

Case Report

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Electroencephalographic Finds - EEG According To 13 Cases of Patients Who Are Cocaine Chronic Users In Comparison To A Healthy Control Group of 10 Patients With Similar Characteristics



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Abstract

In chronic cocaine users, dysfunctional and structural findings have been found, the etiology of this dysfunction is unknown. In our group of patients MRI was completely normal and we have described a mild dysfunctional anomaly in the frontal cortex that is common denominator in this type of patients. The aim of this study is to investigate the cortical EEG activity of these patients. The question we ask is the following is cocaine which generates a dysfunctional disorder in the cortex of these patients or on the contrary on slightly dysfunctional brain the patient get into consumption of toxic.

Keywords: Cocaine; EEG; Frontal; Dysfunctional lesion; Slowing; Brain; Electroencephalogram; Cerebral areas; Drugs

Introduction

The frontal lobe is in charge of complex cognitive processes, called executive functions. These functions are mental operations which go to an end and allow a behavioral control, that is to say, they make possible that we can choose, plan and take voluntary and conscious decisions. The prefrontal cortex has the decisive role in the executive functions by integrating the information and allowing the choice of objectives and the organization of the action plans to do them. It constitutes a cerebral region which is one of the most phylogenetically recent and also it is the last one to mature in the ontogenesis, the most developed in the evolutionary scale [1]. It is also involved in the social-emotional processing and control, in the cooperative work. Its responsibility in frontal functions is caused to his conexión with the amígdala (which interferes in the memory) and motivation system (limbic system) and dopamine gratification. Self-control is an important function of the frontal lobe.

Cocaine effect in the brain: within the normal process of communication, neurons release dopamine inside of the synapses, where it joins to dopamine receptors in adjacent neurons. Normally, a specialized protein called “transporter of dopamine” recycles the dopamine by returning to the

transmitting neuron. When cocaine has been consumed, this one is adhered to the transporter protein of dopamine and blocks the normal process of recycling, resulting in an accumulation of dopamine in the synapses, which magnifies or exaggerates the pleasurable effects of cocaine (Figure 1).

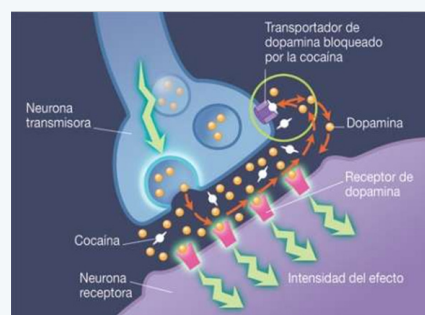


Figure 1: Cocaine effect in the brain.

Methods and material

We conducted a descriptive, longitudinal and retrospective study. It consists of the morphological electroencephalographic (EEG) analysis of 13 patients who are cocaine users in comparison

Sweep: 10 milliseconds. 10 seconds per screen.
Sensitivity 7 microvolts.

II. Clinical case 2: normal EEG (Figure 3) See the absence of specific inter hemispheric calculable anomalies.

III. Clinical case 3: 36-year-old male from the Psychiatry outpatient clinic. In the tracing it is shown an EEG activity within normal limits. It is also shown a frequent and attenuated activity on voltage, with a low incidence in the (background) tracing with acute morphology which can be compatible with bitemporal mild vascular focus of low incidence in the (background) tracing, in right and left fronto-temporal region, being more noticeable on the left side. Vascular activity goes to 7 Hz.

TAC brain without contrast: No signs of acute intracranial pathology.

CAT brain without any contrast: no evidence of acute intracranial pathology.

IV. Clinical case 4: 50-year-old male from Psychiatry outpatient clinic. He has been cocaine user for several years. It is found an EEG tracing in its normal limits. It is registered a focused slight activity in the left and right hemisphere, and it is marker on left side [12]. It is also shown a slight incidence and an electric impact on the rest of the (background) tracing. We can see sharp and slow waves morphology with moderate voltage in the left frontal region; that activity slows slightly the area on which it stabilizes, at a frequency of 7 Hz when this patient's alpha is 9 Hz.

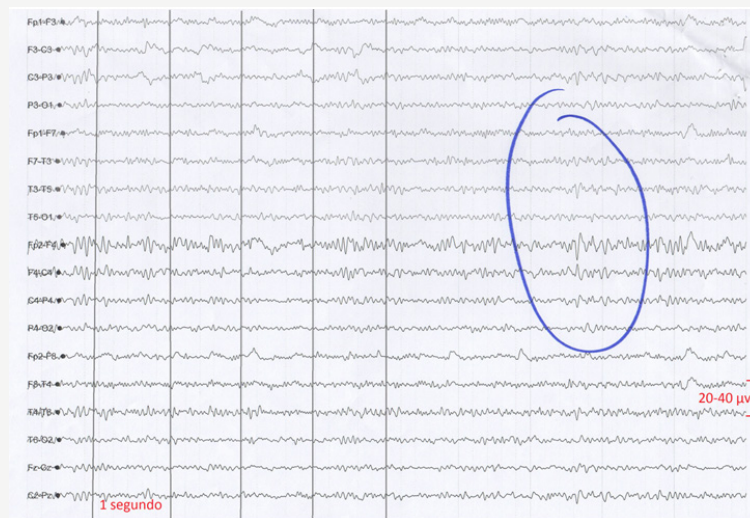


Figure 4: High filter: 30 Hz. Low filter: 0.5 Hz.

