned forcefully towards it.
- There is occasionally guttural, ill-understood speech.

# *Dominant opercular*



- Usually begins with an alteration in speech.
- The alteration may be typical dysphasic speech, a form of non-specific guttural speech, or an arrest of speech.
- Post-ictal dysphasia.

# *Rolandic*



- Rolandic seizures combine both motor and sensory components.
- Sensory features can be positive (e.g. pins and needles, pain, pricking, tingling) or negative (numbness).
- Elementary paraesthesiae are reported to be the most characteristic of seizures arising in the post-central gyrus.
- In pure sensory seizures there is nearly always dysfunction of the part involved, which may be awkwardness, typical sensory ataxia, or paralysis.



- Spread (intracortically) within the Rolandic cortex, e.g., sensory and/or motor, over contiguous parts of the associated homunculus, clinically is reflected in a “march” from one place on the body to another.
- Unlike the tonic activity associated with seizures arising outside of the Rolandic cortex, which often has unreliable localizing and lateralizing value, Rolandic motor activity is clonic and is unambiguously localizing to the contralateral motor cortex in the area of the homunculus from which it arises, and usually is associated with transient post-ictal weakness in the involved part.

# EEG



- Developed in 1930s.
- Awake & Sleep
- Ictal & Interictal
- Eye opening & Closure, Hyperventilation, Photic stimulation.

# Scalp EEG



- Lateralization (left vs. right hemisphere) and localization.
- Additional electrodes that are particularly sensitive to medial temporal discharges.
- The presence of *interictal (between seizures) epileptiform discharges*.
- EDs: sharp waves or spikes.
- **Lack of EDs does NOT rule out epilepsy (up to 10%).**



- Spike waves : negative transients with steep ascending and descending limbs and a duration of 20 to 70 ms.
- Sharp waves have broader potential (70 – 200 ms).
- TIRDA
- PLEDs



- Interictal s/o increased epileptogenic potential.
- Ictal Eds s/o classification of seizure, brain region of seizure activity.
- Sensitivity and specificity depends upon,
  - Localization of epileptic brain tissue.
  - Duration of recording.
  - Use of supplementary electrodes.
  - Frequency of seizures
  - Timing in relation to last seizure.



- Medial temporal lobe epilepsy patients usually have EDs from the anterior-mid temporal lobe (electrodes F7/F8, T3/T4, as well as anterior temporal, sphenoidal, and inferior temporal electrodes if used).
- Bilateral temporal lobe EDs are not unusual(25-30%)
- Diagnostic yield increases to 90 % in TLE by obtaining sleep study (Vs 50 % in awake)

(Chabolla DR ,Casino GD .The treatment of epilepsy ;Principles and Practice, 1996:264-279)

# Video EEG



- ❑ The **combined** use of **video and EEG recording** improves the **sensitivity and specificity** over EEG recording alone.
- ❑ In **many cases** of medically refractory seizures, video-EEG recording of the patient's symptoms should be performed **as a first study in the proper classification of the seizure disorder**.
- ❑ Inpatient monitoring is often necessary when **AEDs are withdrawn to enhance the chance of recording a seizure during monitoring**.



- Ambulatory
- Analog Video
- Digital video
- Telemetered EEG



- ❑ A typical **duration of inpatient monitoring is 3 to 5 days.**
  
- ❑ Video-EEG monitoring has been **demonstrated to-**
  - # to accurately **differentiate between epileptic and non-epileptic seizures,**
  
  - # to **distinguish between primarily generalized and partial onset seizures,**
  
  - # to determine **seizure onset localization and lateralization**

# Clinical applications



- Diagnostic classification
- Surgical localization
- To modify AEDs
- To identify nonepileptic spells

# Imaging CT



- Sturge weber syndrome – Tram track calcification.
- Calcification associated with neurocysticercosis.
- Tuberos sclerosis sub ependymal tubers.
- Abandoned in most epilepsy centers.
- 95 % Vs 32 % sensitivity (MRI Vs CT).
- Probability of positive CT in negative MR 0 % while 80 % positive MR in CT negative cases.

(Bronen RA, Fullbright RF, Spencer SS et al; Refractory epilepsy :comparison of MR imaging with CT and Histopathology, Radiology 201;97-106, 1996.)

# Imaging MRI



- The primary role to locate and define lesions.
- Overall sensitivity 86% compared with surgical findings.
- In TLE sensitivity is 75 % to 100 % for hippocampal sclerosis and more than 90 % for focal abnormalities.
- Specificity 70- 85 % and 90 % respectively.

