The Contribution of Numerical EEG Analysis for the Study and Understanding of Addictions with Substances

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***Abstract*—Computerised electroencephalography (EEG) is one of a wide variety of brain imaging techniques used in addiction medicine. It is a sensitive measure of the effects of addiction on the brain and has been shown to show changes in brain electrical activity during addiction. But, the clinical value of computerised EEG recording in addictions is not yet clearly established. However, several studies argue that this non-invasive technique has an undeniable contribution to the understanding, prediction, diagnosis and monitoring of addictions. The aim of this article is to assess, through a systematic review, the contribution and interest of computerised EEG in the study and understanding of substance abuse by describing the different electrical activities that underlie it across the main frequency ranges: delta, theta, alpha, beta and gamma. We have been conducting a systematic review according to the recommendations of Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) and the Cochrane Group. We included 25 studies with a total of 1897 cases of addiction and 1504 controls. The studies dealt with addictions related to 05 licit and illicit psychoactive substances (alcohol, nicotine, cannabis, heroin and cocaine). The group of addicted patients showed significantly different brain electrical characteristics from the group of controls in the different EEG rhythms, whether during acute substance intoxication, abuse, withdrawal, abstinence, relapse, progression or response to treatment. The majority of studies have used EEG for diagnostic, predictive, monitoring purposes and also to discover electro-physiological markers of certain addictions.**

Keywords*—Electroencephalography (EEG); quantitative electroencephalography; drug addiction; spectral analysis; coherence analysis

I. Introduction

Over the past twenty years, we have witnessed some important advances in the study of the human brain. Possibly the most challenging has been the development of structural and functional brain imaging techniques, which have revolutionised cognitive and behavioural neuroscience by offering us a view into the brain activity underlying complex human behaviour. These technological progresses have also allowed to the rapid conversion of basic neuroscience discoveries into more specific therapies for clinical application.

There is a rich diversity of brain imaging techniques, which can be categorised into three main types: (1) nuclear medicine imaging techniques; (2) magnetic resonance imaging techniques and (3) electro-physiological imaging techniques, which comprise electroencephalography (EEG). Each one of these techniques reveal a distinct facet of brain structure and/or function, providing a wide range of findings on the biochemical, electro-physiological and functional processes of the brain.

Electroencephalography (EEG) has been used to investigate brain function since the publication of Berger's paper in 1929. It registers the synchronised activity of excitatory (EPSP) and inhibitory (IPSP) postsynaptic potentials in the cerebral cortex and exhibits the activity as a tension change in amplitude with time [1]. Over the past forty years, the EEG has been used extensively in drug addiction research. It is known to be a sensibly measurement of the effects of substances on the brain and, in particularly, of the effects of drugs on the size and time course of postsynaptic potentials [2]. The enhancing effects of many substances modulated by the mesolimbic dopamine (MD) channel have been shown to alter EEG recordings [3]. Five frequency ranges are generally recognised and studied: delta (0-4 Hz), theta (4-8 Hz), alpha (8-13 Hz), beta (15-30 Hz) and gamma (<30 Hz).

EEG analysis methods included quantitative EEG, spectral analysis, coherence analysis and visual analysis. Substance use has been found to be associated with broad alterations in intrinsic neural activity, typically expressed as neuronal hyperactivation and decreased neuronal communication between brain regions. Some studies have shown that the use of alcohol, tobacco, cocaine, cannabis and heroin was positively correlated with these changes. These alterations may partially recover after abstinence, which differs between drugs and may reflect their degree of neurotoxicity. In summary, EEG can be used for diagnostic, predictive and monitoring purposes and also to discover electrophysiological biomarkers of certain addictions.

Therefore, this article presents a systematic review of EEG studies in substance addiction, to guide further work in the field. The main objective is to review the empirical research of the last decades, providing crucial information on EEG findings in addiction to the most commonly used substances. It therefore aims to investigate the interest and contribution of EEG in these addictions.

II. METHOD

A. Inclusion and Exclusion Criteria

The selected studies included people with addiction, without restrictive geographical, age, gender, ethnicity or nationality criteria. To be included in this review, studies had to be based on samples of subjects with substance addiction investigated by electroencephalography as the main investigative tool.

In order to limit the abundance of data from different databases, the studies included in this review were those that could provide us with data dealing with the role or use of EEG in addictology in English or French only. All were concerned with the study of brain activity in addicts. They were essentially quantitative studies carried out on addicted subjects without any other concomitant pathology. Priority was given to studies that used quantitative EEG with coherence analysis or spectral analysis.

We excluded any papers that did not directly address the brain activity of addicts as measured by EEG. For example, we eliminated all studies that only dealt with the use of other means of brain investigation such as scans, MRIs, PET scans, etc., without a direct link to the EEG.