Sweep: 10 milliseconds. 10 seconds per screen.

Sensitivity 7 microvolts.

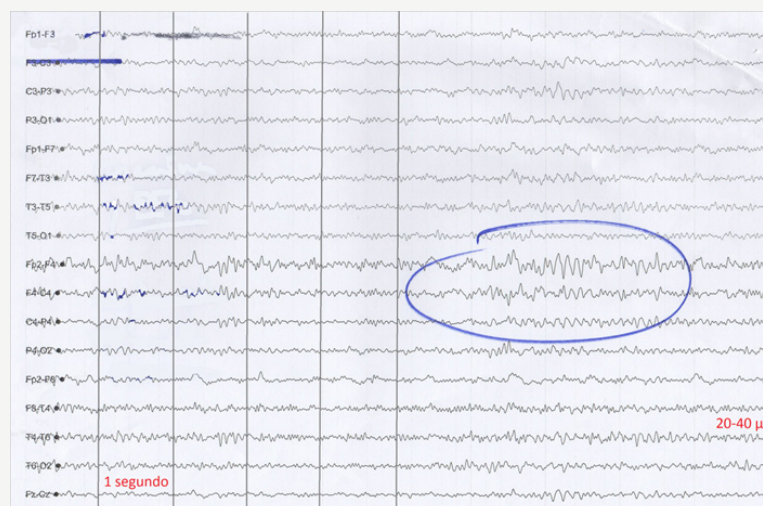


Figure 5: Filtro alto: 30 Hz. Filtro bajo: 0.5 Hz.

Barrido: 10 milisegundos. 10 segundos por pantalla.
Sensibilidad: 7 microvoltios. Sensitivity 7 microvolts.

High filter: 30 Hz. Low filter: 0.5 Hz.
Sweep: 10 milliseconds. 10 seconds per screen.

V. Clinical case 5: 51-year-old woman from Psychiatry outpatient clinic. It is found an EEG tracing in its normal limits. It is registered a slight focused activity in both right and left hemisphere, although it is more pronounced on right side. In addition to this, there is a slight incidence and an electric impact on the rest of the tracing, with a moderate voltage activity in the right frontal region at a frequency of 7 Hz when the patient's alpha is 9 Hz. The activity lasts from 0.5 to 1 msec. Without alterations in the brain magnetic resonance imaging (MRI) scan (Figures 4 & 5).

VI. Clinical case 6: 47-year-old male from Psychiatry outpatient clinic. Cocaine user. It presents normal (background) tracing, with regional activity both in left and right hemisphere, but being more noticeable on left side. There is a moderate incidence and an electric impact on the rest of the tracing, with slow waves morphology, that slow down the area at a frequency of 5-7 Hz. The voltage

is moderate in fronto-temporal region with irradiation to the left parietal area and low diffusion to the right parietal one; that activity lows down at a frequency of 5-7 Hz when the tracing (de fondo) is 9 Hz. The activity last from 0.5 to 1 msec.

VII. Clinical case 7: 42-year-old male who is derived from Psychiatry outpatient clinic. It is observed an activity (background) with normal limits in the tracing, and also it presents diffuse beta Rhythms of probable pharmacological origin with irregular alpha rhythms. A focused, moderate and bilateral activity, mostly in left side and with a frequency of 7-8 Hz when the alpha goes to 10-11Hz. The bilateral frontal lobe is partially slowed down and with left side predominance [13-17]. These slow activities last from 0.5 to 1 msec. Without any alterations in the Cerebral RMN (Figures 6 & 7).