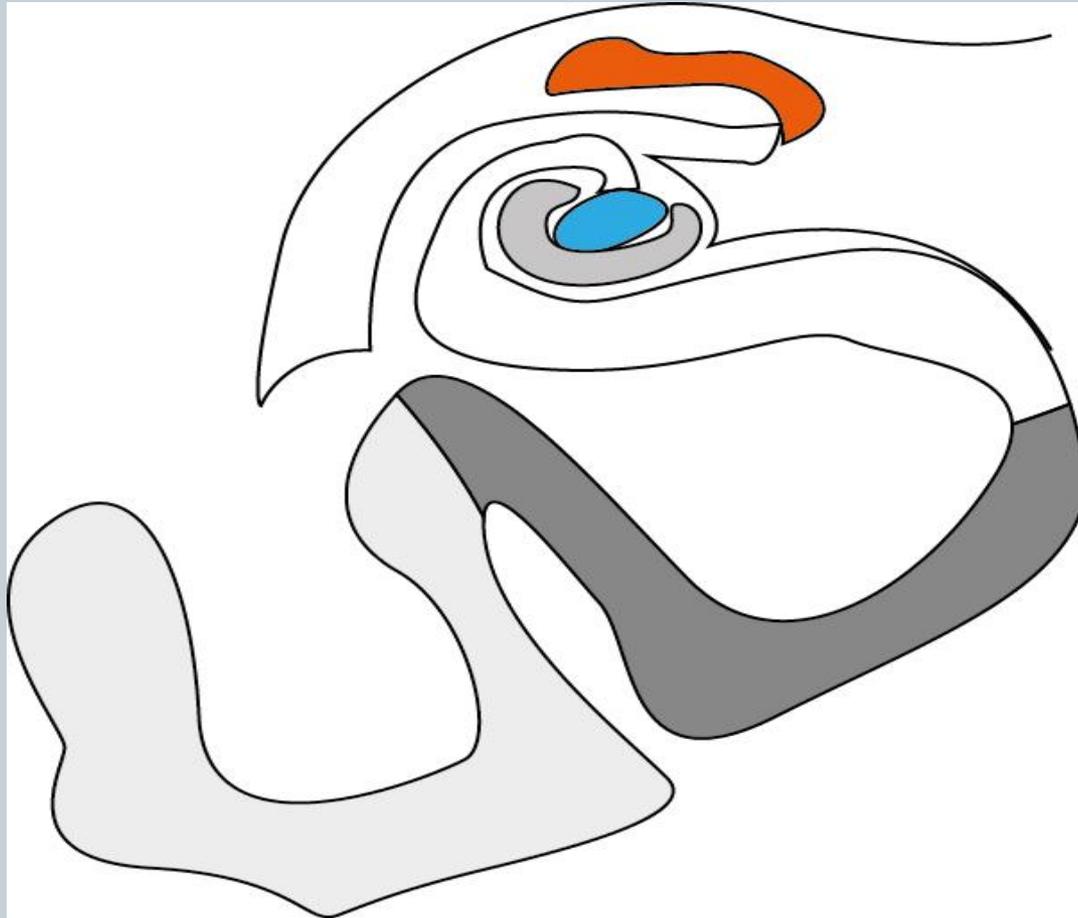
(Bronen RA, Fullbright RF, Spencer SS et al; Refractory epilepsy :comparison of MR imaging with CT and Histopathology, Radiology 201;97-106,1996.)



- MRI also can depict relation of epileptogenic foci and functional important tissue.
- Candidature for surgery
- Need for invasive EEG, planning electrodes.
- Prognostication



- 70 – 90 % seizure control Vs 40 – 55 % seizure control.
- Berkovic et al.: seizure free state
  - 80 % focal lesions
  - 62 % MTS
  - 36 % normal MRI





- Coronal oblique MRI through the temporal lobes is the preferred modality.
- Classic MRI findings in mesial temporal sclerosis include decreased volume, an abnormally increased T2 signal of the hippocampus and abnormal morphology.
- The increased T2 signal is presumed to be a result of gliosis and the subsequent increase in free water content.
- Associated findings may include atrophy of the ipsilateral mammillary body, fornix, and other parts of the limbic system.

- Because fluid-attenuated inversion recovery (FLAIR) imaging nulls the CSF signal, abnormal signal intensity in the hippocampus is relatively more apparent.

- A pitfall of coronal FLAIR imaging is the slight hyperintensity of all limbic structures relative to the neocortex.
- Thin-section volumetric T1-weighted imaging is occasionally used to calculate hippocampal volume; however, because it does not depict abnormal signal intensity, it is less useful than FLAIR and T2-weighted spin-echo imaging for visual detection of mesial temporal sclerosis.



