

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

185,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.  
For more information visit [www.intechopen.com](http://www.intechopen.com)



---

# Anxiety as an Epileptical Equivalent (Temporal Lobe Epilepsy)

---

Luis A. Pando-Orellana

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/intechopen.78241>

---

## Abstract

To consider anxiety as an epileptical equivalent, several considerations have to be taken into account. First of all, what is anxiety and what is if possible a universal conception of epilepsy, and I say this because although we have the DSM series, the International League Against Epilepsy and everybody talks about “consensus” the way we classify almost every syndrome in medicine tends to dilute due to the diversity of clinical manifestations that have to be taken into account, so where and how do we apply such terms. There we have our “new” platform to understand, directly and indirectly, the role of temporal lobe syndromes, which, by the way, we all “have” but need to be discerned, as we can appreciate in EEG quantitative analysis: as to why temporal lobe is always marking for increased activity?

**Keywords:** anxiety, epilepsy, temporal lobe, limbic

---

## 1. Introduction

Examples of these are headaches, even though we have classic definitions and descriptions of such “disease” in Oliver Sacks in his book *Migraine* [1], in which, by the way, he makes a central point and establishes serious questioning toward cultural aspects of photopsias: a kind of environment influence and memory in its manifestations.

Once upon a time, during my practice in the Lacandon jungle during the late 1970s, a patient came in talking and complaining using the indigenous Mayan Tzeltal language; said to my interpreter: I feel **Kuúch, which means pain:** But what kind of pain?

The usual semiology did not work within this context. He had a **heart ache** which at the end of this consultation meant, he was depressed and the anxiety made his heart “ache.”

---

I asked myself if this heart could really “ache” though it came from emotional impact. The answer (to my best clinical knowledge) was YES, it aches. What else could drive this man to search for help, in a context of magical thoughts, shamanes and syncretic ideas?

Years later as a specialist, I became very interested in the temporal lobe, with a very big question: is the temporal lobe and its connections with ventral-lateral thalamus responsible of such symptoms?

Again the answer was YES. And if so, how to prove it?

Again if so: can we consider the temporal lobe and its connections the real rhythm **pacemaker** of the brain, instead of the occipital? Again the answer is YES [2].

The quest started about 20 years ago: Prove it!

So here are the facts that were gradually published in my native language: Spanish [2–4].

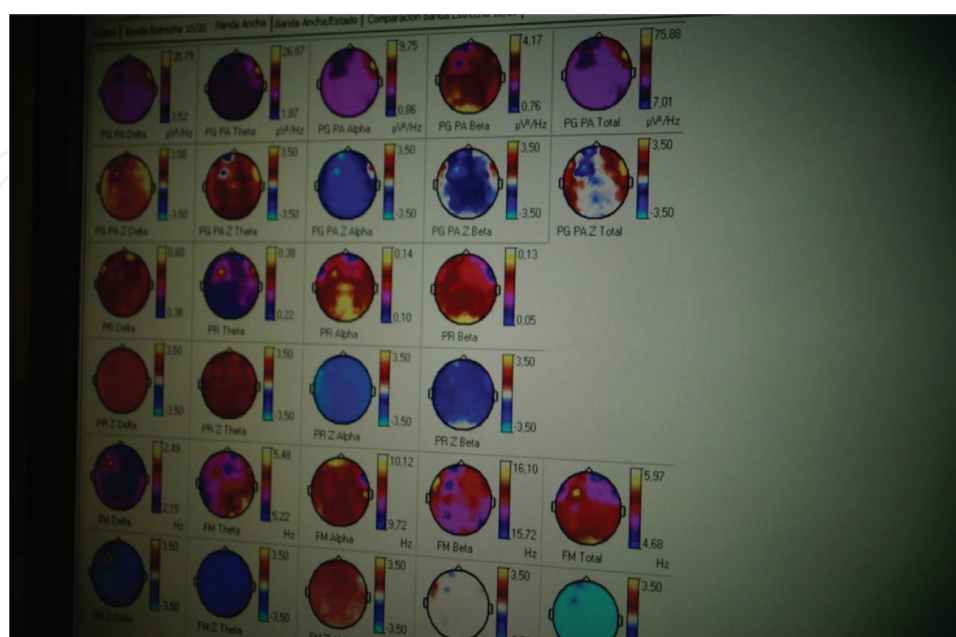
[www.evpoll.com.mx](http://www.evpoll.com.mx)

Reviewing antecedents and history, we owe a great deal to three persons: Paul Broca, Wilder Penfield, and Isaac Costero. We are talking a century later about the Cajal legacy from Spain.

Paul Broca a century ago talked and supported the internal brain or Limbic system, Wilder Penfield mapped the homunculus 1930s, and Costero (1980s) established the neurovegetative connections of carotid sensors with CNS and thalamic connections.

There we have our “new” platform to understand, directly and indirectly, the role of temporal lobe syndromes, which by the way we all “have” but need to be discerned, as we can appreciate in EEG quantitative analysis (**Figure 1**): as to why temporal lobe is always marking for increased activity?

So up to now, we have more data to correlate symptoms with changes in the EEG and sophisticated imaging and biological markers that do not contradict other models of Mind Brain Analysis (like the Game theory originated in the nineteenth century).