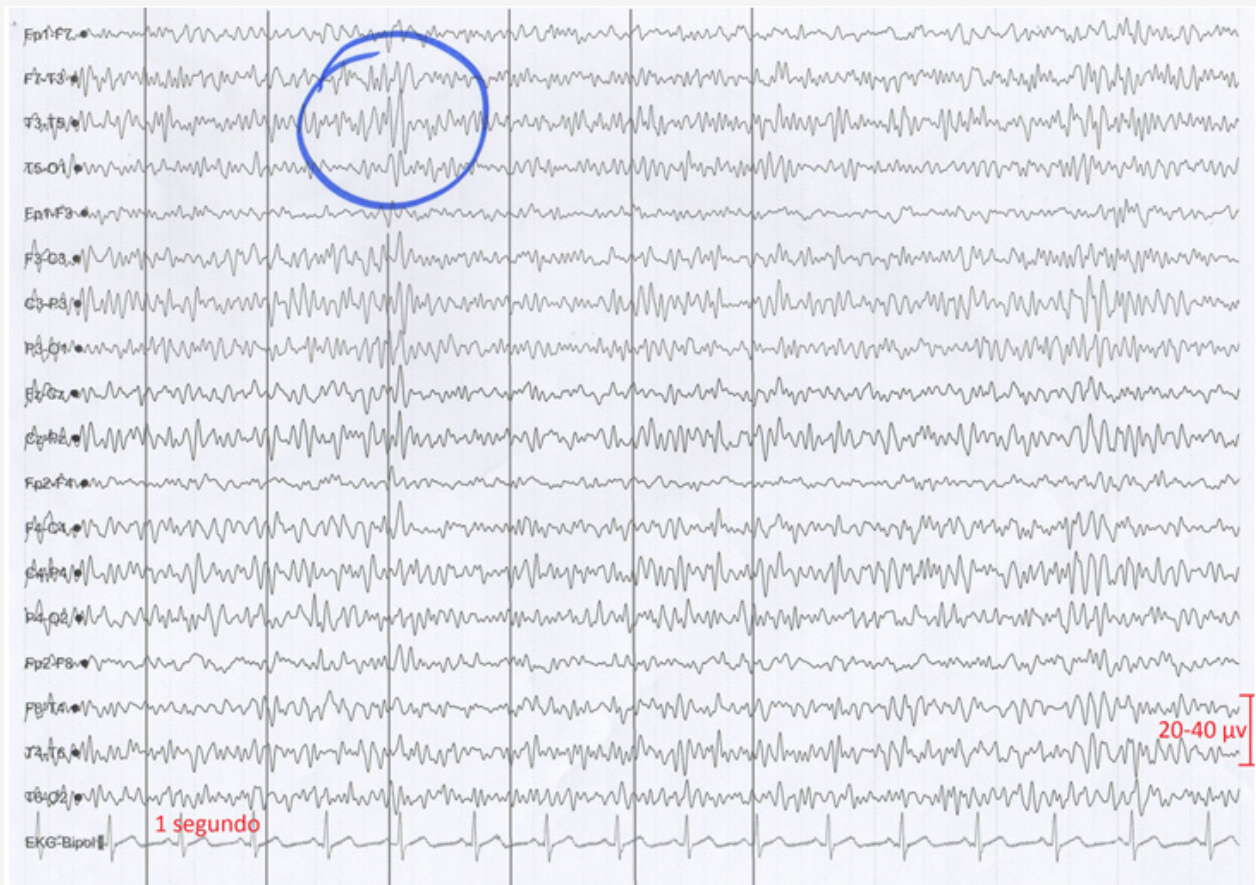


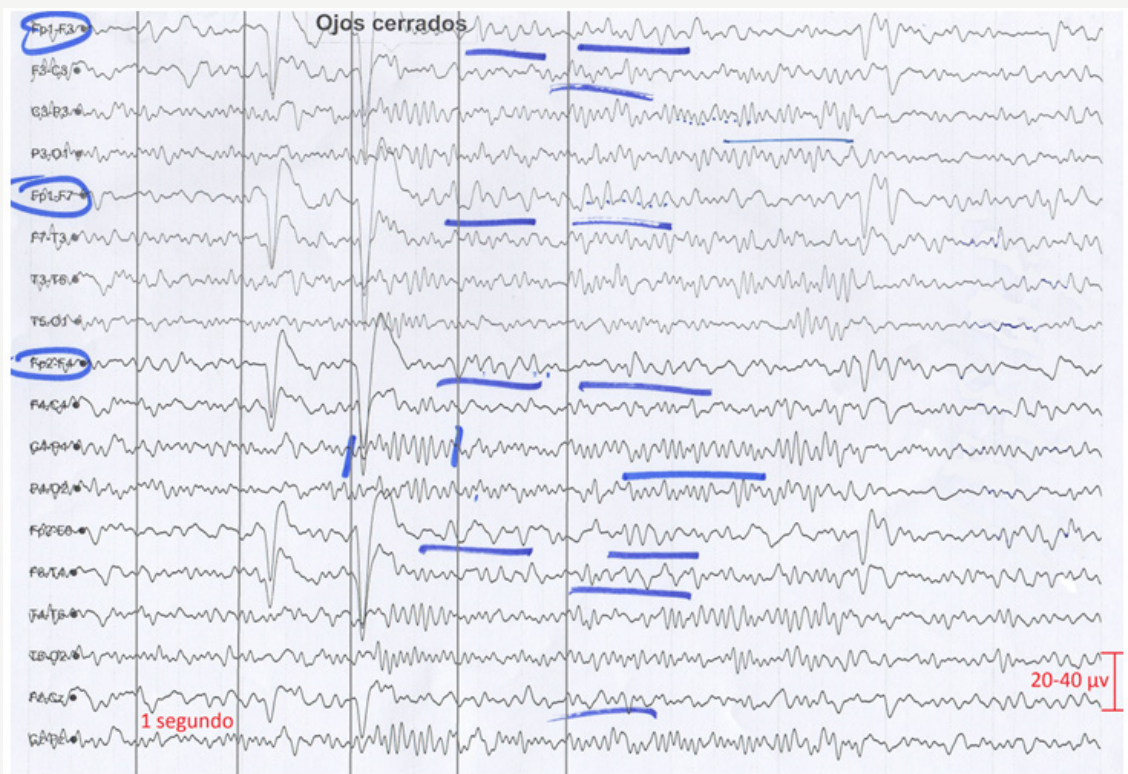
Figure 6: High filter: 30 Hz. Low filter: 0.5 Hz.