B. Research Strategy

MEDLINE, PSYCHINFO, PUBMED, SCIENCE DIRECT and GOOGLE SCHOLAR databases were searched in January, February and March 2022 with publication date limitation since 1990 when computerised automated EEG analyses began using the following search terms: "Electroencephalography, EEG, substance addiction, drug abuse". Both authors (M.A. and D.J.) independently examined the title, abstract and keywords of each identified reference and then selected the studies according to the inclusion criteria. The same method was then used to review the full text of each selected study. In order to identify additional relevant articles that this search strategy would have missed, we also managed and examined the bibliographic references of each article included in the review using ZOTERO software. In case of incongruence between the data extracted by authors M.A. and D.J., the review and opinion of author E.F. was sought.

The initial database search identified 165 references. Following an initial review of titles, abstracts and keywords, 61 studies were not included because they did not correspond to the inclusion criteria. Following a full text review, 25 studies were included in the systematic review. The search strategy is detailed in a flow chart (Fig. 1).

C. Extracted Data

The authors (M.A. and D.J.) independently extracted data from the articles included in the review. A list of analysis criteria was compiled in the form of a reading grid (Table I) to extract data from the articles: author and year of publication, country, characteristics and size of the sample, substance studied, method of electroencephalographic analysis and the main rhythms studied.

The different types of EEG analysis that will be discussed in this paper are: 1) Quantitative analysis (QEEG) which is a modern type of EEG analysis that involves recording digital EEG signals that are processed, transformed and analysed using complex mathematical algorithms; 2) Spectral analysis, which is a frequency analysis technique that breaks down a complex cyclic signal into several sub-functions. This analysis technique is used in particular to finely dissect the electroencephalographic (EEG) signal. It provides information on fluctuations in the quantity and strength of cortical activities that are not visually perceived during the inspection of the trace; 3) EEG coherence, defined as the frequency-normalized cross-power spectrum of two signals recorded simultaneously at different scalp locations, is a sensitive method for assessing the integrity of the structural connection between brain areas, describing the temporal, spatial and frequency relationships of brain activities and 4) Visual analysis of the EEG: It relied heavily on waveform recognition, the EEG trace during wakefulness is much less synchronous than during sleep, but more homogeneous. The amplitude of the awake EEG trace in adults varies on average between 10 and 50 microvolt and the frequency of the wave's ranges from 0.3 to 70 Hz.

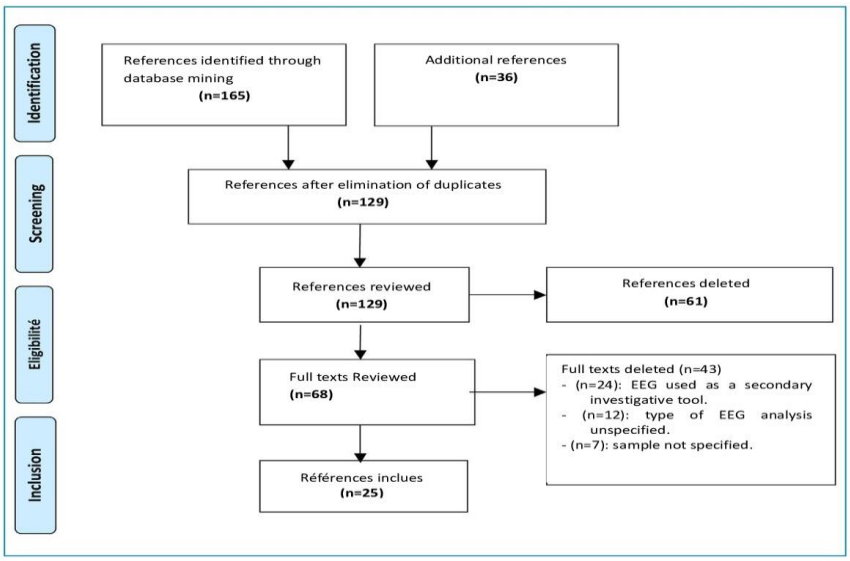

Fig. 1. Flow chart of the search strategy.