- T2-weighted spin-echo imaging is somewhat better than FLAIR imaging for demonstrating the internal architecture of the hippocampus; however, the degree of signal abnormality is somewhat more obvious on FLAIR imaging.
- Special oblique coronal thin sections perpendicular to the plane of the hippocampus have high sensitivity and specificity for mesial temporal sclerosis.

# Quantitative MR Imaging



- In some patients with intractable focal epilepsy, structural MR imaging does not demonstrate a lesion.
- Usually of 3D T1-weighted images, offers an objective means of analyzing MR images and therefore improves the likelihood of detecting subtle lesions.
- Volumetric neocortical measurements may provide an objective way to evaluate the extent of resection and its relation to surgical results.

# Diffusion Tensor Imaging



- In the white matter, diffusion is greatest parallel to the white matter tracts but minimal perpendicular to them.
- Malformations of cortical development and acquired lesions.
- Increased mean diffusivity.
- Increased perpendicular diffusivities.

# Magnetic Resonance Spectroscopy



- Magnetic resonance spectroscopy is a powerful adjunct to MR imaging.
- Reduced NAA and increased Cho in TLE.
- The MR spectroscopy findings were consistent with the histopathological characteristics of reduced neuron cell counts or neuronal dysfunction and increased glial cell numbers.
- Extratemporal lobe epilepsy, the ability of MR spectroscopy to lateralize the epilepsy is less(50%).

Maton BM, Kuzniecky RI: Proton MRS: N-acetyl aspartate, creatine, and choline. **Adv Neurol 83:253–259, 2000**

# Functional MR Imaging



- Used for mapping language, memory, and sensorimotor location for presurgical planning.
- This imaging modality is based on the observation that increased neuronal activity is associated with an increase in CBF and therefore an increase in the oxyhemoglobin/ deoxyhemoglobin ratio.

# Magnetoencephalography (MEG)



- The MEG modality measures extracranial magnetic fields perpendicular to the direction of intracellular currents in apical dendrites.
- One of the advantages of MEG as opposed to EEG studies is that magnetic fields are minimally affected by conductivities of intervening structures and tissues between brain and scalp.
- Presurgical mapping of eloquent cortex.

# Nuclear imaging



- **PET:**
- Positron emission tomography (PET) utilizes an injection of radio-labeled glucose ( $^{18}\text{F}$ FDG) to measure brain metabolism. Interictal PET usually shows hypometabolism in the seizure focus, especially in TLE.
- Ictal PET is not practical due to the extremely short half life of the radiotracers used.
- PET is most useful in MRI-negative TLE, though it may be helpful in extra-temporal epilepsy as well.



- FDG PET identifies sites of interictal metabolism in 70 % patients with TLE and 60 % frontal lobe onset seizure.
- Less than 5 % false positivity.

(Davinsky O, Pacia S : Epilepsy surgery. Neurology clinics 11:951-971,1993)

- C -11 flumazenil PET effective in hippocampal sclerosis & shows pathological foci in a more circumscribed fashion.

(Szeliés B ,Pawlik G etal: MRI guided flumazenil and FDG –PET in temporal lobe epilepsy ,Neuroimage 3 : 109-118,1996.)

# SPECT



- Ictal SPECT ideal method for localization and lateralization of seizure onset,
- Shows hypermetabolism,.
- 89 % sensitivity for localization (Vs PET 63 % )
- SPECT also performed better in the setting of negative MRI.
- False lateralization rare.

(Ho SS, Berkovic SF etal: Comparison of Ictal SPECT and interictal PET in the presurgical evaluation of temporal lobe epilepsy, Annals of neurology 347:738-745.)



- Post Ictal SPECT Comparable to interictal PET (70 %).
- Inter ictal scan – Hypoperfusion large area in the hemisphere of onset.
- Interictal scan has less sensitivity for seizure, 50 %.
- Ictal /Interictal quantitative difference analysis provides for the best and most reliable seizure focus localization

# *Subtraction ictal- interictal SPECT co-registered to MRI (SISCOM):*



- A newer methodology that has greater accuracy than either ictal or interictal SPECT scanning.
- This requires obtaining scans(separated by at least 48 hrs to accommodate radionucleotide wash out) during an interictal period and within seconds of seizure onset.
- These scans are then subtracted from one another with the use of specialized computer software.
- This leaves a better indication of the cortical area of ictal onset.
- This subtracted scan can then be co-registered onto the patient's MRI to provide support for the location of the focus.

