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Presurgical Assessment of Patients with Refractory Temporal Lobe Epilepsy

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1. Introduction

The consideration of temporal lobe epilepsy (TLE) surgery for those with medically refractory seizure disorders requires a well-functioning multidisciplinary team and a systematic approach to the candidates, with the aim of advising patients of their chances of being seizure free following surgery, and the risks of any procedure. It is crucial that patients and their families are given realistic expectations of what may and may not be achieved with surgical treatment, and that long-term follow-up is maintained post-operatively.

Complex partial seizure (CPS) of temporal lobe is the most common type of seizure disorder, and approximately 70% of patients are referred for surgical treatment.

In this chapter we want to discuss the different forms of presurgical evaluation and patient selection. The patients that are considered as possible candidates for epilepsy surgery, need to have a detailed clinical history and demonstration of drug resistant-epilepsy.

We assume that the issues about clinical history have been mentioned in previous chapters, so our approach will start from the identification of the epileptogenic zone (I. Irritative zone, II. Seizure-onset zone, III. Symptomatogenic zone, IV. Epileptogenic lesion and V. Functional deficit zone) and patient selection using different diagnostic techniques, that include:

- Semiology
- Neurophysiological criteria
- Neuropsychological evaluation
- Intracarotid Amobarbital Procedure (IAP), and
- Neuroimaging

2. Semiology

In assessing the clinical semiology of the syndrome of TLE, in medical literature, the first thing to take into account, is that symptoms originate from different areas of propagation of the discharge located in other lobes, rather than the temporal lobe itself.

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Many of the symptoms observed in ictal and postictal mesial temporal sclerosis (MTS) can be attributed to typical patterns of propagation from the mesial regions (automatisms possibly caused by activation of cingulate gyrus, postictal aphasia due to Todd paralysis from the temporal basal area and/or Wernicke's area, etc.) and lateralizing signs seen in the mesial temporal seizure on-set zone are also an expression of the propagation of adjacent brain regions [basal ganglia for contralateral dystonia, areas of language for postictal aphasia, ipsilateral motor area for contralateral version and clonic seizures, etc.] (Lüders, 1999). These manifestations do not reflect per se the cause. That is why this chapter will not mention the propagated symptoms, which help in the clinical context of the lateralization and location of the seizure, but are not part of typical semiology of TLE.
The symptomatogenic area (cortical area that generates symptoms) will be mentioned, but not the epileptogenic area or the preictal and postictal symptoms, which are another semiological feature of TLE (Andrade, 2011).
TLE in children represents a different scenario and a less homogeneous syndrome when compared with its manifestations in adults, and also the process of brain maturation modifies the ictal semiology in both groups. In children under 6 years of age the manifestations are predominantly motor, resembling frontal lobe seizures (Ray, 2005). In adults the auras and automatisms are the most frequent semiological expressions. That is why the signs found in the adult TLE syndrome will be discussed and studied more profoundly in this chapter.
It is important to remember that the diagnostic value of the waking semiology of seizures of TLE is similar to dream semiology (Rodriguez, 2007).
Ictal behavior is sporadic, and an anatomo-clinical correlation is difficult to establish, as opposed to when there is a neurological injury which leaves a persistent deficit.
In the adult population, the temporal lobe seizures are the most commonly presented, and the ones involving mesial structures, representing the classic complex partial seizures (CPS), dominate in the clinical setting. Many of them are preceded by auras (interpreted as seizures), but these may frequently be presented independently.
The temporal lobe can be divided anatomically and functionally in structures:

- Mesiobasal limbic area: The mesial (union of "medial" and "basal" regions) comprise the anterior amygdala to the hippocampus, hippocampus, uncus and parahippocampal gyrus.
- Basal and lateral neocortical area: Lateral temporal cortex includes the gyrus of Heschl's as the primary auditory area and the area of Wernicke´s which is responsible for speech perception.
- Insular area: This region is usually not accessible to epilepsy surgery, for which its manifestations are less defined than the mesial or lateral semiology. The insula is essential for gustatory and autonomic functions.

The manifestations of the TLE may vary in presentation and behavior in relation to the ictal on-set zone and the symptomatogenic zone (Chiosa, 2010).
Taking into consideration the information above, we can divide TLE symptoms as subjective and objective.

2.1 Subjective symptoms or “epileptic auras”

Auras are part of the onset of seizures, occurring in up to 80% of the TLE cases and they can provide a high value to locate and lateralize the epileptogenic zone. However, the aura may

only represent a part of the propagation of the discharge. Within the phenomenology of the aura symptoms are psychic, autonomic, sensory and sensory special.
According to its most frequent anatomical location, they can be divided into 3 groups:

- Mesio-temporal auras, including psychic symptoms such as dysmnesic, affective and cognitive auras.
- Mesio-lateral auras are originated in the mesial temporal-insular network and include autonomic auras as epigastric sensations, with or without other autonomic signs or symptoms.
- Neocortical auras, in which its commitment suggests a temporal and extratemporal neocortical compromise. Include auditory, somatosensory, visual and dizzy auras (Castilho Garcia, 2010).

2.2 Subjective phenomenon

Psychic or experiential auras can be subdivided into dysmnesic, affective and cognitive. It can generate different phenomena: deja vu (already seen), jamais vu (never lived), pleasure, anger, fear, living in another dimension or being transported to another place, structured visual hallucinations associated with pleasure or displeasure, depersonalization, state of "reverie", distortions of time, forced thinking (although these may occur more frequently in frontal seizures) (Andrade, 2011).
The psychic auras can be divided into the "experiential" or "interpretive" phenomena.
Gloor (1990) summarized the characteristics of the "experiential" responses as follows: (a) Can be experienced or intrusive from the past; (b) There is a sense of familiarity or reminiscence (déjà vu, déjà vécu); (c) sense of living a dream; (d) the patient says he is always aware of the incongruity and illusory experience; (e) affective states like fear, anger, sexual arousal are common; (f) these responses have some lack of features such as absence of progression over time and scenes that do not evolve; and (g) auditory hallucinations are said to be felt without semantic content (Kasper, 2010).

2.2.1 Dysmnesic auras

Amnesia of the event can be found with the phenomena of déjà vu and Jamais vú (feeling of familiarity and unfamiliarity) or déjá entendú o Jamais entendú. "Deja vu" refers to a feeling of familiarity associated with the present context; jamais vu is a rare symptom and is a false sense of unfamiliarity.

2.2.2 Affective auras

Patients may have different emotional symptoms, including anger, hatred, contempt, shame, joy, love, excitement, fear. Fear occurs frequently in TLE, but it is also described in frontal onset epilepsy (Biraben, 2001; Manford, 1996).
Ictal fear can easily be confused with a psychiatric disorder, and a definite link with panic disorder may not be fully defined. It has been identified that patients with TLE have an increased incidence of panic disorders (Mintzer, 2002).
Panic attacks with autonomic and behavioral changes (agitation, paralysis, thinking terror, asking for help) have been described, and recordings of deeper EEG electrodes have shown that the areas involved are located in the orbital-prefrontal regions, anterior cingulate gyrus and limbic temporal cortex, and more frequently in the nondominant hemisphere (Biranben, 2001).

2.2.3 Cognitive auras

These are situations in which the perception of internal or external reality is altered. The main cognitive auras are: distortion of the appreciation of time, sense of unreality, depersonalization, changes in body image and forced thinking, which consists of imposed and intrusive thoughts at the beginning of the seizure (Parra, 1999; Fernádez-Torre, 2002).

2.2.4 Interpretative auras: hallucinations and illusions

Based on new studies with stereoelectroencephalography it is clear that there are specific anatomical locations for many hallucinatory states. Specific anatomical areas cause elementary hallucinations that begin in the visual and auditory cortex. For the occurrence of complex hallucinatory states to occur, the involvement of the limbic cortex is a prerequisite.
Generally, elementary hallucinations correspond to the activation of primary sensory areas (eg buzzing, with the involvement of the activation of the Heschl's gyrus, and if unilateral it indicates a contralateral origin) and complex hallucinations (such as music, voices) have a more extensive tracking and seem to correspond to the activation of association areas. For example we can see visual changes in shape, size (macro and mycropsia), color (acromatopsy), movement and distance, hearing impairment in the perception and understanding of volume, tone and character of sounds.
Sensations that are less frequent include gustatory and olfactory auras and unilateral headache. Olfactory sensations can be pleasant or unpleasant, which is thought to be originated in the amygdala. The gustatory auras are rare and are usually described as an unpleasant taste and are associated with insular onset (Chiosa, 2010; Hausser-Hauw, 1987; Fried, 1995).
The olfactory auras are known as ´´uncinate crisis´´ and its origin seems to be in the mesial region close to the amygdala or orbitofrontal regions. The most common underlying diseases are tumors in the temporal region (Archarya, 1998).

2.2.5 Autonomic phenomena

They are produced by a compromise of the amygdala, insula and spread to the hypothalamus.
The seizures originating from the mesial area characteristically begin with a “limbic” aura characterized by epigastric discomfort or abdominal auras, which can be described as a rising sensation, nausea, "butterflies in the stomach," or the feeling of being in an elevator. Although this type of aura is correlated with onset mesial temporal crisis, its exact location between the mesial temporal structures is still controversial (Wieser, 1983; Gloor, 1990; French, 1993; Van Buren, 1963; Henkel, 2002).
The epigastric aura is more common in the mesial TLE subtype that in the lateral TLE, but is not exclusively of the temporal lobe. In all patients with an abdominal aura followed by automatisms, epilepsy is located in the temporal regions (Henkel, 2002).
These auras can be associated with autonomic characteristics (skin color, heart rate, blood pressure, pupil diameter, piloerection, sweating) or perceptual manifestations such as anxiety (amygdala commitment). Extreme bradycardia and even atrioventricular block has been described when compromising autonomic control centers at the temporal lobe. As a general rule, the autonomic manifestations of temporal lobe seizures such as ictal vomiting, urinary urgency, spitting, tend to lateralize to the non-dominant hemisphere for language (Ostrowsky, 2000). Ictal vomiting may be the only manifestation of simple partial seizures, and has been linked to temporal lobe seizures in the non-dominant hemisphere (Kotagal, 1995; Devinsky, 1995).

2.3 Objective phenomenon

2.3.1 Awareness commitment

This is the phenomenon most frequently found. It is considered as the individual's inability to interact appropriately with the environment, with confusion or amnesia of the event, activity inhibition and automatic behavior. CPS could last from seconds to minutes, but in the limbic status from hours to days, where a patient walks around with an automatic behavior, and then not remembering the event. Over 50% of seizures with impaired consciousness were associated with motor events, purposeless, stopping the activity that the individual was carrying out, and this crisis can be unilateral or bilateral (Andrade, 2011).

2.3.2 Automatisms

They occur in 40% to 80% of patients with TLE. They are stereotyped, as involuntary oral and manual movements during the crisis. They can be oroalimentary (swallowing, chewing, tasting, pressing the lips, spitting, drinking water, etc.), gestures (touching, scratching, manipulating objects in the environment surrounding it or touching people), simple or complex (recall a human action, such as unbutton, rub your fingers, cross themselves, pray, clap, walk, etc.), or mimicking (crying, laughing, etc.) (Serrano-Castro, 1998; Ebner, 1995).

Oral automatisms originate more frequently in the amygdala and anterior temporal region. It is an involuntary motor activity where only in 10% of cases, awareness, is not affected. When the right hemisphere is involved the patient is usually amnesic. These automatisms are less violent than those evidenced in frontal lobe seizures. Unilateral dystonic postures may occur, reflecting a compromise of the basal ganglia and can help to lateralize the onset to the contralateral hemisphere. A typical association of ipsilateral hand automatisms and contralateral dystonic posture is often found in temporal lobe seizures.