Sweep: 10 milliseconds. 10 seconds per screen.

Sensitivity 7 microvolts.

VIII. Clinical Case 8: a 25-year-old male is derived from the external consultation of psychiatry. Observations show an activity (background) with normal limits in the tracing. Malfunction frontal left and parietal left with frequency from

5-6 Hz. to 0.5-1 msec of duration. This activity slows down the bilateral frontal activity with left side predominance. Without alterations in the brain magnetic resonance imaging (MRI) scan (Figures 8-10).



Sweep: 10 milliseconds. 10 seconds per screen.
Sensitivity 7 microvolts.

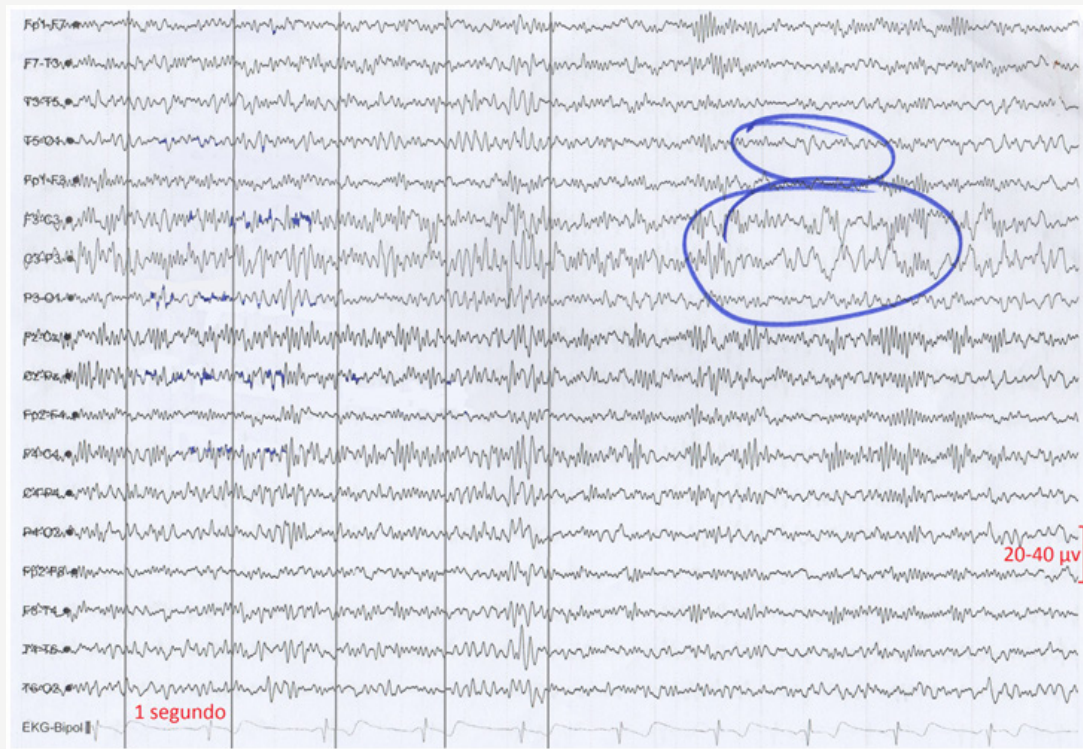