# Invasive EEG monitoring



## ***INDICATIONS:***

- ❑ Seizures are **lateralized but not localized**.
- ❑ Seizures are **localized but not lateralized**.
- ❑ Seizures are **neither localized nor lateralized** (eg, stereotyped complex partial seizures with diffuse ictal changes).
- ❑ Seizure **localization is discordant** with other data.
- ❑ **Relationship of seizure onset to lesion must be determined** (eg, dual pathology or multiple intracranial lesions).

.



- **Temporal lobe seizures**

- Doubtful side

- Normal MRI

- Bilateral pathology

- Discordant non-invasive testing

- **Extratemporal seizures**

- Definition of extent of epileptogenic area

- Extra-operative cortical mapping



- ***TYPES OF ELECTRODES:***

- ❑ **Depth,**
- ❑ **strip**
- ❑ **grid electrodes**
  
- ❑ **Implantable intracranial devices** used to **record the electrocorticogram (ECoG).**
  
- ❑ **to stimulate the cortex to determine function.**
  
- ❑ **Depth electrodes** are multicontact, thin, tubular, rigid or semirigid electrodes.
  
- ❑ **Penetrate the brain substances** for the purpose of **recording from deep structures.**



- ❑ **Intracranial strip electrodes** are a **linear array** of 2-16 disk electrodes **embedded in a strip of silastic (Wyler, 1984)**, or they can be **tubular** in structure, **(Dubeau, 2000)**.
- ❑ **Grid electrodes** are **parallel rows** of similar numbers of electrodes that can be configured in standard or custom designs.
- ❑ **Grid and strip electrodes** are **designed to be in direct contact with brain neocortex**.
- ❑ **Electrodes** are **placed in the subdural space**.
- ❑ **Occasionally** be used in the **epidural space**.



- ❑ Constructed from **biologically inert materials (ie silastic, stainless steel, platinum).**
- ❑ **Platinum electrodes** are more easily seen on fluoroscopic images and are compatible with MRI.

### ***Complications***

- ❑ **Infection rates** from all types of intracranial electrodes range from **0-12%.**
- ❑ The morbidity of surgery depends on the type of electrode implantation.
- ❑ intracranial **strip** electrodes have the **lowest morbidity.**
- ❑ Intracranial **grid** placement has the **highest morbidity.**
- ❑ **transient neurological deficit, hematoma, cerebral edema with increased intracranial pressure, and infarction.**

<b><i>Procedure</i></b>	<b><i>Reported Complications</i></b>
Depth Electrodes	-Hemorrhage – 1- 3%  -Infection – Intracranial abscess  -Malplacement
Subdural Strips	-Hemorrhage  -Infection – meningitis
Subdural Grids	-Infection – bone flap  -Raised intracranial pressure
Foramen Ovale Electrodes	-Facial pain  -Infection – meningitis or abscess

# *Intracarotid amobarbital (Wada) test*



- ❑ Developed by **Jun Wada**.
- ❑ To **preoperatively determine which hemisphere contains language function**, this remains its primary use.
- ❑ **Also used to test memory function within each hemisphere** when considering AMTR.
- ❑ Accomplished by individually **cannulating each internal carotid artery**.
- ❑ After contrast arteriography verifies that blood flows to the corresponding hemisphere and not to the brainstem or contralateral side, =====→ **a dose of sodium amobarbital (sufficient to impede hemispheric function) is injected**.



- ❑ **If speech persists in the face of this hemiparesis, language function is assumed to not be represented within that hemisphere.**
- ❑ **The deficiencies of this evaluation for memory function directly relate to the multiple problems.**
- ❑ **Injection of a drug into the internal carotid artery does not assure drug effect in the basal temporal area in general or the hippocampal region specifically.**
- ❑ **This is due to variations in the direct blood supply to the hippocampus.**



MR Positive

Concordant  
EEG

PET ,SPECT  
Not discordant

Surgery

Discordant  
EEG

Invasive EEG



MR Normal

Concordant  
EEG,PET

Scalp EEG  
Temp  
location

WADA ,SPECT  
Not discordant

Rest  
unconfirmed

Surgery

Invasive EEG

# Case 1



- **24year/ Male**
- **Diagnosis- CPS**
- **Birth History: Normal, slightly delayed milestones**
- **Febrile Convulsions- No**
- **Neurological Deficit : No**
- **Head Injury: No**
- **Sz onset / Duration: 21 years / 3 yrs**
- **Sz frequency- 1-2/month**
- **Semiology: Behavioral arrest with B/L UL in flexion**
- **Drugs- Valproate 1000mg, Levetiracetam 3000mg**