2.3.2.1 Complex automatisms with unique characteristics for their localizer or lateralizing value

- Spitting: It is associated with right temporal lobe seizures, although there are documented cases with an origin in the left temporal lobe. There are reports with coughing up, an automatism with right temporal lobe onset. Until 1999 the literature reported 17 cases, 12 of which began in the non-dominant temporal lobe (Kaplan, 1999).
- Sex Auras: It is pleasant erotic thoughts or feelings. May be accompanied by orgasm. Occur predominantly in women, are most frequently associated with right compromise in TLE (Aull-Watschinger, S., 2008)
- Sign of the Cross: A rare sign of presentation. The patient touches his forehead, chest and hands crossed back and forth touching the shoulders; described in right TLE (Lin, 2009).
- Speech automatism: The patient says phrases, words or verses (automatic language). Can be verbal or nonverbal. (Andriani Rahal, 2006).
- The postictal yawn can occur in up to 2% of patients with TLE and always in the postictal period (Kuba, 2010).
- Gestural automatisms directed to the cephalic portion of the body: movements directed to the nose, face, cheek and head displayed during the crisis, to separate them of the most common, occurring in the interictal and postictal periods, usually seen in patients with temporal seizures (Meletti, 2003).

- Postictal dysphasia, and "nose-wiping": The postictal dysphasia lateralizes the focus to the language dominant hemisphere and the classic sign of "nose-wiping" to the ipsilateral focus in 90% of cases when it is present (Loddenkemper, 2005).

2.3.3 Disorders characterized by non-automatic movements

In TLE, when it involves the posterior and superior temporal gyrus, the patient displays a horizontal motion and sudden eye movement, with its fast phase directed toward the side contralateral to the epileptogenic zone (epileptic nystagmus), although they can also be seen in front crisis.

The insula seizures deserve special mention. It has been shown that in this area there is a major somatosensory, language and movement integration.

By stimulating the insula, patients may describe sensations such as numbness, pins and needles, electricity, heat, feel that they have air inside them. The areas most frequently involved are the naso-pharyngeal, cervical region and limbs.

Most of the time the sensations are evoked contralaterally to the site of stimulation but they may also be bilateral. According to the frequency of symptoms reported, there is involvement with the viscera; causing nausea, vomiting, hot ascending feeling, gustatory sensations and facial flushing. Other symptoms may occur as the elevation of contralateral limb and eye movements that impede fixation of an object in the visual field, as well as bimanual and hyperdrive automatisms.

Stimulation can evoke an auditory response referred to as sound going away, hearing an echo, or having their ears plugged. There is sometimes vertigo and sudden loss of speech.

It is interesting to note that in the insula exists a somatotopic, tonotopic and viscerotopic map: In the anterior insula there appears to be a visceral integration, somatotopic to the area of the face (eyes, nose, mouth and neck) and partial integration of eye movements, while the posterior region integrates related somatic sensations with foot and leg movements and the antero-superior region exists proprioceptive, vestibular and language integration in the dominant hemisphere (Ostrowsky, 2000).

2.4 Differentiation between mesial and neocortical onset of the seizure

Making this differentiation is essential to find the epileptogenic zone, necessary for surgical decision making in the case of drug resistance. There are several studies that attempt to find such differences that are named as followed: complex partial seizures with secondary generalization are more frequently of cortical origin, but simple partial seizures may also occur.

Temporal lobe seizures may have a more gradual onset, gradually developing during the course of a minute and at the same time having a longer duration of the ictal and postictal period compared to extratemporal seizures (particularly frontal onset).

3. Neurophysiological criteria: ictal EEG and interictal EEG

3.1 EEG-video monitoring and technical considerations

The continuous, non-invasive EEG-video monitoring is a technique that uses both electroencephalographic and video features for electroclinical correlation. This diagnostic tool has been used by most authors as the gold standard for the study of patients with refractory epilepsy. For the temporospatial analysis of the electroencephalographic activity,

an arrangement of electrodes is placed on the patients scalp, following the standard method known as the international 10-20 system (Jasper, 1958). To increase the sensitivity of the diagnosis, and to analyze certain EEG abnormalities it is necessary to use non standard locations such as sphenoidals, foramen ovale, anterior temporal (T1/T2), nasopharyngeal, zygomatic and mandibulary electrodes. (Morris, 1987; Silverman, 1960). When surface EEG shows non-conclusive results, the ictal semiology is atypical or the neuroimaging studies are normal, it is necessary to use invasive recording techniques, using subdural electrodes or intracerebral depth electrodes. (Dubeau, 2000; Noachtar, 2009).

3.2 Interictal abnormalities in temporal lobe epilepsy

It´s possible to observe focal temporal or regional persistent slowing, if it´s associated with structural alteration or intermittent when it´s associated with a ´´functional´´ alteration, which can indicate a subjacent epileptic zone (Mohammed, 2010). Temporal intermittent rhythmic delta activity (TIRDA) is a good interictal indicator of temporal lobe crisis. It´s arrhythmic variant can also be present even thought is less specific. TIRDA has had more association with MTLE (Geyer, 1999; Koutroumanidis, 2004). Spikes or sharp waves with an electronegative peak over the anterior temporal region (F7/F8) are the main interictal abnormality in TLE. For surgical purposes, it is necessary to establish the difference between mesial origin (MTLE) and a neocortical origin (NTLE).

In MTLE the surface recordings show spikes and localized sharp waves mainly in sphenoidal electrodes or in the anterior temporal region, while in NTLE discharges are observed more frequently in the mediotemporal (T3/T4) or posterior electrodes (T5/T6). There are studies that try to establish differences in the irritative epileptogenic activity between MTLE and NTLE showing overlapping results of both entities. Pfander et al., and O'Brien et al. Couldn´t find differences in the interictal EEG in patients with MTLE or NTLE (Lüders, 2008).

Similarly, there haven't been any differences found between both epilepsies subtypes of the temporal lobe with respect to the lateralization of the irritative epileptogenic activity.

3.3 Ictal abnormalities in temporal lobe epilepsy

Multiple ictal patterns exists, the ones that stand out are irregular 2-5 Hz lateralized activity, background attenuation, start-stop-start phenomenon, 5-10 Hz sinusoidal waves and repetitive epileptiform potentials. Similarly is not possible to observe an EEG change, being minimum or presenting an increase in heart rate just before the onset of ictal EEG discharges, the latter associated with an origin at mesial temporal structures (Ebersole, 1996; Vossler, 1998; Walczak, 1992; Di Gennaro, 2004).

Multiple studies have investigated the differences between the patterns found in MTLE and NTLE. For example, Lüders (2008) describes an increase in frequency of rapid sharp rhythmic waves (>4Hz) in the ictal EEG of patients with MTLE, while patients with NTLE develop bilateral ictal EEG changes more frequently and rapidly. Dantas (1998) demostrated that the EEG is important to lateralize the epileptogenic lobe, considering the rythmic ictal activity and the postictal findings. Nevertheless, is not possible to determine with certainty the difference between MTLE and NTLE. Dericioglu (2008) found in patients with MTLE thirteen different ictal onsets, being the most frequent the cessation of interictal discharges followed by ipsilateral delta-theta temporal rythmic activities.

Ebersole (1996) showed that the rhythmic waves of 5–9 Hz that are first seen in the temporal region during seizures, are specific to MTLE, but he also found rhythmic waves of various frequencies from the delta to beta band in both MTLE and NTLE, indicating that these are not specific to MTLE.
Sakai (2002) concludes that scalp EEG activity during the ictal period could provide information with which differentiation between MTLE and NTLE can be deduced with high accuracy. However, they provide an incomplete assessment of laterality in MTLE. Others have also reported that EEG seizure patterns of MTLE and NTLE patients showed no differences (Gil-Nagel, 1997; Saygi, 1994; Gates, 1990).

3.4 Invasion for temporal lobe epilepsy

There is debate in the epilepsy group around the world about the risk of the use of depth electrodes located in both hippocampus, to lateralize the ictal-onset zone in patients with medial TLE and previous discordant results. The reason is that while it provides greater accuracy for lateralizing the epileptogenic zone, this would generate a potential damage to the healthy hippocampus, which may lead the patient to have consequences from the neuropsychological and / or neurophysiological points of view. However there are studies that conclude the safety of both, postoperative and in the long-term monitoring. When there is reasonable evidence that the patient has a resectable epileptogenic focus, but the information obtained non-invasively is imprecise and requires more specific data, it is necessary intracranial electrode implantation. In MTLE, surface video-EEG does not give a definitive lateralization in all cases and some of them lead to a false lateralization (Alssadi, 2001; Sammaritano, 1987; Napolitano, 2010; McIntyre, 2008; Napolitano, 2008). Therefore it is necessary for some patients with TLE on surface video-EEG to require intracranial recording, particularly when neuroimaging does not help to locate (Engel, 1993; Immonen, 2010).
In conclusion, surface video-EEG allows interictal and ictal patterns in patients with TLE to manifest. There are numerous electroclinical differences between patients with MTLE and NTLE, but none of them are sufficient to arrive to a clear distinction between both types of epilepsies. It is possible to differentiate lesional NTLE from MTLE based on features of the history of the crisis, sintomatology of the crisis and surface ictal EEG recordings. Nevertheless, many of the overlapping electroclinical findings make it necessary to realize invasive studies for the correct localization and lateralization of the epileptogenic zone.

4. Neuropsychological evaluation

For Kochen, Oddo and Solís (2003), the neuropsychological evaluation allows characterizing the cognitive aspects of patients with epilepsy, and in humans it confronts us with an in vivo model of the study of brain plasticity, making it an almost ideal model for the study of cognitive functions. Neuropsychological evaluation provides relevant information for the diagnosis, prognosis and rehabilitation of patients undergoing surgery for epilepsy (Drake, 2002). It allows:

- Assessing the current cognitive status, knowing the possible effects of epilepsy and/or consumption of long-standing AEDs on cognitive function. In the case of patients with TLE, the prevalent cognitive functions are memory and language in particular the denomination.

- Obtain data to draw conclusions about lateralization (right hemisphere, left hemisphere) and the location of the neuropsychological symptoms and their possible correlation with ictal onset zone.
- Predicting cognitive potential risks of surgery. In the case of temporal lobe epilepsy might be, first, a memory deficit that may range from a global amnesia, to amnesia for specific material, or a reduction of premorbid performance level. On the other hand a deficit in language, more specifically an anomia with varying degrees of severity. The compromise is explained by the structural and functional relationship of the hippocampus with memory system, and that is functionally lateralized, the left hippocampus related with memory for verbal material (the decrease or loss is well documented in cases of left temporal lobectomy) and the right for visuospatial memory (the decline is not so consistently documented as the previous one). This point has an important predictive value in surgical decision and should be part of the information given to the patient and the family, which also will help in surgery related decisions. Another important consideration, and well documented, is related to the preoperative level of memory performance and magnitude of the possible postoperative amnesia. Thus, patients with adequate preoperative performance on tests of verbal memory (left temporal), are at increased risk of amnesia after surgery when resection was performed in the dominant temporal lobe.
- To establish current and future needs of rehabilitation. Current because the cognitive status might now draw cognitive symptoms that are affecting the performance level of the patient and future because the characteristics of the surgery or any of its possible complications could cause sequelae susceptible of rehabilitation.
- To compare the neuropsychological performance before and after surgery, determining what functions remain intact, which have been improved and which have been deteriorated and then required to redirect the rehabilitation.

The evaluation protocol must be general and specific, covering if not all, the majority of cognitive functioning; it must also contain more specific and concrete tests for dysfunctional issues. This implies the need for an extensive protocol that meets the characteristics specified, including assessment of intellectual capacity, attention, memory, gnosis, praxis, language and executive functions.