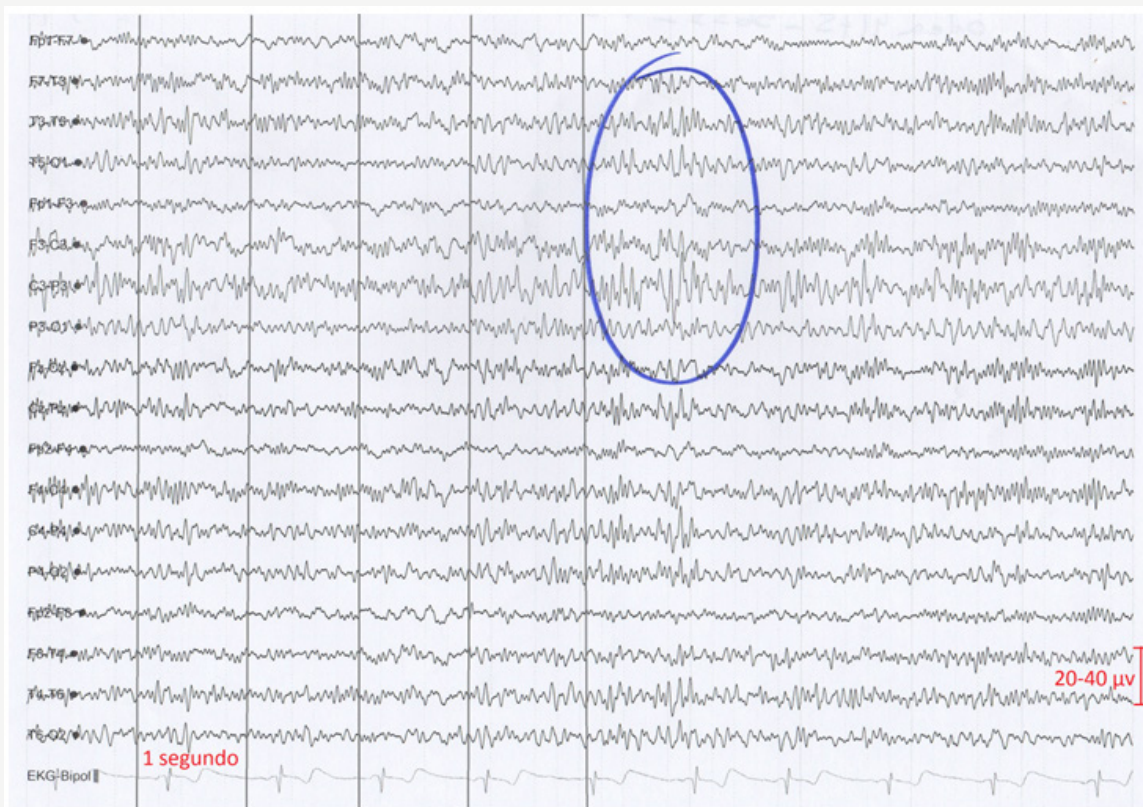
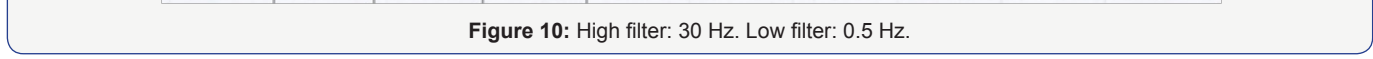
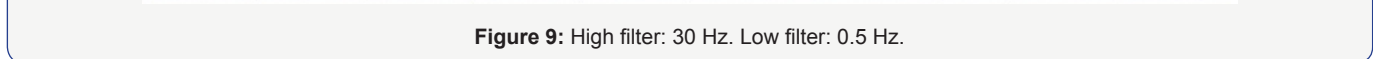


Figure 8: High filter: 30 Hz. Low filter: 0.5 Hz.

Sweep: 10 milliseconds. 10 seconds per screen.

Sensitivity 7 microvolts.





Sweep: 10 milliseconds. 10 seconds per screen.

Sensitivity 7 microvolts.

IX. Clinical case 9: 30-year-old male derived from the external consultation of psychiatry. Normal tracing of 9 Hz

with a slow activity in the left front-temporal región with a frequency of 7 Hz. They present common information to the previous EEG cocaine consumers. Normal skull TAC (Figure 11)