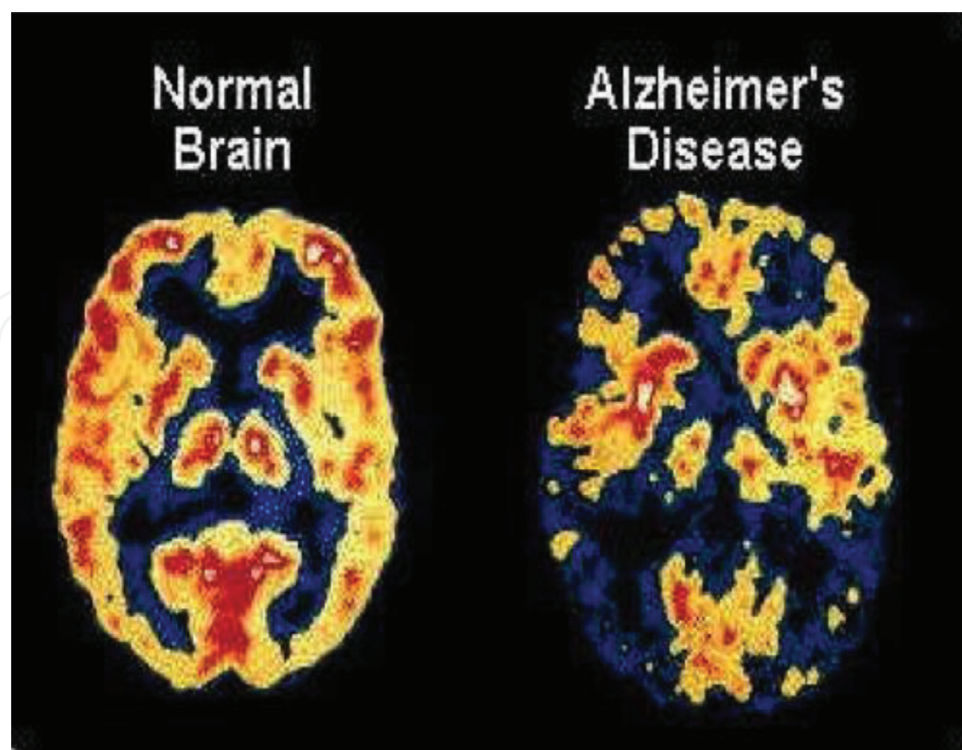


**Figure 1.** Geometrical power frontotemporal syndrome (courtesy of Pando et al.).

The models involve blood-brain barrier (BBB) studies of markers like S100B protein (Pando et al. in print); choline/creatine ratio visualizes through spectroscopy the affective disorders or PET/CT as marker for dementia and epilepsy particularly temporal lobe, then it has to mean something correlating it with clinical manifestations (**Figures 1–5**).

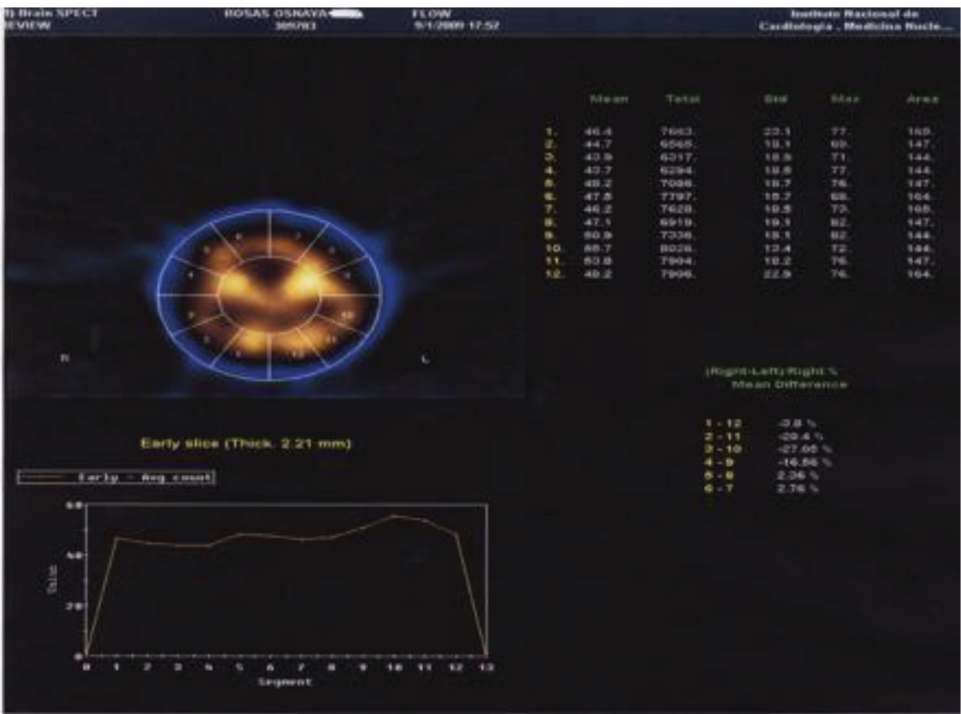


**Figure 2.** Frontotemporal views of one of our specimens. (courtesy of Pando et al. National institute of Cardiology “Ignacio Chavez”).



**Figure 3.** PET/CT with temporal lobe asymmetry (FDG marking). Courtesy of Pando et al., images from CT scanner, Mexico City.





**Figure 4.** Single positron emitted tomography with cortical hypo-perfusion post hypovolemic shock. (Courtesy of Pando et al.).



**Figure 5.** Wilder Penfield (public photograph). Mapped the homunculus (Johns Hopkins University).

## 2. Mind and game theory

These few examples of technological biomedicine can and must be applied to brain function and neuropsychiatry and not only statistics and few specialists writing “consensus” for everybody.

We all experience clinical variations of the very same frontotemporal syndrome, and these instruments are allowing us to understand how to aboard them (including autopsies) (**Figure 2**), which are being underestimated nowadays.

So if anxiety means a constant or increasing FEELING of emotional discomfort, with an urgent necessity of relief by searching just about any means available; like in the game theory, the brain responds:

If you have a certain set of cards and it is the upper hand to the “house,” you pass through to the reward that is transmitting the message that your senses are sending whatever the environmental stimuli is.

There are times though in which the message does not go through, with a barrier of an overwhelmed house (brain) with demand of reward, and central temporal lobe like in medial sclerosis discharges (**Figures 1–3**), that is, with an alarm manifestation but abnormally with persistent anxiety, no matter the prefrontal cortex. So the frontal is freed from emotional filters and thought containment is surpassed within the balance that the frontal and temporal lobe must have. A psychosis is established at least for the duration of the ictus, perpetuating itself until either by internal homeorrexic mechanisms or external intervention (**Figure 2**).

The intensity of the phenomena varies from mild to panic attacks and the process can be triggered by any disrupting affective loss up to discharges that come from altered networks from a growing tumor.

Everybody “has” a frontotemporal syndrome, in any time given; [2–5] because that is how we respond emotionally; but it is to the clinician to determine the underlying pathology if so [6–11].