Certainly, the neuropsychologist must have an extensive knowledge of cognitive activity, its development and its different forms of alteration, so that you can, from the patient's history and performances in the different tests, make a reading beyond quantitative psychometric data. In this regard, and to maximize the results of neuropsychological assessment it is necessary to manage a variety of knowledge related to: 1) functional brain asymmetry, 2) asymmetry in case of the injury, 3) performances suggestive hemispheric damage, and 4) brain plasticity.

First, in terms of functional brain asymmetry, according with Ardila & Rosselli (1992) indicate that for the majority of people, regardless of whether they are right or left handed, the dominant hemisphere for logical linguistic aspects is the left hemisphere. Thus, interhemispheric differences exist not only in the type of information processing, but how it is done. While the left hemisphere processes logical, linguistic, and temporal information, the right hemisphere does so with emotional information, rhythm, image, color and Spatial. The first processing (left hemisphere) is sequential, serial, symbolic and analytical; the second (right hemisphere) is simultaneous, parallel, rhythmic, melodic and emotional.

Given this, each hemisphere is involved in cognitive function in an unequal manner, each one performs different cognitive tasks but are complementary, thus, no function seems to be completely independent of the activity of a single cerebral hemisphere, and then the interaction of the both hemispheres is essential for proper operation. In addition, hemispheric specialization grades vary from one person to another and interact with variables such as sex, education, training, age at which the patient suffered the injury, characteristics of the crisis and extent of the injury.

Second, asymmetries in case of brain injury. The above information essentially leads to understanding how an injury to the left or right hemisphere leads to different changes, even within the same cognitive activity. Therefore, if there is a language disturbance found, the left injury would cause aphasia, with varying degrees of alteration in phonological, morphological, syntactic, semantic aspects corresponding with their reading, writing and arithmetic alterations. In a right injury it would be aprosodia, with the possibility of finding agraphia, alexia and acalculia, moreover, changes in paralinguistic aspects may be found. If praxis are affected, in left lesions ideomotor apraxia will be found and in right lesions visoconstructional apraxia; if it alters the corporal perception, in left lesions will find autotopoagnosia and in right lesions hemisomatoagnosia; if it alters the memory, in left lesions will encounter verbal amnesia and in right lesions nonverbal amnesia; in terms of affection, if the injury is left, the patient will have a catastrophic reaction, by their awareness of the defect, but if there is a right injury the reaction will be of indifference, which is closely related with their anosognosia.

Third, executions suggesting hemispheric dysfunction. In this case, for example, behavioral and emotional changes, emotional mutism as apathy, disinterest, concrete thinking, lack of spontaneity and initiative, adynamia and catastrophic reaction against the deficit suggest left frontal lesions, whereas symptoms like behavioral disinhibition , hyperreactivity, childish behavior, pseudo-psychopathy, inability to judge emotions, behavioral inadequacy and indifference reactions to the deficit would be indicative of right lesions.

Also associative agnosia, right - left disorientation and finger agnosia involve left lesions, but apperceptive agnosia, a non verbal auditory agnosia, unilateral Spatial agnosia and hemisomatoagnosia would be indicative of right lesions, visoconstructional apraxia with features like: scheduling problems, simplification of the drawing, lack of internal details, microforms, copy from left to right, would suggest a left side injury.

By contrast, features such as visuospatial problems (rotations, inversions), complexity of design, lack of perspective, better picture to the right side macroreproduction and copy from right to left, would suggest a right injury. Corporal apraxias would be indicative of left lesions, while the dressing apraxia would be right. Problems in thinking processes as: concrete, nominalism, syncretism, animism, alterations in solving verbal problems and poor verbal fluency, indicate left lesion, whereas failure in visuospatial problems and poor design flow, would point to right side injury.

Fourth, the concepts of brain plasticity. According with Aoki & Stekevitz (1988), brain plasticity is the ability to renew or reconnect neural networks in order to perform new tasks and adapting, recovering lost function after an accident or an injury, in some degree, the brain is constantly changing. The recovery of function does not occur all of a sudden, it is a slow change following a logical sequence. According with this, the brain can continue to develop in adulthood. Brain plasticity has physiological and biochemical bases, which explains why when a brain injury occurs during the first years of life, the opposite hemisphere and healthy adjacent areas can assume the functions involved. According with this, patients with epilepsy with early brain damage can have reorganization of functions by brain plasticity.

Neuropsychological evaluation provides the differential diagnosis of temporal and extratemporal epilepsies (Maestu, 2000), from cognitive deficits, which also is a differential predictive value. Patients with extra-temporal neuropsychological signs or diffuse brain disorder, have a worse prognosis for seizure control after surgery, but on the contrary patients with cognitive alterations lateralized and localized, have a greater predictor of seizure control, if it includes the existence of a single focus, such as medial temporal discharges restricted to one hemisphere would have a positive predictive value of seizure control (Jones-Gottman, 2000; Rausch, 2001). It is also important to consider that in patients with TLE there can be frontal functions affected, which can be explained by the connections between the two lobes (Orozco-Giménez, 2002).

Another fact that becomes important is the multifactorial nature of cognitive impairments. There are different variables, such as the etiology of seizures, drug treatment, age of onset, seizure type, duration of illness, seizure frequency and academic and cultural limitations, therefore, the cognitive impairment in this disease is variable and may be, from very severe, or not identify significant changes. Thus, in people with mental retardation there can be found immature development of certain cognitive domains, whereas in patients where the epilepsy is the manifestation of a focal brain disease, defects in memory may be the only evidence of cognitive alterations. All these turns the neuropsychological evaluation in a multivariate process that requires several hours to complete, but it is essential to know the patient's mental status (Oddo, 2000; Drake, 2002).

5. Intracarotid Amobarbital Procedure (IAP)

The Intracarotid Amobarbital Procedure (IAP) or Wada Test is considered the gold standard for lateralization of language and memory functions in patients who are candidates for epilepsy surgery (Sharan, 2010; Baxendale, 2009; Spencer, 2000; Trenerry, 2006). The interpretation of the IAP results is presented in terms of hemispheric dominance: right, left or bilateral / mixed, taking into account that the cerebral organization of cognitive functions is complex and that this complexity can be increased even further in refractory epilepsy, due to the effects of brain plasticity and atypical cognitive organization presented in focal brain lesions. Additionally, they must take into consideration the individual and gender differences that may occur in the hemispheric lateralization of functions.

Given its invasive nature, frequency of complications of the IAP ranges from 0 to 10.9%, those are mainly related to the angiography and not to the Amobarbital injection (including: encephalopathy, seizures, stroke or transient cerebral ischemia and hemorrhage at the catheter insertion site), but most centers report rates below 1% per year and only 0.36% of permanent morbidity (Sharan, 2010).

All the above considerations require a solid theorical knowledge and clinical experience of the neuropsychologist, neurologist and neuroradiologist involved in the IAP.

The importance of the IAP in the presurgical evaluation of patients with temporal lobe epilepsy lies in: 1) determining the cerebral representation of language, 2) identify patients at risk of decline in memory or amnesia after surgery and 3) provide information about lateralization and localization of the epileptogenic zone (Trenerry, 2006).

5.1 Intracarotid Amobarbital Procedure and cerebral representation of language

The value of the language test during the IAP depends in first instance of the concept we have of language. This is a universal competition which implies the existence of symbols

and signs, represented mentally; this is a cortical process, expressed through speech. During the IAP, when injected into the dominant hemisphere for language, it could produce an alteration of language that can range from global aphasia to a complete expressive and partial receptive aphasia, or vice versa, or only partially one of the two.
The interpretation of the results can be affected by cross-dominance or right hemispheric dominance for language. It is unclear the effect of atypical dominance in neuropsychological tests, as there are no pathognomonic indicators of this one in traditional batteries. Almost 6% of healthy people may have an atypical language representation; condition even more evident in people with developmental disabilities or chronic illnesses such as epilepsy. In fact, nearly 30% of people with epilepsy have altered language lateralization (bilateral hemispheric representation or variability in dominance). This atypical representation for language is more common in left-handed patients with epilepsy and those with: extrahipocampal structural or functional compromise, early onset of epilepsy, short latency period between the initial precipitating injury and the onset of the seizures, and the presence of epileptiform discharges interictal temporal bilateral and extratemporal in the EEG (Sharan, 2010; Trenerry, 2006).

5.2 Intracarotid Amobarbital Procedure and cerebral representation of memory

The IAP as a technique for assessing cerebral representation of memory was introduced by Milner in the fifties, to predict whether the contralateral hemisphere to the epileptogenic area could support memory functions after temporal lobectomy. If after unilateral anesthesia, ipsilateral to the focus, the patient could remember the events immediately following, it could be concluded that the contralateral hemisphere could support memory function, and consequently, surgery does not involve risk of postoperative amnesia (Milner, 1966).
One of the main applications of the IAP is that the asymmetric execution of the memory can predict postoperative memory impairment. This prediction is based on the concepts of "functional reserve" and "functional adaptation" (Chelune, 1995; Chelune, 2001; Loring, 2001). Although several studies report that the results of the asymmetry of memory on the IAP are associated with the postoperative prognosis of crisis management, the results are not consistent and this could be explained by additional clinical factors, differences in the interpretation of the asymmetry in the procedure and dissimilar postoperative follow-up times (Yu, 2010; Trenerry, 2006).

5.3 Lateralization and localization of epileptogenic zone with the Intracarotid Amobarbital Procedure

The hemispheric asymmetry scores in language and memory in the IAP has a close relationship with the side of seizure onset in patients with TLE, especially with structural abnormalities in MRI´s. Its diagnostic accuracy for identifying the epileptogenic area is 80-98%. Thus patients show a poor performance on the IAP during injection into the contralateral hemisphere to the focus.
By contrast, the ipsilateral injection to the focus, being functional the opposite hemisphere, will not produce more deficits than that reported in the baseline. It should be mentioned that, while it is true that the lateralized value of the IAP is not one of its major applications, it is particularly important when other presurgical evaluations are not conclusive, providing information that may even obviate the need of more invasive intracranial EEG records (Sharan, 2010; Trenerry, 2006).

5.4 Non-Invasive alternative to the Intracarotid Amobarbital Procedure: Functional Magnetic Resonance Imaging (fMRI)

Among the new imaging techniques available for the preoperative evaluation of patients with refractory epilepsy, who are candidates for surgery, Functional Magnetic Resonance Imaging (fMRI) is perhaps the most promising alternative in our scenario. Unlike the IAP which is a test of inactivation, fMRI is an activation test to visualize areas of regional brain activity in response to cognitive paradigms. fMRI is a noninvasive alternative to electrocorticography and the IAP for mapping of eloquent cortex and language lateralization. With regard to language lateralization, studies report nearly 90% concordance between fMRI and the IAP. Although less widely studied, it is also in the process of validating the role of fMRI in predicting postoperative memory outcome and the localization of ictal activity (Paolicchi, 2008; Abou-Khalil, 2007; Kesavadas, 2008; Dupont, 2010).

5.5 Present and future of Intracarotid Amobarbital Procedure

In recent years, it has been proposed that fMRI is a noninvasive alternative to the IAP, however, this technique cannot answer the question of how a task could be executed if part of a particular hemisphere is resected, which itself is done by IAP. People question the validity and reliability of the IAP, and although there is no standard procedure for the IAP and fMRI, when addressing memory paradigms.

Predictive models of postoperative memory changes have major implications for preoperative counseling of patients. The amnesic risk cannot be neglected specially after surgery of TLE. For example, the ability to predict the development of an amnesic syndrome differs from the ability to identify patients at risk of significant impairment of memory for specific material, verbal or nonverbal, that although amnesic syndrome is not considered fully established, has the sufficient severity to limit vocational options and other aspects of quality of life for patients, that deserve the neuro-rehabilitation plan.