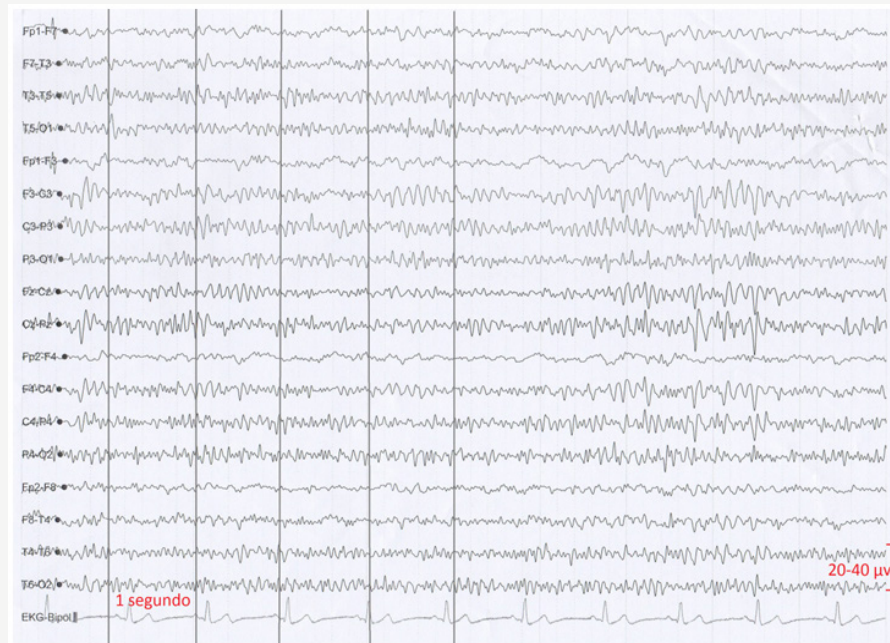


Figure 11: High filter: 30 Hz. Low filter: 0.5 Hz.

Sweep: 10 milliseconds. 10 seconds per screen.

Sensitivity 7 microvolts.

X. Clinical case 10: 34-year-old male derived of

Psychiatry outpatient clinical. We can observe a dysfunction in bilateral front-temporal region. There is not any alterations in the Cerebral RMN (Figure 12).

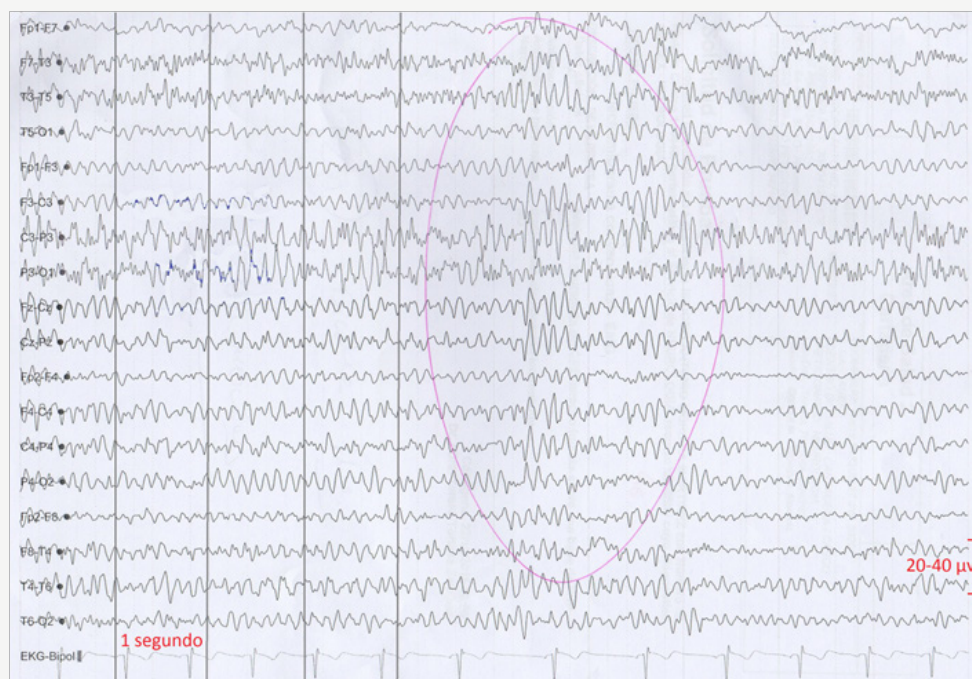


Figure 12: High filter: 30 Hz. Low filter: 0.5 Hz.

Sweep: 10 milliseconds. 10 seconds per screen.

Sensitivity 7 microvolts.

XI. Clinical case 11: 40-year-old male from the psychiatry outpatient clinic. Violent conduct and cocaine user. We notice a brain bioelectric activity which consists of a mixture of frequencies of 10 Hz with parieto-occipital Alpha rhythm, and fast rhythms in previous areas. There is a dysfunction in right front-temporal region, 7 Hz frequency that radiates a little to the left hemisphere. Without any alterations in brain MRI.

XII. Clinical case 12: 27-year-old male from psychiatry outpatient clinic. Focused and moderate activity and with moderate incidence in the tracing (background) We can

see a focused activity in right front-parieto-temporal region, of sharp morphology that slows regionally the tracing, and with a frequency of 5-6 Hz, clearly more focused in the right hemisphere, in parieto-temporal region. We can see a slight incidence and an electric impact in the rest of the tracing (background), with sharp and slow waves morphology, moderate voltage involved in F4, C4., P4, T4, T6 regions and with mild diffusion to the adjacent C3-P3 area, activity with nonspecific morphology for epilepsy, with the same reactivity in the tracing (background), that excels partially and constitute a slight inter-hemispheric asymmetry. The ELI with 5 -10 Hz generates a very characteristic drug in this type of patients [18-21]. The HV slows down in a physiological way the (background) tracing. Without alterations in brain MRI (Figures 13 & 14).