The temporal lobe epilepsy is one of them characterized to behave as a seizure and marking (EEG) to the temporal inferior and superior temporal gyrus [6–9, 11] (**Figure 2**).

Clinically corresponds to nine subtypes obtained through the clinical analysis and combination of temporal and frontal manifestations, considering the following variables:

C = psychotic manifestations.

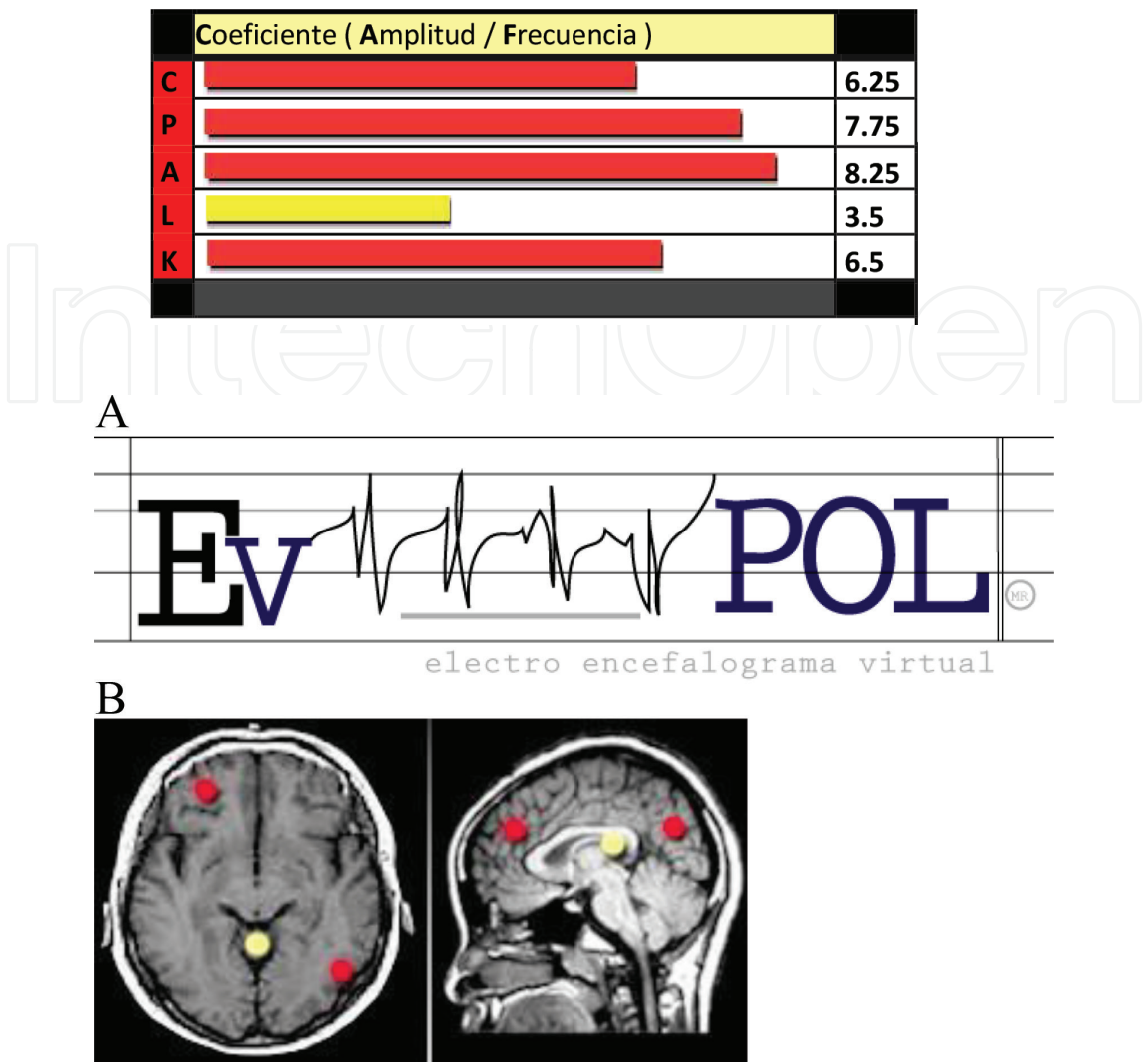
P = neurodevelopmental antecedents including genetical traits.

A = epileptic behavior, including panic attacks, generalized anxiety and depressive refractory mood that fluctuates within hours.

L = individuals that simulate or plainly lie.

K = sensoperceptual dysfunction. We see what we want to see not corresponding to proper protective neural networks function and memory.

Attached a report from the EVPOL (Virtual EEG, Pando et al.) SYSTEM that correlates the geometric power of the cerebral map with a 100 question inventory of frontotemporal manifestations related as well to anxiety and panic attacks [2–5] (**Figure 6**).



**Figure 6.** (A) EVPOL logo. Courtesy of Pando et al. research lab in CPALC., Mexico City. (B) MRI of brain with marking of abnormal activity. Courtesy of Pando et.al research lab in CPALC. Mexico City.

The following is described in the opinion that is shown later. The image shows the points in the brain linked to the behaviors.

- Opinion:
  - There are antecedents of risk to health, both of a genetic nature and of consequences against environmental interactions, with implications depending on the individual's predisposition to physical or psychological illness over the course of growth and development
  - Due to family history, you cannot perceive the information correctly. You may have obvious or compensated learning disorders.
- This opinion is the product of deep scientific and clinical research. Dr. Luis Pando-Orellana endorses and issues this opinion to the best of his knowledge and belief. Dr. Luis Pando-Orellana is a doctor and Doctor of Sciences with a specialty in Neuropsychiatry and Doctor in Immunology.