In our experience, the IAP provides valuable information not only for hemispheric dominance of functions, but also because in conjunction with other diagnostic evaluations can support the surgical decision regarding: lateralization, localization, cognitive risk, surgical planning, extent of resection, seizure management and neuro-rehabilitation (Galeano, 2011; Wada, 2008; Baxendale, 2008).

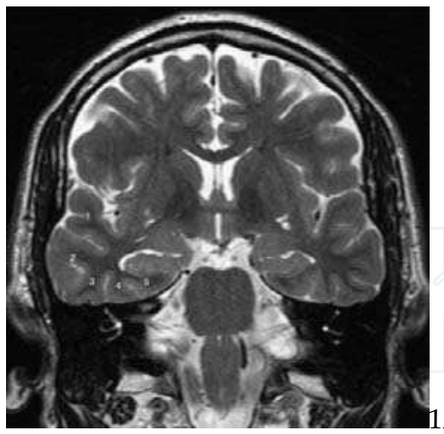
6. Neuroimaging

Neuroimaging is one of the current mainstays for the study, diagnosis, treatment and follow-up of patients with TLE.

Since Wilhelm Roentgen discovered X-rays in 1895, the most important milestones in the field of neuroimaging are given by the advent of computerized tomography [CT] (Hounsfield - Cormack) in 1971 and magnetic resonance imaging [MRI] (Damadian – Lauterbur - Mansfield) in 1976 (Shorvon, 2009; Canals, 2008). No less important is the development of functional MRI (BOLD) thanks to the work of Ogawa and Kwong in the early 90s (Ogawa, 1992; Kwong, 1992). Magnetic resonance spectroscopy (MRS) was introduced in the field of epilepsy around 1984.

6.1 Radiological anatomy

In the macroscopic examination of the temporal lobe magnetic resonance can recognize two surfaces: lateral, with the superior, middle, inferior and basal temporal gyrus, with fusiform and parahippocampal gyrus. They are delimited by the respective fissures: superior


1A

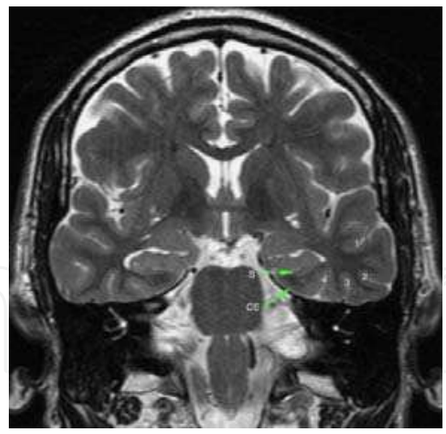

1B

Fig. 1. Normal hippocampus
1A. 1, Superior temporal gyrus. 2, Middle temporal Gyrus. 3, Inferior temporal gyrus. 4, Fusiform gyrus. 5, Parahippocampal gyrus
1B. 1, Superior temporal sulcus. 2, Inferior temporal sulcus. 3, Temporo-occipital sulcus.4, Collateral sulcus. EC, Entorhinal cortex. S, Subiculum

temporal, inferior temporal, temporo-occipital and collateral. The parahippocampal gyrus includes the entorhinal cortex and subiculum (Fig. 1).
Superior to the parahippocampal gyrus is the hippocampal formation (Ammon´s horn and dentate gyrus) which is divided into head (digitations), body and tail (Fig. 2). Anterior, medial and superior to the hippocampal head is the amygdala. Adjacent is the uncus, the medial reference of the temporal lobe.

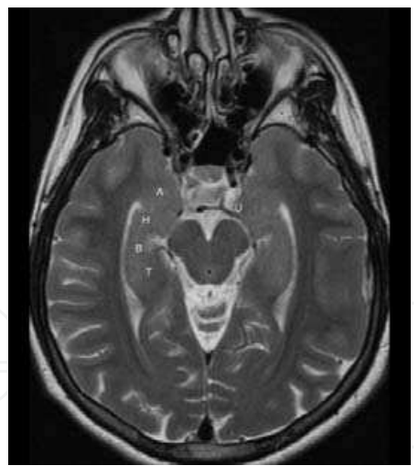
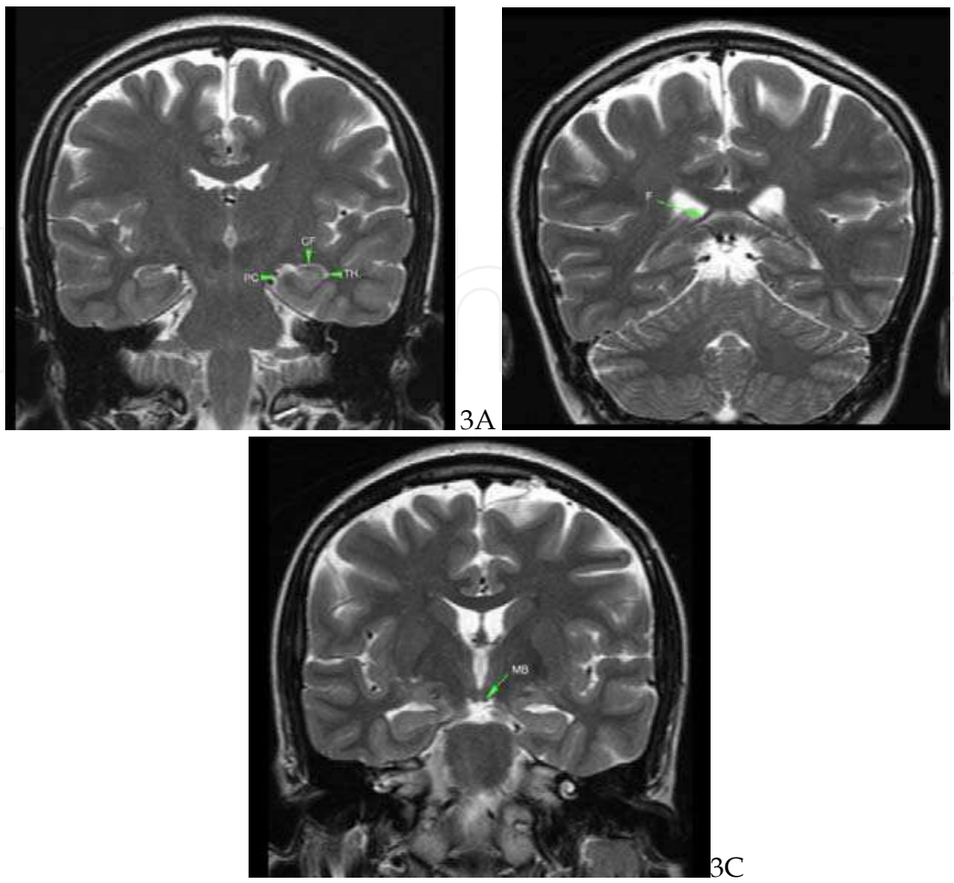


Fig. 2. Normal hippocampus
H=Head, B=Body, T=Tail, A=Amygdala, U=Uncus

Three structures with cerebrospinal fluid (CSF) surround the hippocampus: lateral, temporal horn, medial, the perimesencephalic cistern and superior, the choroid fissure. Hippocampal efferent system is evaluated with the fimbria, the fornix and mamillary body (Fig. 3). It Continues with the mamilothalamic tract that reaches the anterior nuclei of the thalamus and thence to the cingulate gyrus that culminates in the parahippocampal gyrus. It´s the so-called Papez circuit.



3A 3B 3C

Fig. 3. Normal hippocampus
A. PC = Perimesencephalic cistern, CF = Choroidal fissure, TH = Temporal horn
B. F=Fornix
C. MB= Mamillary body

Keep in mind that irrigation depends mainly on the posterior cerebral artery and in a smaller proportion, to the anterior choroidal artery.

6.2 Anatomical image

It refers to data from CT either sequential, helical or multidetector and magnetic resonance imaging (MRI) of low (<1.5 T), high (1.5-3 T) or ultrahigh field (> 3 T). In the diagnosis of refractory epilepsy we are not going to mention CT because of its poor performance (Bronen, 1996).

The MRI study should be done with high field equipment, with orthogonal planes to the axis of the hippocampal formation with high resolution antenna with the minimum possible slice thickness (Fig. 4). Protocol should include series of T1- weighted volumetric images that allows cuts to resolution of 1 mm (1.5 T) or 0.25 mm (3 T). The routine use of coronal FLAIR is not necessary due to artifacts and can generate false positives when it comes to assessing the hippocampal formation (Vattipally, 2006). Instead, we recommended the STIR sequence that implies the IR series in its acquisition, which is essential to review the cortex in cases of malformation of cortical development.

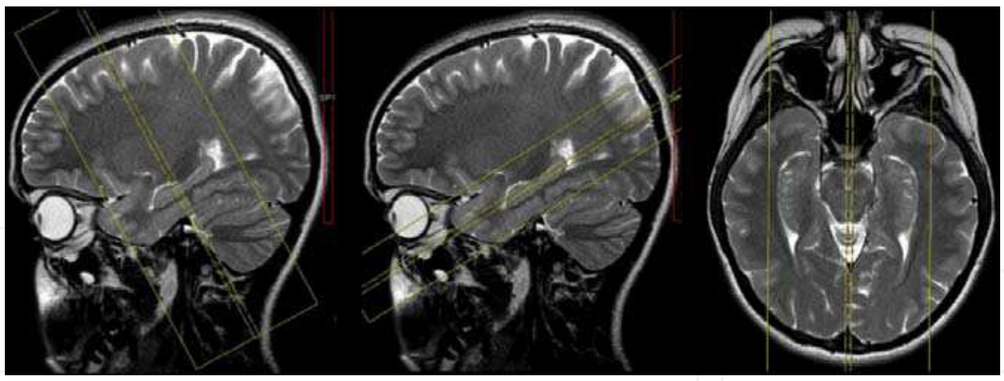
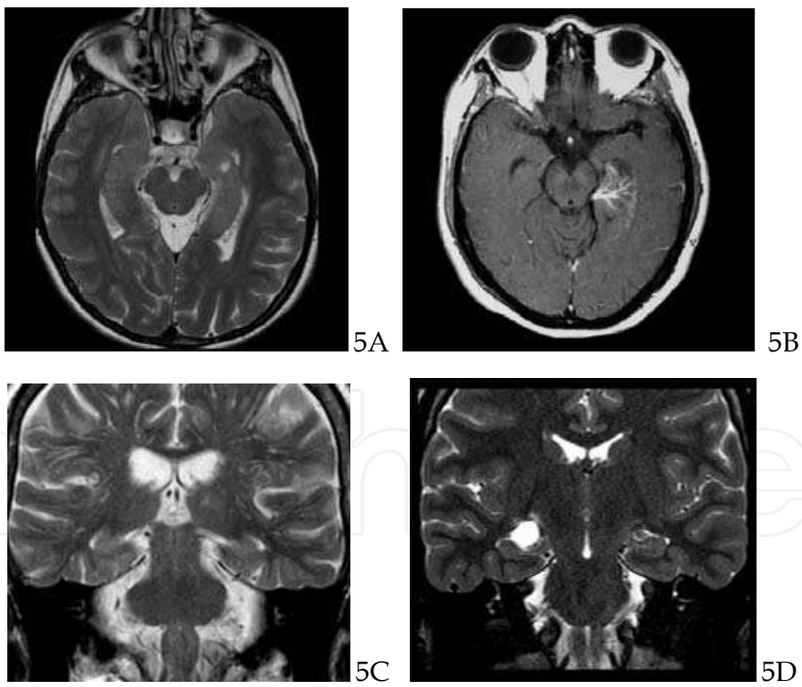


Fig. 4. Cutting planes. The diagram illustrates the orthogonal planes to be acquired in the coronal , axial and sagittal images of the hippocampus.