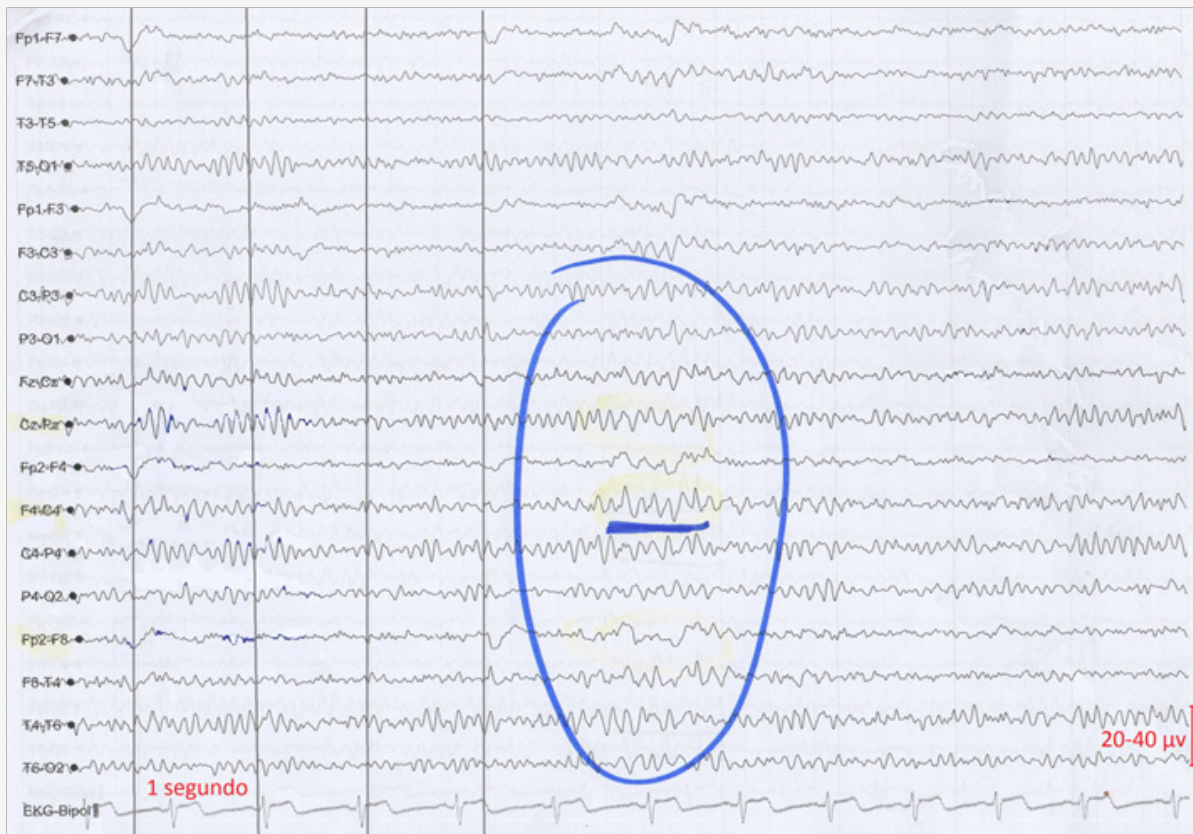


Figure 13: High filter: 30 Hz. Low filter: 0.5 Hz.

Sweep: 10 milliseconds. 10 seconds per screen.

Sensitivity 7 microvolts.

XIII. Clinical case 13: 48-year-old woman who is derived from psychiatry outpatient clinic. Normal brain bioelectric activity, which consists of a diffused beta rythm in relation to an ontogeny caused by benzodiazepines. There is not any brain RMN alterations.

XIV. Clinical case 14: 24 year-old male derived from Psychiatry outpatient clinic. He is a prior ethyl user, cocaine

and cannabis user, nictameral deprivation. We can see Irritative EEG signs of focused character in both right and left front-central region. There is a tendency to generalize with hyperventilation. Normal caneal NMR. Signal Intensity and morphology changes in temporal lobes are not detected. Periventricular or peripheral alterations are not found either.

XV. Clinical case 15: 19-year-old male derived from psychiatry outpatient clinic. We can notice a slight slowing down in the EEG tracing (background) which is compatible

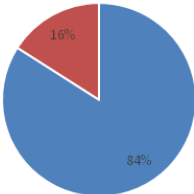
8-9 Hz, that means a progress in the cerebral bioelectrical tracing.