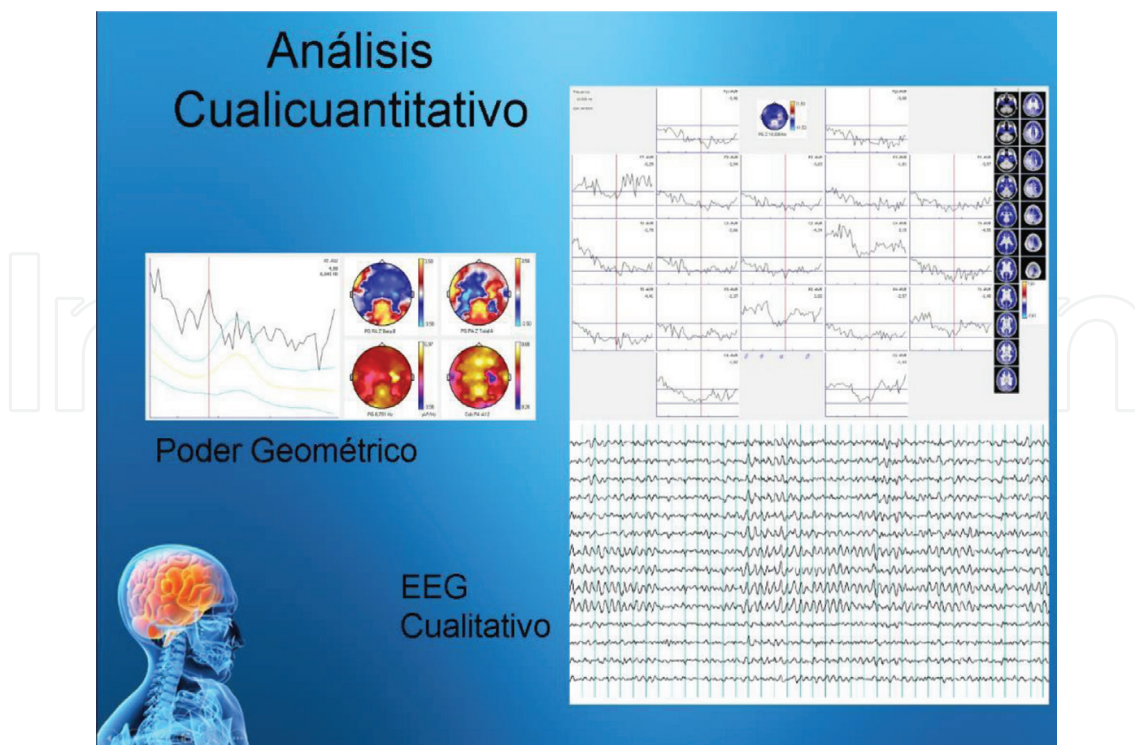
- Recommendation:
  - The Neuropsychiatrist recommends consulting your doctor to improve your quality of life
- This report describes behaviors that can be changed by endogenous factors [2–5].

So as in Migraines [12], we should start to deal with an unsolved classification (at least up to now) of anxiety as epileptical equivalent and it starts with: how many days should we support the patient sedated in order to substitute benzodiazepines [4] for a better neuroregulation through anticonvulsive psycho-neuropharmacology with much lesser risks of addictions or nephron-hepatic conditions with sometimes smaller doses than the ones recommended by the pharmaceutical industry [4, 6–11].

The definition of epilepsy is a paroxistic abnormal electrical discharge of any given group of neurons and any group of neurons based on the principle, one neuron, one function: networks develop through vectorial signals that are received at the level of neurofilaments which have quantic sensors in the concept of space, time, and structure that Schrodinger developed in the 30s of the past century, using Riemann tensors [2, 13].

As such if any group of given neuron discharges and recruits a network, ectopically, any given signs and symptoms should or potentially have to manifest themselves and can be registered in cuanti-qualitative analysis of the EEG and or other imaging techniques (**Figure 7**).

That is why the International League against epilepsy has not closed the door for future incorporation of such epileptic manifestations.



**Figure 7.** Quantitative analysis mapping of frontotemporal syndromes. (Courtesy of Pando et al., Mexico City).



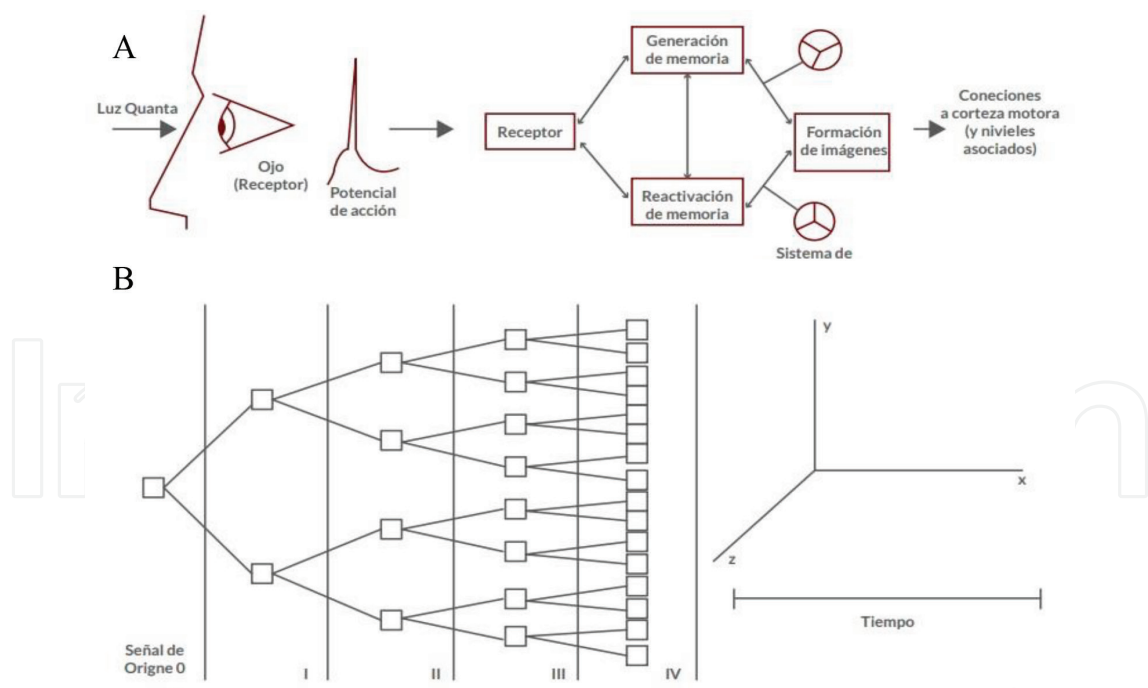
Signals that are transcribed as semiconductors (neurofilaments) transit through synaptic trees in real time and stock these information for short periods of time between 1 ms up to more than 1 s from one membrane to another through an inter-synaptic space; the first membrane of 50 Å (thickness) initiates the process.