Due to the detail and the high number of images obtained (about 250) the neuroradiologist should interpret the results on a workstation that allows their proper handling.
Clinicopathological conditions are grouped into the following seven categories for descriptive purposes: variants, hippocampal malrotation, hippocampal sclerosis, malformations of cortical development, tumors, vascular lesions and gliosis.

6.2.1 Variants



5A 5B 5C 5D

Fig. 5. Variants

A. Cyst of the left hippocampal fissure
B. Anomaly of the venous development of left hippocampus
C. Generalized perivascular spaces
D. Cyst of the right choroidal fissure

About 10-15% of the population may have cystic remnants of the hippocampal fissure, a structure that separates the Ammon´s horn of the dentate gyrus. Is present from week 10 of pregnancy and regularly regresses (Kier, 1997) [Fig 5A]. The medial temporal lobe may be the place of venous developmental abnormalities, disorders have not shown causal effect in terms of epileptogenicity (Topper, 1999) [Fig. 5B]. In the temporal lobe perivascular spaces can be found, defined as round or oval structures in the MRI, sharply defined, isointense to CSF in all sequences, according to the route of the vessel and without mass effect (Ogawa, 1995) [Fig 5C].The presence of a temporal arachnoid cysts or cyst of the choroid fissure are not of great value except if they are of a significant size (Bronen, 1996) [Fig 5D].

6.2.2 Hippocampal malrotation (HIMAL)

Defined in 2000 by Barsi et al. as a group of ten characteristics: incomplete hippocampal investment with rounded shape, unilateral commitment, signal intensity and normal size, internal structure blurred, abnormal angle collateral sulcus, anomalous fornix size and position, temporal lobe size preserved, temporal horn enlargement and normal corpus callosum (Barsi, 2000) [Fig. 6].

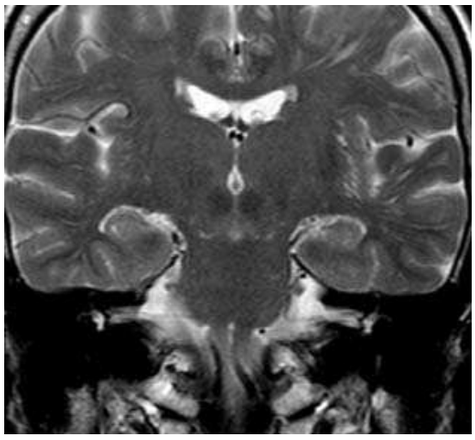


Fig. 6. Left HIMAL

In our experience we found that it can be focal or even bilaterally. This discrepancy can be explained in part by the original study methodology where they used low field resonators (0.5-1 T). Although the original description was present in 6% of 527 epileptic patients examined, it is unclear whether it can have a causal relationship.

6.2.3 Hippocampal sclerosis (HS)

Is the most common pathologic substrate in TLE in most of the series and the MRI is the most sensitive and specific for detection in vivo. Although it may be bilateral or symmetrical, it is more often unilateral or asymmetric (Fig. 7A). Among the characteristic findings are atrophy, loss of internal architecture and the increase in signal intensity on T2 series (Wieser, 2004). It has been proposed, however, that when there is no obvious changes of atrophy and signal alterations, complete loss of hippocampal head fingering has a sensibility of 92% or a specificity of 100% for the diagnosis (Oppenheim, 1998) [Fig. 7B].

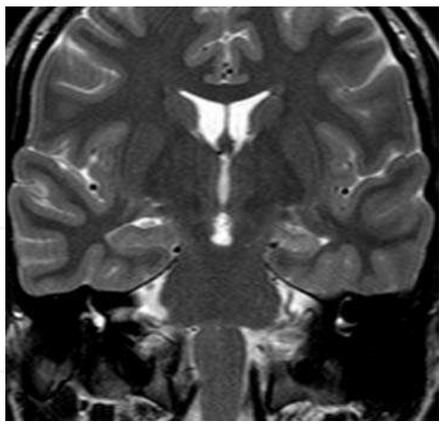
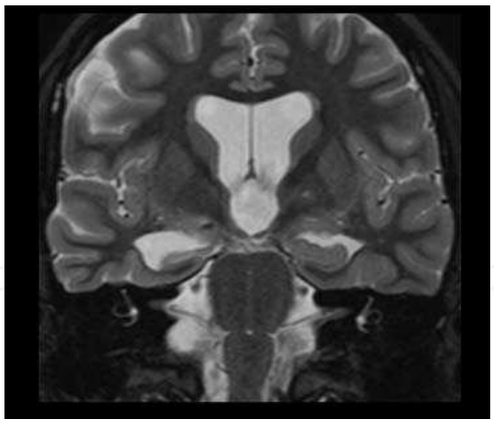

7A

7B

Fig. 7. Hippocampal Sclerosis
A. Atrophy and hyperintensity (T2) of the hippocampal formation
B. Loss of the digitations of right hippocampus head

In many cases ipsilateral extra-hippocampal findings can be observed as:

- Atrophy-signal alterations of the temporal neocortex, temporal white matter, amygdala, fornix, mamillary body, thalamus and basal frontal cortex.
- Atrophy-signal alterations in the contralateral hippocampus
- Dilatation of the ipsilateral and contralateral temporal horn.
- Diffuse ipsilateral hemispheric atrophy (Wieser, 2004; Ng, 1997; Deasy, 2000; Oikawa, 2001)

In approximately 15-20% of cases of HS can be associated with other extra-hippocampal epileptogenic entity, between which are malformations of cortical development (eg. cortical dysplasia) and gliosis in the first instance. It's called dual pathology (Palacios, 2008).
Furthermore, it is important to note that incidental hippocampal sclerosis, an entity recognized in the field of neuroimaging and debated in the epilepsy groups, requires special mention. From the histopathological point of view there are reports of HS in up to 10% of individuals without epilepsy (Menzler, 2010). This incidental MRI finding in patients without epilepsy is rare and requires the strict clinical correlation discarding undocumented seizure syndrome (Kevin, 1999).

6.2.4 Malformations of cortical development

Corresponds to a heterogeneous group of entities characterized by abnormal cerebral cortical structure, usually related to genetic, vascular, infectious and toxic alterations. It may occur during states of proliferation, migration or cellular organization in pre or postnatal stage depending of the case. It should be noted that these processes are synchronous and are continuous from temporal point of view(Abdel, 2009).
Although several classification schemes, the most common and practical approach due to concepts of Barkovich (Barkovich, 1996; Barkovich, 2001; Barkovich, 2005). They can be grouped into three categories according to the original classification based on the alteration in the development process microlisencephaly, hemimegalencephaly and cortical dysplasia (proliferation), complete lissencephaly, congenital muscular dystrophy and heterotopia (migration), polymicrogyria and schizencephaly (organization) [Fig. 8].

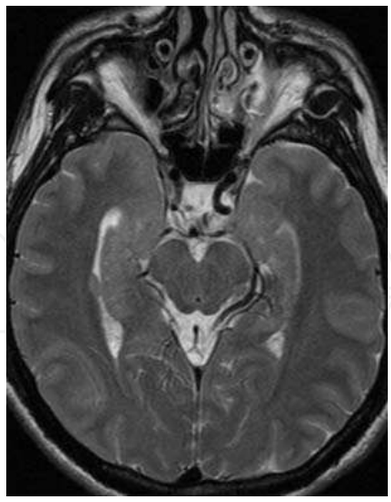

8A

8B

Fig. 8. Malformations of cortical development
A. Polymicrogyria in the right inferior temporal sulcus
B. Subependymal nodular heterotopia of the right temporal horn

Particular interest receives the entity of cortical dysplasia, a source of intense study in recent years. Blümcke recently published an international consensus that modifies the traditional classification of Palmini with the aim of improving the clinical-pathologic characterization of the entity (Blümcke, 2011).
The MRI features include: hyperintensity in T2, FLAIR and STIR (explained by cellularity, gliosis, hypomyelination or ball cells), Giral abnormal pattern, increased cortical thickness, cortical depression and absence of calcification [Fig. 9A] (Widdess, 2006). In the case of transmantle cortical dysplasia it could be found a path that reaches the ventricular surface (Fig. 9B).

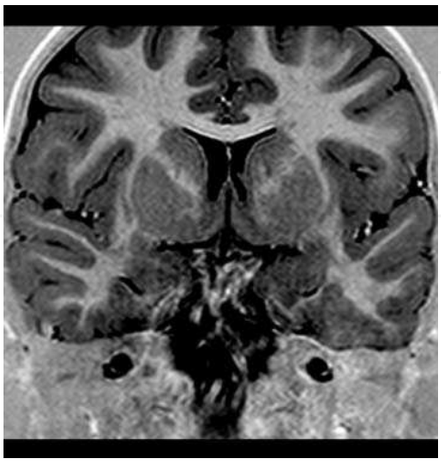
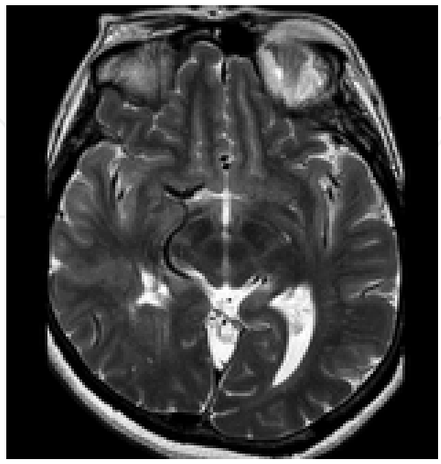
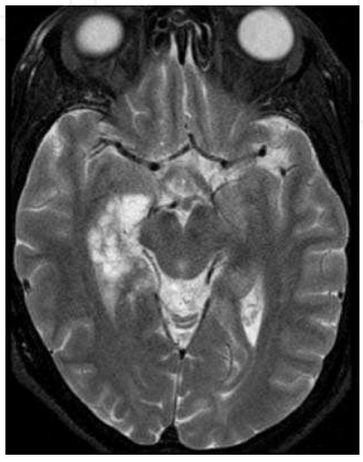

9A

9B

Fig. 9. Cortical dysplasias
A. Dysplasia of the left temporo-occipital and fusiform gyrus
B. Trasmantle right temporal dysplasia

6.2.5 Tumors

Neoplasias are usually of low-grade malignancy predominantly gangliogliomas and dysembryoplastic neuroepithelial tumors (DNET). It is important to remember its close association with malformations of cortical development not only for their coexistence, and similar imaging appearance but by their possible common origin (Urbach, 2004). It´s appearance are small lesions, cortical-subcortical, with varying degrees of calcification, microcystic component and little or no enhancement with gadolinium administration (Fig. 10).



10A



10B

Fig. 10. Tumors
A. DNET of right hippocampus
B. Left temporal medial ganglioglioma

6.2.6 Vascular

Cavernous malformation is the most common vascular disorder, in Lehéricy´s series, these were found in 4.5% of 222 patients with TLE studied with MRI (Lehéricy, 1997). This configuration is called "popcorn" in the T2 series, explained by the presence of central extracellular methemoglobin and peripheral hemosiderin (Fig. 11).

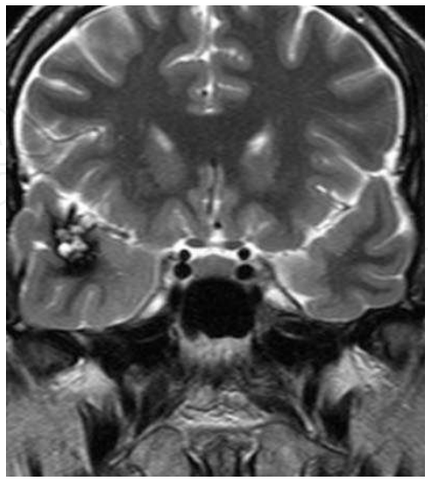


Fig. 11. Cavernous malformation

Image of "popcorn" on the anterior part of the right temporal.