- EEG : Normal
- VEEG- : - 5 CE – clinically behavioral arrest and flexion b/l UL ; EEG – 4 CE marked by ictal onset Right Frontal ( 3 at F8 and 1 at F4 ) and 1 CE has onset at T4Cz.
- MRI : Rt MTS
- SPECT –

Ictal : R temporal lobe

Inter ictal : R temporal lobe

## Case 2



- **6 year/male**
- **Diagnosis- CPS**
- **Birth History: Normal**
- **Febrile Convulsions- No**
- **Semiology:** Automatic activity, aggressive behavior, vocalizations, bizarre movements of limbs
- **Sz onset/ Duration /frequency-** 4 years/ 2 yrs/ 12-15/day
- **Drugs-** Ox CBZ 600, Levetiracetam 1000, Valproate 800



- **VEEG-LTV-32/06- 5 CE**, all were non localizing CPS. Ictal EEG s/o LRE from **right centrotemporal** in three and unclear in two. Interictal EEG s/o bilateral fronto temporal discharges **R>L**
- **MRI Brain** :- Focal Cortical Dysplasia Rt inferior frontal and insular gyri
- **SPECT: R frontal lobe**
- SISCOS-likely localization of foci in right inferior frontal cortex. the focus in right ant - temporal region appears to be due to spread from right frontal cortex.

# Case 3



- **Age / Sex: 13 year/ Male**
- **Diagnosis- GTCS , ? Focal seizures**
- **Birth History: FT NVD, meconium aspiration and nursery stay 3 days of life ; normal milestones**
- **Febrile Convulsions- No**
- **Neurological Deficit : No**
- **Head Injury: No**
- **Sz onset / Duration: 4 years / 9 yrs**
- **Sz frequency- 1-2/ month**
- **Semiology:** Aura: Epigastric pain / discomfort with / without - Left head version with stiffening of both the UL ( speaks – hua tha repeatedly and goes to sleep) ;
- **Drugs- OxCBZ 600**



- **VEEG-LTV-73/10-**
- **MRI 2000 Pvt . – Report normal**
- **MRI (Pvt 12.2.10)- B/L MTS with R> Left**
- **SPECT – Not done**



- **Age / Sex: 17year/ Male**
- **Diagnosis- Left CPS seizures**
- **Birth History : FT LSCS for contracted pelvis ? Cry; normal milestones**
- **Febrile Convulsions- Yes ; 1sz at 1 ½ yrs of age , clinching of teeth with high grade fever**
- **Neurological Deficit : Left Hemiparesis , hypothyroidism +**
- **Head Injury: H/O blunt trauma to head at the age of 3 yrs ; no LOC no CT brain.**
- **Sz onset / Duration: 8 years / 9 yrs**
- **Sz frequency- 10-15/day**
- **Semiology: Abduction of Rt UL and clonic mvt Rt UL and Left LL and UL ; face deviated to left , eye uprolling**
- **Drugs- Valproate 1500mg, Clobazam 20mg.**

- **EEG pvt: normal**
- **VEEG-LTV-43/10; 64 LTM : - 6 CE seen ; Clinically all marked by abduction of the Rt UL and clonic mvts of the Left sided Limbs LL > UL , uprolling of eyeballs, ; EEG – 3 CE have onset at C4, 2 around C3 and 1 CE has onset best appreciable at Cz; interictal frequent sharps seen at Cz C4.**
- **MRI (Pvt ; 20.11.06 ; 30.10.2000)- normal ; AIIMS 48735/ NR /10- 25.2.10 - normal**
- **SPECT – Ictal – 19.3.10 (ECDS / 292/10) ; Inter ictal - (ECDS / 403/10) 21.7.10: B/L Temporal hypoperfusion ; SISCOS : Large focus of differentiated perfusion in the Rt Frontocentral region**



- **19 year/ male**
- **Diagnosis- intractable Left CPS**
- **Birth History: FTNVD at hospital, Normal mile stones**
- **Febrile Convulsions- No**
- **O/E: no focal neurological deficit**
- **Sz Onset / Duration: 4 years / 15 yrs**
- **Sz frequency- 3-4 per month**
- **Drugs- PHB 120, Cloba- 10mg, VLP- 1000**
- **Semiology: stiffening of Left UL with deviation of head to Left and then generalization**



- **EEG: jun 99 (pvt): LRE Right temporal region**
- **VEEG-LTV- 45-10- s/o 4 CE characterized by Ictal onset F8 T4 in 3 and F8 F4 in one.**
- **MRI: pvt-2009- - Large inferior right frontal lesion**
  - ? DNET ??Cortical dysplasia**
- **CT head: 2009: right frontal hypodense enhancing mass**
- **SPECT: not done**