All the way in a tetra-dimensional quanta information traveling through tensors and counter tensors, finding their way through space networks, and respecting the thermodynamical laws with an infinitesimal delay in this essential form of computational quantic central nervous system activity [2, 13] (Figures 7–10).

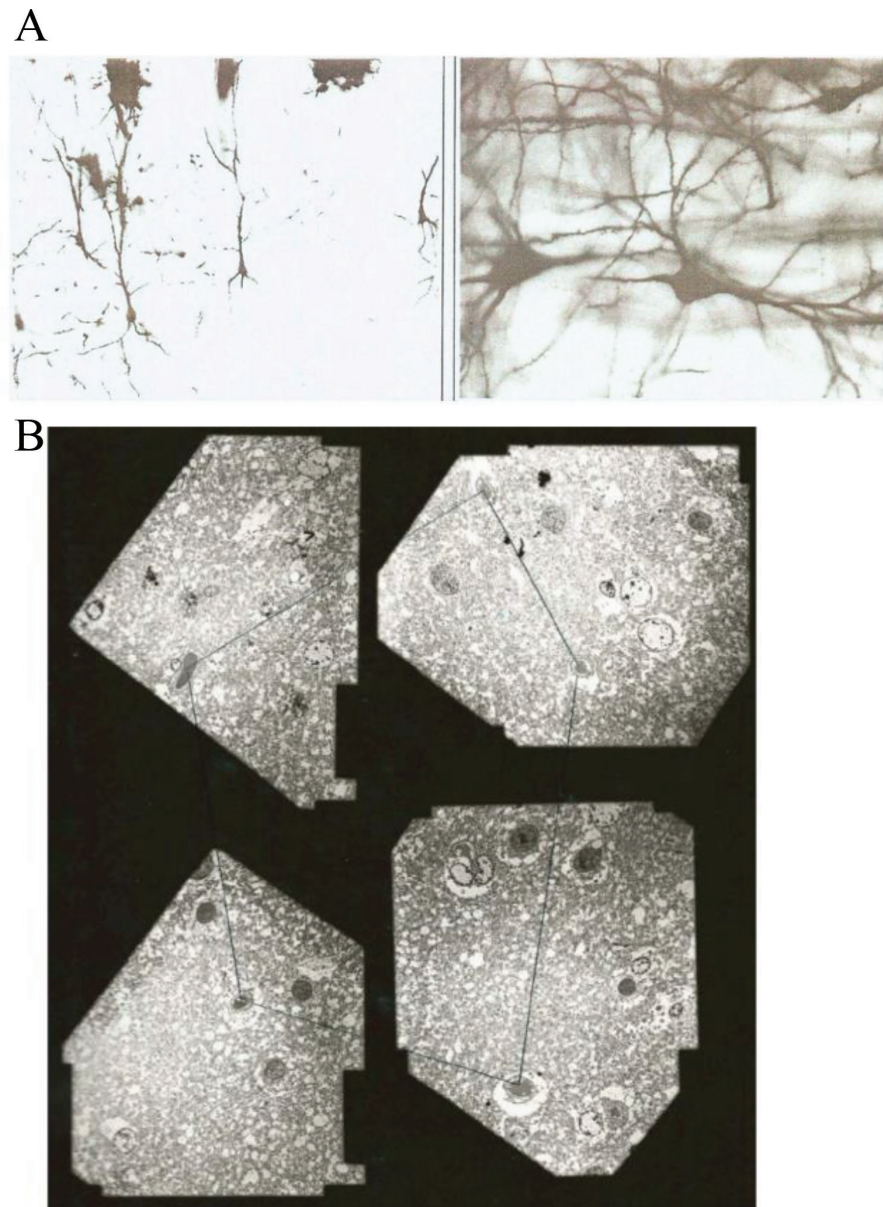
We have worked with human models from the cuts taken during anatomic-pathological autopsies of children around 8 years of age who died from accidents (Figure 2).

The findings corroborate the convergence/divergence composition from the third, pyramidal neurons of the cerebral cortex. The cortex in the hippocampus shows the same composition, and we think that this is a step for recruiting neurons and to form neural networks provide the structural basis of holograms, as shown in the following sketches [2, 13] (Figures 8–10).

So if anxiety can be accepted as a most common form of co-morbidity or “aura” in most of structural or functional alterations of the CNS, we must also consider the probable fact that



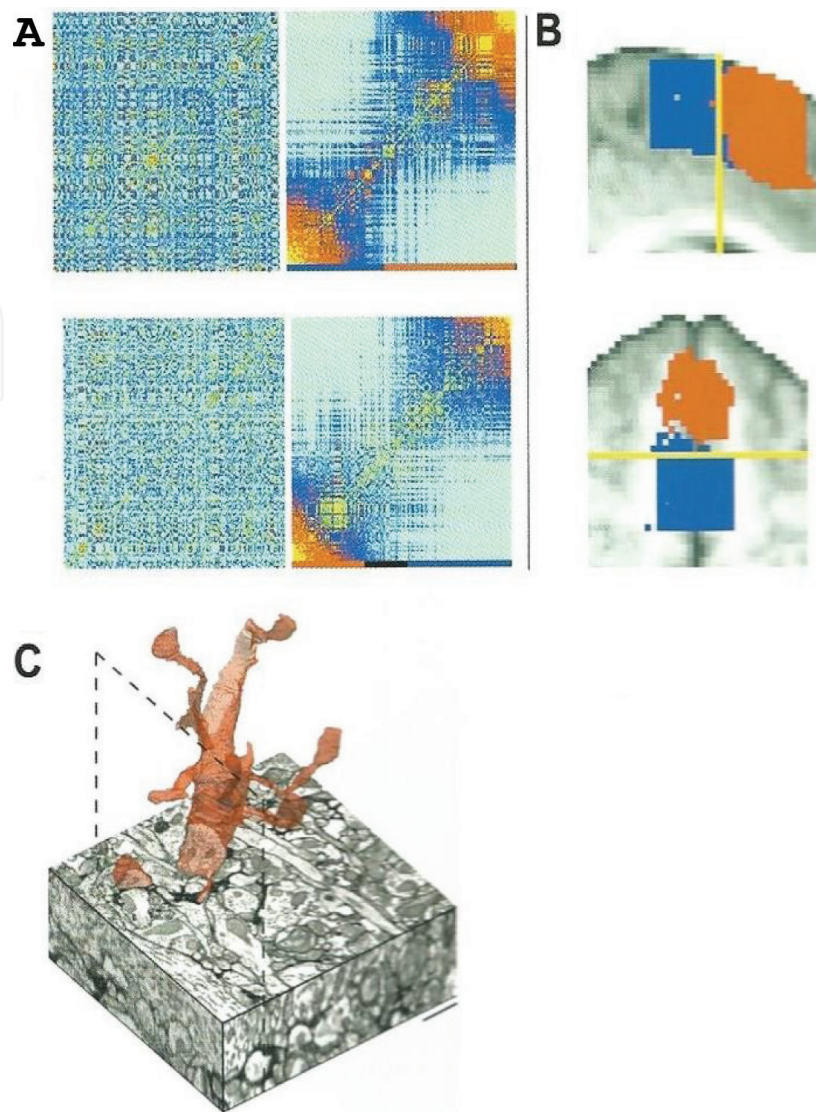
**Figure 8.** (A) Diagram showing the transformation of a photic stimulus and its amplification in the encephalon, generating the formation of an image and an associated memory. (Courtesy Pando et.al). (B) Diagram showing the phenomenon of amplification of information through the convergence/divergence principle in the time domain. (Courtesy Pando et al.).