6.2.7 Gliosis

This is the manifestation of a reactive process to different insults. Becomes relevant when talking about post-traumatic, post-infectious and postinfarct epilepsy. However, it is usually an epiphenomenon secondary to trauma in an epileptic patient with ictal onset zone with a different location, and may be indistinguishable from very small cortical dysplasias. Images are observed as hyperintense in FLAIR series (Fig. 12).

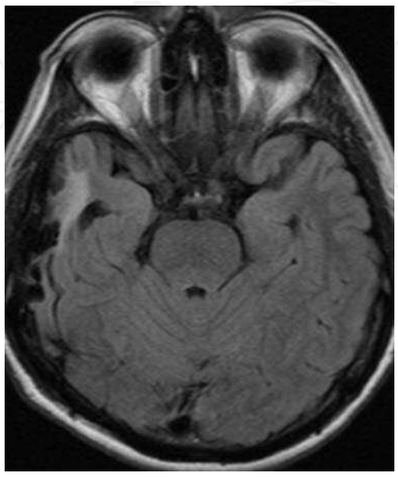


Fig. 12. Gliosis

Traumatic malacia and gliosis in the lateral part of right temporal lobe

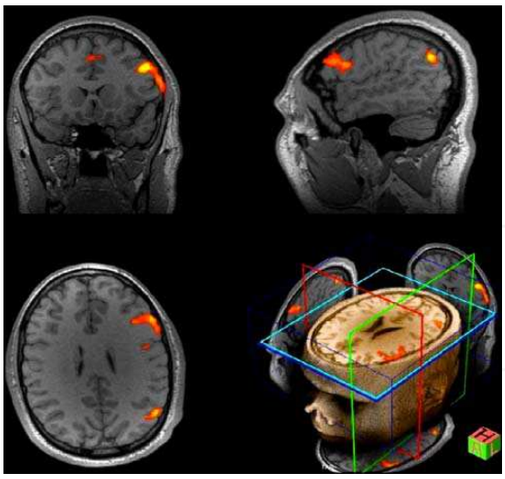
6.3 Functional image

It refers to the imaging techniques that allow the assessment of physiological, biophysical and metabolic properties of a tissue. We will talk about images by MRI (MRS, DTI and tractography or BOLD).

The MRS has shown disorders of certain metabolites such as N-acetyl aspartate (NAA), choline (Cho), creatine (Cr), myoinositol (Mio) and lactate (Lac) in TLE. It is well known the decrease in NAA and the indexes of NAA / Cho-Cr in the hippocampus with HS (Wieser, 2004). It has been reported significant hippocampal changes in neocortical TLE (Lee, 2005) and also described a possible relationship with the severity of epilepsy (Hammen, 2007). However, their greatest utility is reserved for patients with normal MRI as a potential lateralizing study. It should be noted that some changes seen in this context may be transient and reversible.

The DTI is based on microstructural tissue organization delimited by the anisotropic diffusion of water in the brain. The approach allows axonal trajectories, that report anomalies in shape, size, number and location of tracts and white matter fibers in addition to their state of myelination. Information closely related to the different cortical abnormalities has been described (Rastogi, 2008). It also has become an element almost for routine surgical planning.

Functional magnetic resonance imaging (fMRI) itself or BOLD (Blood Oxygen Level-Dependent) allows to obtain maps of cortical activity during an specific test (paradigm), based on hemodynamic changes resulting of neuronal activity. With respect to the presurgical evaluation, the most important to consider are language and memory functions (Fig. 13).


13A

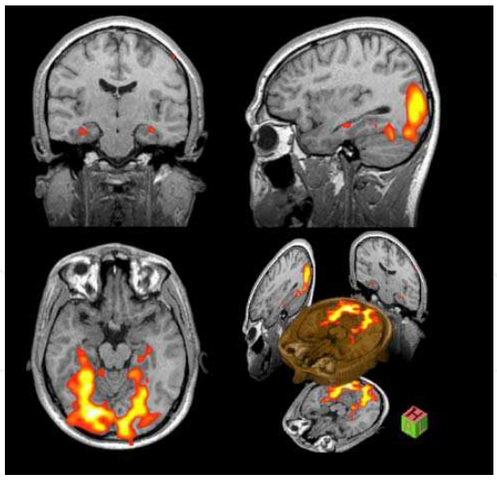

13B

Fig. 13. fMRI
A. Cortical activation of the left inferior frontal and supramarginal gyrus, corresponding to Broca's and Wernicke's areas respectively. Paradigms of Word-generation and comprehension (hear stories).
B. Bilateral medial temporal activation mediated by visual stimuli in a paradigm of memory (complex scenes).

The determination of lateralization of language is critical in TLE since in this population there are higher incidences of atypical dominance. That assessment has been done traditionally with the Intracarotid Amobarbital Procedure (IAP) or Wada test (Wada, 1960). In 1996, Binder made a comparative study in a group of 22 epileptic patients with IAP and a semantic decision paradigm in fMRI. He found a high correlation in terms of laterality indices. Emphasizing the potential of fMRI such as a noninvasive technique, without significant risks, independent of variations in arterial anatomy and easily reproducible (Binder, 1996). Gaillard subsequently studied 30 patients with complex partial epilepsy with a nomination paradigm and identified frontotemporal activation consistent with the IAP and cortical mapping (Gaillard, 2002). An important comparison between the two methods was performed by Woermann in a group of 100 patients, which used the word generation paradigm and showed a concordance of 91% (Woermann, 2003). Despite these good results it shouldn't be forgotten that like any diagnostic test there is always the possibility of false positives and false negatives. For example, Jayakar reported a case of false lateralization of language in a 14-year-old with left-HS who was assessed in the postictal period (Jayakar, 2002). On the other hand, it is necessary to keep in mind the cost of fMRI versus IAP for the assessment of candidates for surgery. A cost analysis study recently showed that the IAT is 3.7 times more expensive than fMRI in the evaluation of language lateralization (Medina, 2004). In contrast to the lateralization, the localization of critical areas for language has been very difficult. It seems that the use of different paradigms (verb generation, nomination, verbal fluency, and reading comprehension) gets more sensitive in the detection of these critical areas (Rutten, 2003).
With respect to the study of memory there are good results in the assessment of episodic memory with paradigms such as encoding of complex scenes, test that have shown good correlation in terms of lateralization with respect to the memory dominance found in IAP (Detre, 2004).

6.4 Future of neuroimaging in temporal lobe epilepsy

Continued progresses in different areas of knowledge converge on technological developments that in the case of magnetic resonance will generate key points in the field of early and accurate diagnosis. In that order of ideas we must note the possibilities that are opened with the use of ultrahigh field in the image not only structural but functional. We should think in the anatomical resolution in microscopic scale which are obtained from images of the hippocampal histologic slices that are now available with equipment of 7 and 9.4 T (Fatterpekar, 2002; Prudent, 2010).

7. Conclusion

The approach and management of patients with refractory epilepsy requires the presence of a multidisciplinary team with different sub-specialties and with the motivations to achieve the best possible outcome. Confirmation of the diagnosis of epilepsy and its medical intractability is the essential prerequisite for epilepsy surgery. The main objective of surgical treatment of epilepsy is seizure control and improvement of quality-of-life of patients with medically intractable epilepsy. The role of the different diagnostic methods with respect to identification of the epileptogenic zone is vital for surgical patient selection and outcome.

Based in our experience with the epilepsy group, and as expressed by many authors, the best results that are obtained when patients are submitted to epilepsy surgery, regarding crisis control, are those in which there is convergence of the different diagnostic methods towards the epileptogenic zone. But in other group of patients, there is no convergence of the diagnostic methods; these do not lateralize or lateralize to the contralateral side of the lesional zone. In this situation, each method takes its own specific weight, and based on the analysis of these results, a surgical decision can be made. Video-EEG is considered for many authors as the "gold standard" in the evaluation of refractory epilepsy and temporal mesial sclerosis. When it does not lateralize, or lateralizes to the contralateral side, other diagnostic methods such as the IAP or MRI take a much more important role, when it comes to decision making. The IAP defines the functional memory reserve, and limits the surgeon when it comes to resecting the affected area, defining if besides the lesion he can proceed with mesial structures, if the contralateral temporal lobe has a normal function. By this, we can avoid an unnecessary invasive intracranial EEG recording in this group of patients. It has been demonstrated that the propagation of ictal activity can be generated in the contralateral side of the lesion and transmitted to other areas using different networks. For example, if the MRI demonstrates a lesion in the contralateral lobe (temporal mesial sclerosis) to the one reported by the video-EEG as the ictal-onset zone, and the IAP reports functional memory reserve contralateral as well, this test would have less importance. In patients with dual pathology, when there is a tumor or a malformation, in addition to the demonstration that the seizure-onset zone is ipsilateral, the IAP has more specific weight. If there is a functional memory reserve contralateral to the lesion, the surgeon can make a wider resection, but if the functional memory reserve is ipsilateral, the surgeon should focus on the specific lesion.

8. References

Abdel, A.A.K., et al. (2009). Disorders of Cortical Formation: MR Imaging Features. *AJNR*, Vol. 30, pp. 4–11.

Abou-Khalil, B. (2007). An update on determination of language dominance in screening for epilepsy surgery: the Wada test and newer noninvasive alternatives. *Epilepsia*, Vol. 48, No. 3, pp. 442-55.

Alssadi, TM., et al. (2001). False lateralization by subdural electrodes in two patients with temporal lobe epilepsy. *Neurology*, Vol. 57, pp. 532-534.

Andrade, R., et al. (2011). Semiología de las crisis en las epilepsias temporales y tipos semiológicos de las epilepsias temporales, In: *Epilepsias del lóbulo temporal*. Cornejo, JW., & Toro, ME., pp. 23-33. ISBN: 978-958-44-8359-1, Medellín.

Andriani Rahal, M., et al. (2006). Somatosensory Aura in Mesial Temporal Lobe Epilepsy: Semiologic Characteristics, MRI Findings and Differential Diagnosis with Parietal Lobe Epilepsy. *J Epilepsy Clin Neurophysiol* , Vol. 12, No. 3, pp. 155-160.

Aoki, C. & Stekevitz, P. (1988). Plasticity in Brain Development. *Scientific American*, Vol. 259, pp. 34-42.

Archarya, V. et al. (1998). Olfactory epileptic auras. *Neurology*, Vol. 51, No. 1, pp. 56-61.

Ardila, A. & Rosselli, M. (1992). *Neuropsicología Clínica*, Manual Moderno, ISBN: 9707292792.

Aull-Watschinger, S., et al. (2008). Sexual auras: Predominance of epileptic activity within the mesial temporal lobe. *Epilepsy & Behavior, Vol.* 12, pp. 124–127.

Barkovich, J., et al. (1996). A classification scheme for malformationsof cortical development. *Neuropediatrics*, Vol. 27, pp. 59–63.

Barkovich, J., et al. (2001). Classification system for malformations of cortical development: update 2001. *Neurology*, Vol. 57, pp. 2168–78.

Barkovich, J., et al. (2005). A developmental and genetic classification for malformations of cortical development. *Neurology* , Vol. 65, pp. 1873–87.

Barsi, P., et al. (2000). Hippocampal malrotation with normal corpus callosum: a new entity?. *Neuroradiology,* Vol. 42, pp. 339-345.

Baxendale, S. (2009). The Wada test. *Curr Opin Neurol*, Vol. 22, No. 2, pp. 185-9.