Right Front-Parietal-Temporal	1
Left Front-Parietal-Temporal	1
Front-Central Bilateral	1

Results (Figure 15)

Sexo



A pie chart titled 'Sexo' showing the distribution of sex. The chart is divided into two segments: a large blue segment representing 'Hombres' at 84%, and a smaller red segment representing 'Mujeres' at 16%. A legend at the bottom identifies the colors: blue for 'Hombres' and red for 'Mujeres'.

Sexo	Percentage
Hombres	84%
Mujeres	16%

■ Hombres ■ Mujeres

In our study there is an 84% male and a 16% female, aged between 19 and 51 years old, and the average is 36 years old.

None clinical case out of all 13 contains any alteration in cerebral nuclear magnetic resonance (RMN) (Figure 16).

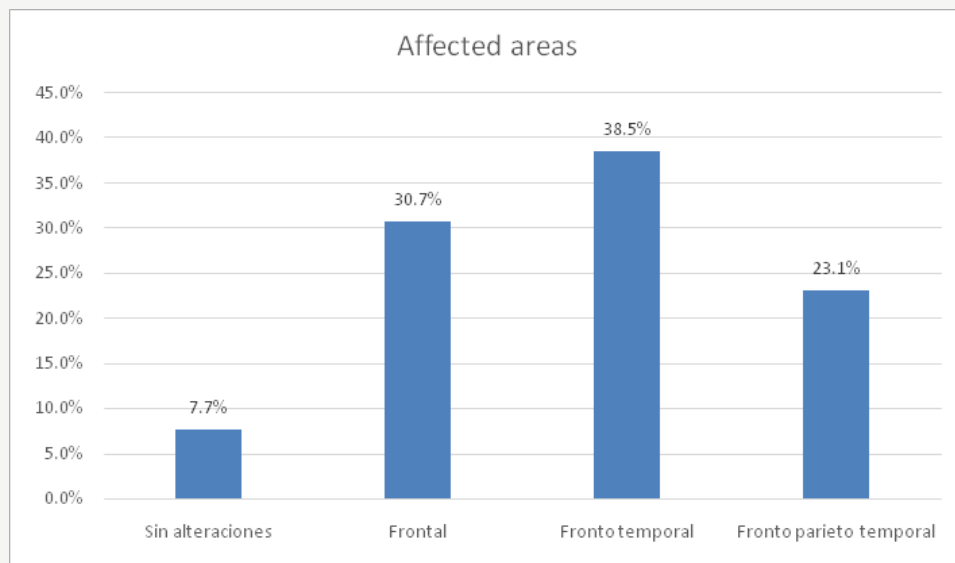


Figure 16: Chart showing affected areas.

84.6% out of the electroencephalograms of our study reveal the existence of minimum cortical dysfunctions in frontal areas with more or less spread to adjacent cortical areas. The most affected area with a 38.5% is the front-temporal one with higher predominance of the left hemisphere. These slow activities are often registered at a 7 Hz frequency when the normal (background) tracing is 9-10 Hz. That activity lasts from 0.5 to 2 msec (Figure 17).

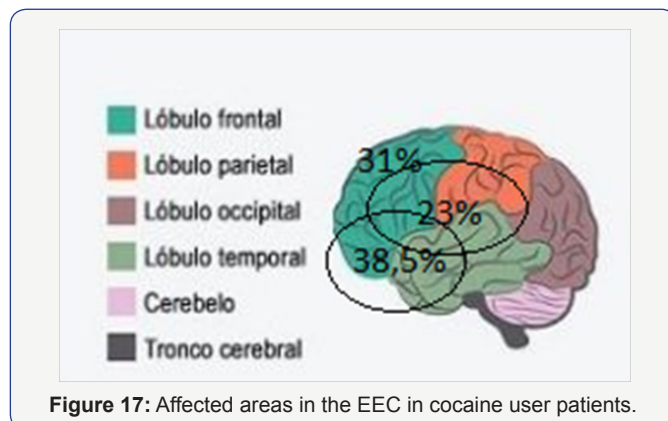


Figure 17: Affected areas in the EEC in cocaine user patients.

Conclusion

Frontal area is responsible for self-control, so an alteration in frontal lobe causes alteration of the executive functions, mental deterioration, personality and desinhibition behavior alterations.

Images of the brain show a decrease in dopamine receptors (D2) in a person's brain with cocaine addiction in comparison to someone who does not consume drugs.

These minimum dysfunctional injuries support the theory, which is so often seen in the literature, that not all pathology is structural, but there is pathology with minimum dysfunctional or metabolic lesions without an associated anatomopathological

substrate. That is why our study defines the importance of the EEG in the diagnosis of dysfunctional disorders like ADHD, schizophrenia, and these 13 chronic patients of cocaine with MML anodyne, as we have seen.

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