**Figure 9.** (A) Microphotographs with optical microscopy of cells of the primary visual cortex that were studied morphologically in the present study. Pando-et. al. Pathology Hospital Infantil de México. (Courtesy of Pando, et al.) (B) Microphotographs showing the superposed lines of the geometrical relation of the cell bodies.

within the definition of epilepsy, it can and must also be considered as an epileptical equivalent [1, 3, 4, 6–11, 13].

Mechanisms underlying pathological characteristics have yet to be fully elucidated as stated by Nemeroff et al. (2003), but since then the biochemical research regarding GABA and receptor bioengineering of drugs that block GABA receptors, thus inciting in anxiety response, particularly with gabapentine and derivatives working as neuromodulators and compared to the use of benzodiazepines. This must be taken as a proof that anxiety and panic attacks [14]



**Figure 10.** From bidimensional formations through fractal mathematics, we obtain images as “holograms” that are the perception of each individual during the process of each individual’s perception and networks from the outside or inside world (A–C). Courtesy from Neuroradiology authored book by Stoop M, García R. *Advances in Diagnosis through Images*. 2010. Journal ed. Arg. (Spanish).

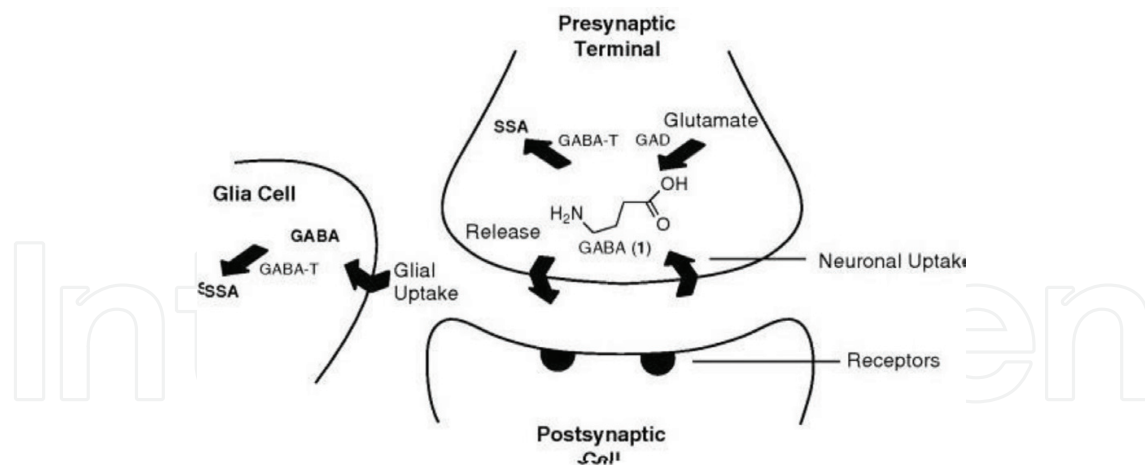
as well as post-traumatic stress [1, 15–17] respond to drugs that act within the context of neuromodulation of epileptical phenomena [18] (**Figures 11–13**).

So GABA deals with phosphorylation, highly reactive for activation of mRNA and process of transcription to produce antioxidative products, regulating these proceedings through NADPH and also regulates succinate and glutamate to stabilize neurons (includes Astroglía) (Shatsberg et al. 2008; Pando et al. 2017; Doctoral thesis National Politechnical Institute, National School of Biological Science) [18].