Baxendale, S., et al. (2008). The role of the Wada test in the surgical treatment of temporal lobe epilepsy: an international survey. *Epilepsia*; Vol. 49, pp. 715–20; discussion 720-7.

Binder, JR., et al. (1996). Determination of language dominance using functional MRI: A comparison with the Wada test. *Neurology*, Vol. 46, No. 4, pp. 978-84.

Biraben, A. et al. (2001). Fear as the main feature of epileptic seizures. *J Neurol Neurosurg Psychiatry*,Vol. 70, No. 2, pp. 186–191.

Blümcke, I., et al. (2011). The clinicopathologic spectrum of focal cortical dysplasias: A consensus classification proposed by an ad hoc Task Force of the ILAE Diagnostic Methods Commission. Epilepsia, Vol. 52, No. 1, pp. 158–174.

Bronen, R., et al. (1996). Refractory Epilepsy: Comparison of MR Imaging, CT, and Histopathologic Findings in 117 Patients. *Radiology,* Vol. 201, pp. 97-105.

Canals, M. (2008). Historia de la resonancia magnética de Fourier a Lauterbur y Mansfield: en ciencias, nadie sabe para quién trabaja. *Rev Chil Radio,* Vol. 14, No. 1, pp. 39-45.

Castilho Garcia, MT. et al. (2010). Auras and clinical features in temporal lobe epilepsy: A new approach on the basis of voxel-based Morphometry. *Epilepsy Research*, Vol. 89, pp. 327—338.

Chelune, GJ. (1995). Hippocampal adequacy versus functional reserve: Predicting memory functions following temporal lobectomy. *Archives of Clinical Neuropsychology,* Vol. 10, pp. 413-432.

Chelune, G., & Najm, IM. (2001). Risk factors associated with postsurgical decrements in memory. In: *Epilepsy Surgery*. Lüders, H., & Comair, Y., editors. pp. 497-504, Lippincott Williams & Wilkins, Philadelphia.

Chiosa, V., et al. (2010). Temporal Lobe Epilepsy: From Electro-Clinical Semiology to Surgical Outcome. *Epileptologie*, Vol. 27, pp. 94 – 100.

Chiosa, V., M.S., S. Vulliémoz. (2010). Temporal Lobe Epilepsy: From Electro-Clinical Semiology to Surgical Outcome.*Epileptologie*, Vol. 27, pp. 94 – 100.

Dantas, FG., et al. (1998). Clinical and EEG analysis of mesial and lateral temporal lobe seizures. *Arq Neuropsiquiatr.,* Vol. 56(3A), pp. 341-9.

Deasy, N., et al. (2000). Thalamic changes with mesial temporal sclerosis: MRI. *Neuroradiology*, Vol. 42, pp. 346-351.

Dericioglu, N., & Saygi, S. (2008). Ictal scalp EEG findings in patients with mesial temporal lobe epilepsy. *Clin EEG Neurosci.,* Vol. 39, No. (1), pp. 20-7.

Detre J. (2004). fMRI: Applications in Epilepsy. *Epilepsia*, Vol. 45 (Suppl. 4), pp. 26–31.

Devinsky, O., et al. (1995). Ictus emeticus; further evidence of non-dominant temporal involvement. *Neurology*, Vol. 45, pp. 1158-1160.

Di Gennaro, G., et al. (2004). Ictal heart rate increase precedes EEG discharge in drug-resistant mesial temporal lobe seizures. *Clin Neurophysiol.*, Vol. 115, pp. 1169-77.

Drake, M., et al. (2002). Evaluación neuropsicológica y test de Wada en el pronóstico de la lobectomía temporal en pacientes con epilepsia del lóbulo temporal mesial. *Rev. Neur Argentina*, Vol. 27, pp. 254-260.

Dubeau, F., & McLachlan, RS. (2000). Invasive electrographic recording techniques in temporal lobe epilepsy. *Can J Neurol Sci.*, Vol. 27, No. 1, pp. 29-34.

Dupont, S., et al. (2010). Functional MR imaging or Wada test: which is the better predictor of individual postoperative memory outcome?. *Radiology*, Vol. 255, No. 1, pp. 128-34.

Ebersole, JS., & Pacia, SV. (1996). Localization of temporal lobe foci by ictal EEG patterns. *Epilepsia*, Vol. 37, No. 4, pp. 386-99.

Ebner, A., et al. (1995). Automatisms with preserved responsiveness: a lateralizing sign in psychomotor seizures. *Neurology,* Vol. 45, pp. 61-64.

Eisenschenk, S., et al. (2001). Lateralization of temporal lobe foci: depth versus subdural electrodes. *J Clin Neurophysiol*, Vol. 112, pp. 836-844.

Engel, Jr J., & Ojemann, GA. (1993). *Surgical treatment of the epilepsies*. [ed.] Jr J Engel. Raven Press, pp. 319-329, New York.

Fatterpekar, G., et al. (2002). Cytoarchitecture of the Human Cerebral Cortex: MR Microscopy of Excised Specimens at 9.4 Tesla. *AJNR*, Vol. 23, pp. 1313–21.

Fernádez-Torre JL. (2002). Auras epilépticas: clasificación, fisiopatología, utilidad práctica, diagnóstico diferencial y controversias. *Rev Neurol*, Vol. 34, No. 10, pp. 977-983.

French, JA., et al. (1993). Characteristics of medial temporal lobe epilepsy. I. Results of history and physical examination. *Ann Neurol,* Vol. 34, pp. 774–80.

Fried, I. et al. (1995). The anatomy of epileptic auras: focal pathology and surgical outcome. *J Neurosurg*, Vol. 83, No. 1, pp. 60-66.

Gaillard, WD., et al. (2002). Language dominance in partial epilepsy patients identified with an fMRI reading task. *Neurology*, Vol. 59, pp. 256-65.

Galeano, LM., et al. (2011). Importancia del procedimiento con amobarbital intracarotideo en la evaluación prequirurgica del paciente con epilepsia del lóbulo temporal, In:

Epilepsias del lóbulo temporal. Cornejo, JW., & Toro, ME., pp. 181-193. ISBN: 978-958-44-8359-1, Medellín.

Gates, JR., & Cruz-Rodriguez, R. (1990). Mesial temporal sclerosis: pathogenesis, diagnosis and management. *Epilepsia*, Vol. 32, pp. 55-65.

Geyer, JD., et al. (1999). Significance of interictal temporal lobe delta activity for localization of the primary epileptogenic region. *Neurology*, Vol. 52, pp. 202-5.

Gil-Nagel, A., & Risinger MW. (1997). Ictal semiology in hippocampal versus extrahippocampal temporal lobe epilepsy. *Brain*, Vol. 120, pp. 183-92.

Gloor, P. (1990). Experiential phenomena of temporal lobe epilepsy. Facts and hypotheses. *Brain*; Vol. 113 (Pt 6), pp. 1673–94.

Hammen, T. et al. (2007). H-MR Spectroscopy Indicates Severity Markers in Temporal Lobe Epilepsy: Correlations between Metabolic Alterations, Seizures, and Epileptic Discharges in EEG. *Epilepsia*, Vol. 48, No. 2, pp. 263–269.

Hausser-Hauw, C. et al. (1987). Gustatory hallucinations in epileptic seizures. Electrophysiological, clinical and anatomical correlates. *Brain*, Vol. 110, No. 2, pp. 339–359.

Henkel, A., et al. (2002). The localizing value of the abdominal aura and its evolution: a study in focal epilepsies. *Neurology,* Vol. 58, No. 2, pp. 271-276.

Immonen, A., et al. (2010). Long-term epilepsy surgery outcomes in patients with MRI-negative temporal lobe epilepsy. *Epilepsia*, Vol. 51, pp. 2260-2269.

Jasper, H. (1958). The ten-twenty electrode system of the International Federation. *Electroencephalogr Clin Neurophysiol,* Vol. 10, pp. 371-5.

Jayakar, P., et al. (2002) False lateralization of language cortex on functional MRI after a cluster of focal seizures. *Neurology*, Vol. 58, pp. 490-2.

Jones-Gotman, M., et al. (2000). Neurosychological assessment for temporal lobe epilepsy surgery. *Can J Neurol Sci*, Vol. 27 (Suppl. 1), pp. S42-S46.

Kaplan, PW., et al. (1999). Ictus expectoratus: a sign of complex parcial seizures usually of non dominant temporal lobe origin. *Seizure,* Vol. 8, pp. 480-484.

Kasper, BS., et al. (2010). Phenomenology of hallucinations, illusions, a delusions as part of seizure semiology. *Epilepsy Behav*,Vol. 18, No. 1-2, pp. 13-23.

Kesavadas, C., & Thomas, B. (2008). Clinical applications of functional MRI in epilepsy. *Indian J Radiol Imaging*, Vol. 18, No. 3, pp. 210-7.

Kevin, R., et al. (1999). Incidental Detection of Hippocampal Sclerosis on MR Images: Is It Significant?. *AJNR*, Vol. 20, pp. 1609–1612.

Kier, E., et al. (1997). Embryology of the Human Fetal Hippocampus: MR Imaging, Anatomy, and Histology. *AJNR*, Vol. 18, pp. 525–532.

Kotagal, P., et al. (1995). Psychomotor seizures of temporal lobe onset: analysis of symptom clusters and sequences. *Epilepsy Res*, Vol. 20, pp. 49-67, 1995.

Koutroumanidis, M., et al. (2004). Interictal temporal delta activity in temporal lobe epilepsy: correlations with pathology and outcome. *Epilepsia,* Vol. 45, No. 11, pp. 11351-67.

Kuba, R., et al. (2010). Peri-ictal yawning lateralizes the seizure onset zone to the nondominant hemisphere in patients with temporal lobe epilepsy. *Epilepsy Behav.*, Vol. 19, No. 3, pp. 311-4

Kwong, KK., et al. (1992). Dynamic magnetic resonance imaging of human brain activity during primary sensory stimulation. *Proc Natl Acad Sci USA*, Vol. 89, pp. 5675-9.

Lee, S., et al. (2005). Effect of seizure on hippocampus in mesial temporal lobe epilepsy and neocortical epilepsy: an MRS study. *Neuroradiology*, Vol. 47, pp. 916–23.

Lehéricy, S., et al. (1997). Temporal lobe epilepsy with varying severity: MRI study of 222 patients. *Neuroradiology,* Vol. 39, pp. 788-96.

Lin, K., et al. (2009). Sign of the Cross (Signum Crucis): Observation of an uncommon ictal manifestation of mesial temporal lobe epilepsy. *Epilepsy & Behavior, Vol.* 14, pp. 400–403.

Loddenkemper, T., & Kotagal, P. (2005). Lateralizing signs during seizures in focal epilepsy. *Epilepsy Behav,* Vol. 7, pp. 1-17.

Loring, D., & Meador, K. (2001). Wada testing to define Hippocampal Function. In: *Epilepsy Surgery*. Lüders, H., & Comair, Y., editors. pp. 531-536. Lippincott Williams & Wilkins, Philadelphia.

Lüders, HO. (2008). *Textbook of Epilepsy Surgery,* Informa Healthcare UK Ltd., ISBN-10: 1841845760, London.

Lüders, HO. (1999). Chapter 16 Mesial Temporal Sclerosis, In: *The Epilepsies: Etiologies and Prevention.* Kotagal, P., & Lüders, HO. Academic Press, San Diego.

Maestú, F., et al. (2000). Evaluation of epilepsy surgery. *Rev Neurol*, Vol. 15, No. 5, pp. 477-82.

Maestú, F., et al. (1999). Neuropsicología y deterioro cognitivo en epilepsia. *Rev Neurol,* Vol. 28, pp. 793-798.