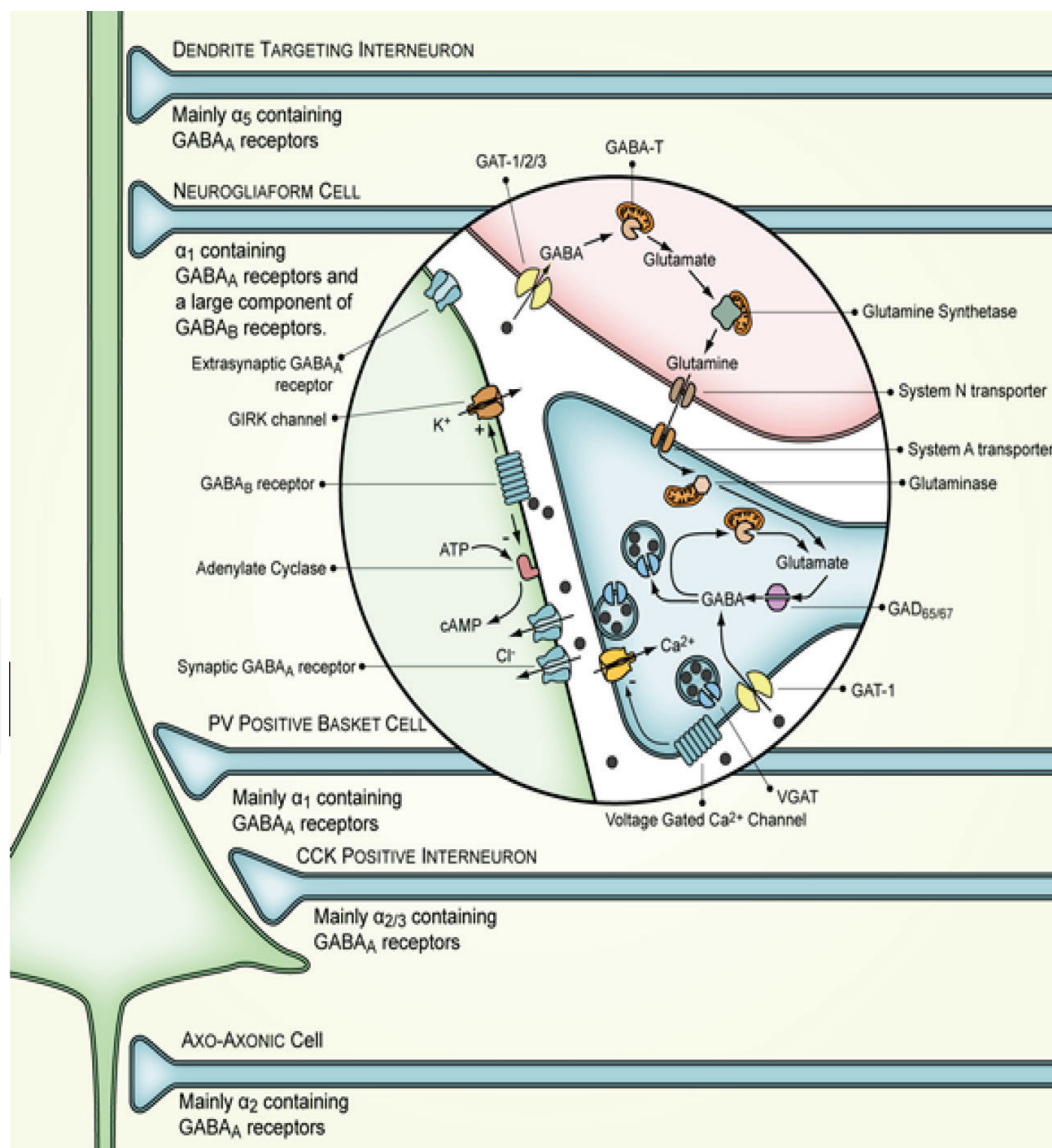
So which drugs interact with GABA if associated to anxiety?

The answer to this question goes rapidly to anticonvulsive drugs. More than inhibitors of the recapture of serotonin, and particularly at the subcortical level in the basic ganglia [18].





**Figure 11.** GABA synopsis diagram. Courtesy from Neurochemistry by Laguna et al. Biochemistry 2013, Unam, Mexico. Lehninger 6th edition and Nihon Yukirigaku, The life cycle of GABA. Faculty of Dental Science, Japan.



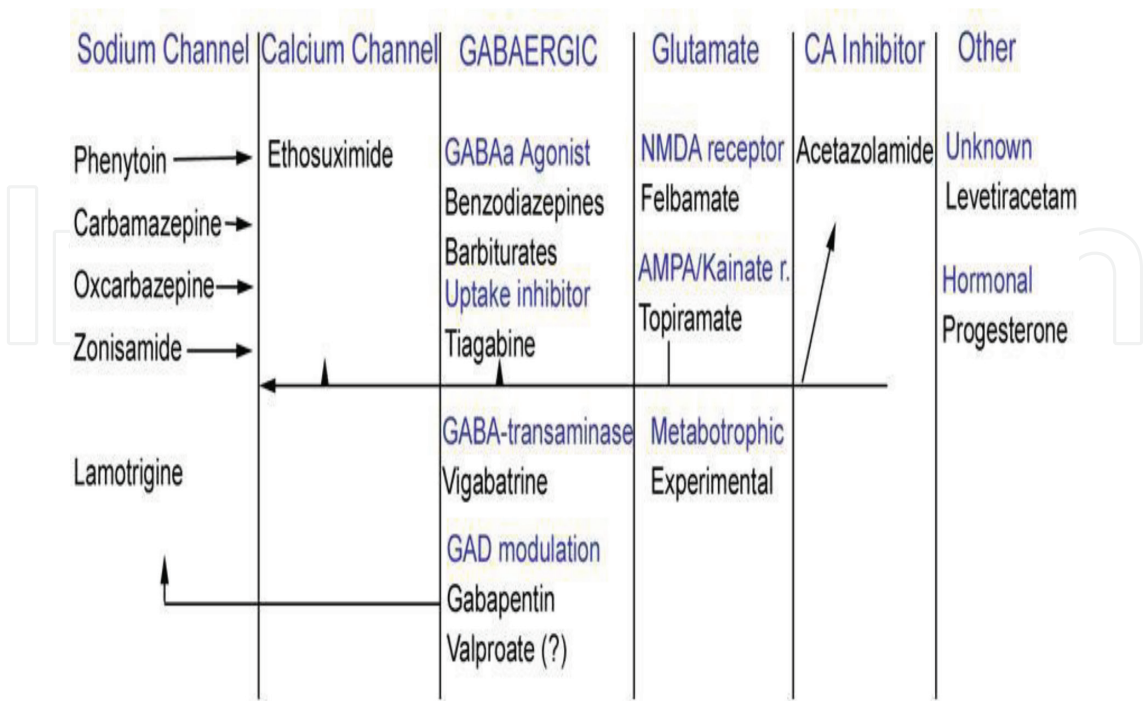
**Figure 12.** Drugs Associated to Gaba: Gaba receptors. Ubication of Gaba Receptors. (public scheme).



Natural Transmitter	Selected Associated Effects	Interacting Psychotropic Drugs
Acetylcholine	Learning, motor functions	Nicotine (at nicotinic sites)
Amino Acids Glutamate GABA	Excitatory responses, memory, learning Anxiety/relaxation, coordination, intoxication	Ethyl alcohol (ethanol) Barbiturates, benzodiazepines, ethanol
Monoamines Dopamine Norepinephrine Serotonin	Pleasure/reward, craving, mental illness Stress, arousal, learning, sleep Sleep, craving, mood regulation, anxiolysis	Cocaine, amphetamines, ethanol Cocaine, amphetamines Ethanol, MDMA, hallucinogens
Neuropeptides Enkephalins Beta-Endorphins	Pain response, learning, eating Pain sensitivity, euphoria, pleasurable effects	Heroin, morphine, other opioids Heroin, morphine, other opioids, ethanol

**Figure 13.** Functions of neuromodulators. From Hospital Fray Bernardino Alvarez resident’s internal operating manual, Mexico.

Antiepileptic drugs can be grouped according to their major mechanism of action. Some antiepileptic drugs work by acting on combination of channels or through some unknown mechanism of action (**Figure 14**).



**Figure 14.** Drugs and site of action. (Courtesy of Fray Bernardino Alvarez Hospital).

As seen, the proposition to treat anxiety with other neuromodulators, including fitopharmacology different from antidepressives or benzodiazepines, is a serious and documented stand in anxiety therapeutics [18].

Most literatures talk about anxiety, but a very few intend to define it, because it is a subjective feeling and even the DSM in any version talks about it as a comorbidity of many pathologies, but does not define it as such; the proposal is to promote anxiety as an epileptic entity; its definition as another variety of epilepsy when related to temporal lobe epilepsy or diencephalic alterations belonging to the limbic system [19–22].

## Conflict of interest

There are no conflict of interests in this manuscript. The funding comes from CPALC Mexico city (Centro Para la Atención de Lesiones Cerebrales).

## Author details

Luis A. Pando-Orellana

Address all correspondence to: [drpando@hotmail.com](mailto:drpando@hotmail.com)

CPALC/INCICH., Mexico City, Mexico

## References

- [1] Torta R, Keller R, et al. *Epilepsia*. 1999;**40**(Suppl. 10):s2-s20
- [2] Pando-orellana LA, Cabrera ML, Diaz Cintra S, et al. Modelo matemático de la relación funcional neural, basado en microestructura. *Revista Mexicana de Neurociencia*. 2016;**17**(3):3-1
- [3] Vera F, Pando-Orellana LA, et al. EEG in patients with anxiety disorder. *Revista Mexicana de Neurociencia*. 2013;**14**(6):335-340
- [4] Pando LA. et al. Medical addictions. *Revista Mexicana de Neurociencia*. 2014;**15**(3):179-182
- [5] Pando-Orellana LA et al. EVPOL questionnaire confiability and validity of the instrument in a sample of Mexican patients. *Revista Mexicana de Neurociencia*. 2009;**10**(6):443-445
- [6] Johnson EK, Jones JE, Seidenberg M, Hermann BP. The relative impact of anxiety, depression, and clinical seizure features on health-related quality of life in epilepsy. *Epilepsia*. 2004;**45**:544-550
- [7] Reuber M, Andersen B, Elger CE, Helmstaedter C. Depression and anxiety before and after temporal lobe epilepsy surgery. *Seizure*. 2004;**13**:129-135

- [8] Park SP, Song HS, Hwang YH, Lee HW, Suh CK, Kwon SH. Differential effects of seizure control and affective symptoms on quality of life in people with epilepsy. *Epilepsy & Behavior*. 2010;**18**:455-459
- [9] Ettinger AB, Wisbrot DM, Nolan EE, et al. Symptoms of depression and anxiety in pediatric epilepsy patients. *Epilepsia*. 1998;**39**(6):595-599. DOI: 10.1111/J1528-1157.1988.tb01427.X
- [10] Heicemovich H, Salpekar J, Kamer AM, et al. Suicidal and epilepsy: A neuropsychobiological perspective. *Epilepsy & Behavior*. 2011;**22**(1):77-84. DOI: 10.1016/j.yebeh.2011.04.059
- [11] Jacoby A, Snape D, Baker GA. Determinants of quality of life in people with epilepsy. *Neurologic Clinics*. 2009;**27**:843-863
- [12] Sacks O. *Migraine*. USA: Random House; 1992. Collection Vintage 1999
- [13] Schrödinger E. *Space, Time and Structure*. Cambridge Classics. Cambridge University Press; 1950. Reprint: 1985
- [14] Thompson SA, Duncan JS, Smith SJM. Partial seizures presenting as panic attacks. *BMJ*. 2000;**321**(7267):1002-1003
- [15] Markand ON, Salanova V, Whelihan E, Emsley CL. Health-related quality of life outcome in medically refractory epilepsy treated with anterior temporal lobectomy. *Epilepsia*. 2000;**41**:749-759
- [16] Jones JE, Jackson DC, Chambers KL, et al. Children with epilepsy and anxiety: Subcortical and cortical differences. *Epilepsia*. 2015;**56**(2):283-290
- [17] Kanner AM. Depression and epilepsy: Do glucocorticoids and glutamate explain their relationship? *Current Neurology and Neuroscience Reports*. 2009;**9**(4):307-312
- [18] Schatzberg AF, Nemeroff CB. *Textbook of Psychopharmacology*. 4th ed. The American Psychiatric Publishing; 2000
- [19] Hamit H, Blackmon K, Cong X, et al. Mood, anxiety, seizure control affect quality of life after epilepsy surgery. *Neurology*. 2014;**82**(10):887-894
- [20] Herman JP, Mueller NK, Figueiredo H. Role of GABA and glutamate circuitry in hypothalamic-pituitary adrenal axis stress integration. *Annals of the New York Academy of Sciences*. 2006;**29**
- [21] Pando-Orellana LA et al. Mental health? *Revista Mexicana de Neurociencia*. 2012;**13**(3): 168-169
- [22] Van Campen JS, Hessel EVS, Bombach K, et al. Stress and corticoids aggravate morphological changes in dentate gyrus after early life, experimental seizures in mice. *Frontiers in Endocrinology (Laussane)*. 2018;**3**. DOI: 10.3389/fendo.2018.eCollection 2018