Manford, M. et al. (1996). An analysis of clinical seizure patterns and their localizing value in frontal and temporal lobe epilepsies. *Brain*, Vol. 119, pp. 17–40.

McIntyre, DC., & Gilby K. (2008). Mapping seizure pathways in the temporal lobe., *Epilepsia*, Vol. 49, pp. 23-30.

Medina, LS., et al. (2004). Functional MR Imaging versus Wada Test for Evaluation of Language Lateralization: Cost Analysis. *Radiology*, Vol. 230, pp. 49-54.

Meletti, S., et al. (2003). The expression of interictal, pericital, y postictal facial-wiping behavior in temporal lobe epilepsy: a neuro-ethological analysis and interpretation. *Epilepsy & Behavior,* Vol. 4, pp. 635-643.

Menzler, K., et al. (2010). Evaluation of MRI criteria (1.5T) for the diagnosis of hippocampal sclerosis in healthy subjects. *Epilepsy Research,* Vol. 89, No. 2, pp. 349-354.

Milner, B. (1966). Amnesia following operation on the temporal lobes. In: Amnesia. Whitty,CWM. & Zangwill, OL., editors. pp. 109–33. Butterworths, London.

Mintzer, S., Lopez, F. (2002). Comorbidity of ictal fear and panic disorder. *Epilepsy and Behavior*, Vol. 3, No. 4, pp. 330–337.

Mohammed, M., et al. (2010). Electroencephalographic Features of Temporal Lobe Epilepsy. *Can. J. Neurol. Sci.,* Vol. 37, pp. 439-448.

Morris, HH., et al. (1987). Value of closely spaced electrodes in the localization of epileptiform foci: a study of 26 patients with complex partial seizures. *Electroencephalogr Clin Neurophysiol*, Vol. 63, pp. 107-11.

Napolitano, CE., & Orriols, MA. (2010). Graduated and sequential propagation in mesial temporal epilepsy: analysis with scalp ictal EEG. *J Clin Neurophysiol*, Vol. 27, No. 4, pp. 285-291.

Napolitano, CE., & Orriols, M. (2008). Two types of remote propagation in mesial temporal epilepsy: analysis with scalp ictal EEG. *J Clin Neurophysiol*, Vol. 25, pp. 69-76.

Ng, S., et al. (1997). MRI of the fornix and mamillary body in temporal lobe epilepsy. *Neuroradiology*, Vol. 39, pp. 551–5.

Noachtar, S., & Rémi, J. (2009). The role of EEG in epilepsy: a critical review. *Epilepsy Behav.*, Vol. 15, No. 1, pp. 22-33.

Oddo S., et al. (2000). Protocolo de evaluación neuropsicológica para pacientes candidatos a cirugía de la epilepsia. *Rev Neurol. Arg.*, Vol. 25 (S2), pp. 68.

Ogawa, S., et al. (1992). Intrinsic signal changes stimulation: functional brain mapping with magnetic resonance imaging. *Proc Natl Acad Sci USA*, Vol. 89, pp. 5951-5.

Ogawa, T., et al. (1995). Unusual Widening of Virchow-Robin Spaces: MR Appearance. *AJNR,* Vol. 16, pp. 1238–1242.

Oikawa, H., et al. (2001). The circuit of Papez in mesial temporal sclerosis: MRI. *Neuroradiology,* Vol. 43, pp. 205-210.

Oppenheim, C., et al. (1998). Loss of Digitations of the Hippocampal Head on High-Resolution Fast Spin-Echo MR: A Sign of Mesial Temporal Sclerosis. *AJNR*, Vol. 19, pp. 457–463.

Orozco-Giménez, C., et al. (2002). Neuropsicología Clínica en la Cirugía de Epilepsia del lóbulo Temporal. *Rev Neurol*, Vol. 35, No. 12, pp. 1116-1135.

Ostrowsky, K., et al. (2000). Functional mapping of the insular cortex: clinical implication in temporal lobe epilepsy. *Epilepsia*; Vol. 41, pp. 681-686.

Palacios, R., et al. (2008). Hippocampal Sclerosis: Histopathology Substrate and Magnetic Resonance Imaging. *Semin Ultrasound CT MRI*, Vol. 29, pp. 2-14.

Paolicchi, JM. (2008). Is the Wada test still relevant? Yes. *Arch Neurol*, Vol. 65, No. 6, pp. 838-40.

Parra, J., & Iriarte, J. (1999). Valor del registro ictal con vídeo-EEG en la evaluación prequirúrgica de pacientes con epilepsia del lóbulo temporal. Semiología y patrones electroencefalográficos. *Rev Neurol*, Vol. 28, No. 9, pp. 898-908.

Placantonakis, DG., et al. (2010). Bilateral intracranial electrodes for lateralizing intractable epilepsy: efficacy, risk, and outcome. *Neurosurgery*, Vol. 66, pp. 274-283.

Prudent, V., et al. (2010). Human Hippocampal Subfields in Young Adults at 7.0 T: Feasibility of Imaging. *Radiology*, Vol. 254, No. 3, pp. 900-6.

Rastogi, S., et al. (2008). Neuroimaging in Pediatric Epilepsy: A Multimodality Approach. *RadioGraphics*, Vol. 28, pp. 1079–95.

Ray, A., & Kotagal, P. (2005). Temporal lobe epilepsy in children: overview of clinical semiology. *Epileptic Disord*, Vol. 7, No.4, pp. 299-307.

Rausch, R. (2001). Psychological evaluation. In: *Surgical treatment of epilepsies*. Raven Press, New York.

Rodriguez, AJ., et al. (2007). Temporal lobe seizure semiology during wakefulness and sleep. *Epilepsy Research,* Vol. 74, No. 2-3, pp. 211—214.

Rutten, GJ., et al. (2003). Toward functional neuronavigation: implementation of functional magnetic resonance imaging data in a surgical guidance system for intraoperative identification of motor and language cortices. *Neurosurgical Focus* , Vol. 15, No. 1, pp. 1-6.

Saygi, S., et al. (1994). Differentiation of temporal lobe ictal behavior associated with hippocampal sclerosis and tumors of temporal lobe. *Epilepsia*, Vol. 35, pp. 737-42.

Sammaritano, M., et al. (1987). False lateralization by surface EEG of seizure onset in patients with temporal lobe epilepsy and gross focal cerebral lesions. *Ann Neurol*, Vol. 21, pp. 361-369.

Sakai, Y., et al. (2002). Localization of epileptogenic zone in temporal lobe epilepsy by ictal scalp EEG. *Seizure*, Vol. 11, pp. 163–168.

Serrano-Castro, PJ., et al. (1998). Mesial temporal sclerosis (II); Clinical features and complementary studies. *Rev Neurol,* Vol. 26, No. 152, pp. 592-597.

Sharan, A., et al. (2010). Intracarotid amobarbital procedure for epilepsy surgery. *Epilepsy Behav*. 2010 Dec 27. [Epub ahead of print]

Shorvon, SD. (2009). History of epilepsy 1909–2009: The ILAE century. *Epilepsia,* Vol. 50 (Suppl. 3), pp. 39–49.

Silverman, D. (1960). The anterior temporal electrode and the ten-twenty system. *Electroencephalogr Clin Neurophysiol*, Vol. 12, pp. 735-7.

Spencer, DC., et al. (2000). The role of the intracarotid amobarbital procedure in evaluation of patients for epilepsy surgery. *Epilepsia*, Vol. 41, No. 3, pp. 320-5.

Topper, R., et al. (1999). Clinical significance of intracranial developmental venous anomalies. *J Neurol Neurosurg Psychiatry,* Vol. 67, pp. 234–8.

Trenerry, M., & Loring, D. (2006). The intracarotid Amobarbital Procedure. In: The *Treatment of Epilepsy: Principles and Practice.* Wyllie, Elaine, Editor. pp. 1043-1052. Lippincott Williams & Wilkins, Philadelphia.

Urbach, H., et al. (2004). MR Imaging in the Presurgical Workup of Patients with Drug-Resistant Epilepsy. *AJNR*, Vol. 25, pp. 919-26.

Vattipally, V., & Bronen, R. (2006). MR Imaging of Epilepsy: Strategies for Successful Interpretation. *Radiol Clin N Am*, Vol. 44, pp. 111–133.

Van Buren, JM. (1963). The abdominal aura: a study of abdominal sensations occurring in epilepsy and produced by depth stimulation. *Electroenceph Clin Neurophysiol,*Vol. 15, pp. 1–19.

Vossler, DG., et al. (1998). Temporal ictal electroencephalographic frequency correlates with hippocampal atrophy and sclerosis. *Ann Neurol.*, Vol. 43, pp. 756-62.

Wada JA. (2008). A fateful encounter: sixty years later – reflections on the Wada test. *Epilepsia*, Vol. 49, pp. 726–727.

Wada, J., & Rasmussen, T. (1960). Intracarotid injection of sodium amytal for the lateralization of cerebral speech dominance. *J Neurosurg*, Vol. 17, pp. 266-82.

Widdess-Walsh, P., et al. (2006). Neuroimaging of Focal Cortical Dysplasia. J *Neuroimaging*, Vol. 16, pp. 185-96.

Walczak, T., et al. (1992). Accuracy and interobserver reliability of scalp ictal EEG. *Neurology*, Vol. 42, pp. 2279-85.

Wieser, HG. (1983). Electroclinical Features of the Psychomotor Seizure: A Stereoelectroencephalographic Study of Ictal Symptoms and Chronotopographical Seizure Patterns Including Clinical Effects of Intracerebral Stimulation. London: Butterworths.

Wieser, H.G. (2004). ILAE Comisión Report. Mesial Lobe Temporal Epilepsy with Hippocampal Sclerosis. *Epilepsia*, Vol. 45, No. 6, pp. 695-714.

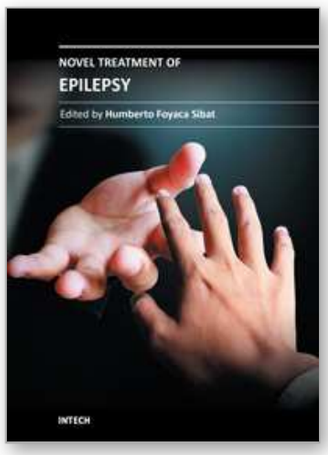
Woermann, FG., et al. (2003). Language lateralization by Wada Test and fMRI in 100 patients with epilepsy. *Neurology*, Vol. 61, pp. 699-701.

Yu, HY., et al. (2010). The Wada memory test and prediction of outcome after anterior temporal lobectomy. *J Clin Neurosci*., Vol. 17, No. 7, pp. 857-61. Epub 2010 May 14.

Zapata, JF., et al. (2010). Ictal electrocorticographic dissemination pattern in mesial temporal epilepsy with epileptogenic zone discordance. Proceedings of 6th Latin American Congress on Epilepsy (LACE), Cartagena, August 2010.

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Epilepsy continues to be a major health problem throughout the planet, affecting millions of people, mainly in developing countries where parasitic zoonoses are more common and cysticercosis, as a leading cause, is endemic. There is epidemiological evidence for an increasing prevalence of epilepsy throughout the world, and evidence of increasing morbidity and mortality in many countries as a consequence of higher incidence of infectious diseases, head injury and stroke. We decided to edit this book because we identified another way to approach this problem, covering aspects of the treatment of epilepsy based on the most recent technological results â€œin vitroâ€ from developed countries, and the basic treatment of epilepsy at the primary care level in rural areas of South Africa. Therefore, apart from the classic issues that cannot be missing in any book about epilepsy, we introduced novel aspects related with epilepsy and neurocysticercosis, as a leading cause of epilepsy in developing countries. Many experts from the field of epilepsy worked hard on this publication to provide valuable updated information about the treatment of epilepsy and other related problems.

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