TABLE I. LIST OF INCLUDED STUDIES

Author	Year	Country	Sample size				Addiction studied	EEG and Analysis method	Main rhythms studied:				
			case		control				yes (1) no (0)				
			M	F	M	F			α	β	δ	θ	γ
[4] Levin KH et al.	2007	USA	16	4	8	0	Cocaine	Quantitative EEG	1	1	1	1	0
[5]Bauer, L. O.	2001	USA	73	34	13	9	Alcohol, heroin and cocaine	Quantitative EEG	0	1	0	0	0
[6] Böcker, K.B.E.	2010	Netherlands	16	0	0	0	THC	Quantitative EEG	0	0	0	1	0
[7] Rass et al.	2016	USA	13	9	14	16	tobacco	Spectral analysis	1	1	1	1	1
[8]Cortes-Briones, J.	2015	USA	14	6	0	0	THC	Quantitative EEG	0	0	0	0	1
[9]Costa, L., & Bauer, L.	1997	USA	63	25	10	4	Heroin, alcohol and cocaine	Quantitative EEG	0	1	0	0	0
[10]Domino EF.	2003	USA	65	0	20	0	tobacco	Quantitative EEG	1	1	1	1	0
[11]Ehlers C.L.	2010	USA	141	147	118	220	Cannabis and alcohol	Quantitative EEG	0	1	1	0	1
[12]Fingelkurts, A.	2006	Finland	14	8	6	8	Opioids	Visual analysis	1	1	0	0	0
[13] Herrera-Morales et al.	2019	Mexico	28	17	22	26	alcohol	Quantitative EEG	1	1	1	1	0
[14]Franken, I. H. A.	2004	Netherlands	18	0	12	0	heroin	Coherence analysis	0	1	0	0	1
[15]Grace Y. Wang	2015	New Zealand	29	20	14	11	Methadone and opiates	Spectral analysis	0	1	0	1	0
[16]Herning, R. I.	2008	USA	48	27	18	15	Marijuana	Quantitative EEG	1	1	0	0	0
[17]Herning, R. I.	1994	USA	14	0	0	0	Cocaine	Quantitative EEG	1	1	0	0	0
[18]Polunina, A. G., & Davydov, D. M.	2004	Russia	33	0	13	0	Heroin	Spectral analysis	1	0	0	0	0
[19] Prichep, L. S.	1996	USA	42	25	0	0	Crack	Quantitative EEG	1	0	1	1	0
[20]Rangaswamy M, et coll	2003	USA	150	157	150	157	alcohol	Spectral analysis	0	0	0	1	0
[21]Rangaswamy M, et coll	2004	USA	94	77	89	115	alcohol	Spectral analysis	0	1	0	0	0
[22]Rangaswamy M,.	2002	USA	150	157	150	157	Alcohol	Spectral analysis	0	1	0	0	0
[23]Reid, MS.	2006	USA	11	2	0	0	Cocaine	Spectral analysis	1	1	1	1	0
[24]Saletu-Zyhlarz, G. M.et coll.	2004	Austria	15	7	15	7	Alcohol	Spectral analysis	1	1	1	0	0
[25]Shikha,P et col.	2018	USA	17	6	21	9	Cannabis	Quantitative EEG	0	1	1	1	1
[26]Struve, F. A et col.	1998	USA	15	0	57	0	Cannabis	Quantitative EEG	1	1	1	1	0
[27]Teneggi V et col.	2004	Italy	12	0	0	0	tobacco	Spectral analysis	1	1	1	1	0
[28]Winterer, G., et coll	1998	Germany	45	33	0	0	alcohol	Quantitative EEG	1	1	1	1	0
			Σ =1897		Σ =1504								

III. RESULTS

A. EEG and Substance Addiction

1) Alcohol: Several studies have found a more accentuated theta rhythm in alcoholics when compared to corresponding controls, theta power appears higher in the central and parietal regions in male alcoholics, and in the parietal region in female alcoholics[20]. The offspring of alcoholics do not show an increase in resting theta, which suggests that this measurement may signify a state of alcohol dependence [29].

Previous EEG mapping studies have found increased beta power and decreased alpha and delta/theta power in de-addictive alcoholic patients, compared to normal controls. Since slow activity is considered inhibitory, fast beta activity excitatory and alpha activity an expression of normal brain function, the desynchronised low-voltage fast patterns can be translated as CNS hyperexcitation [30], [31].

Opinions as to the cause of these EEG changes in alcoholics show that incoherencies in brain function, particularly in frontal parts of the brain, may be participating in the development of alcoholism [5], [30], [31]. This is documented by a variety of studies: beta activity has been linked to the combination of the two pre-morbid factors of childhood behaviour disorders and paternal alcoholism. [5]. As well as subjects with a positive family history of alcoholism have EEGs characterized by an increase in relative beta power and a reduction in absolute and relative alpha power in both the occipital and frontal regions. Also Hazardous alcohol consumption (HAC) is a pattern of alcohol use that may result in harm for the user and/or for those around them. Prior research has suggested that HAC and alcohol dependence share some neurophysiological features but differ in others, the HAC group presented with higher beta absolute power and relative power, as well as a lower beta mean frequency than the control group, while the group with risk of alcohol dependence presented lower delta absolute power than controls [13]. Therefore, the majority of this research has found that alcoholics are distinct from controls in that their beta power is increased [5], [22].

Furthermore, beta power is faster in relapsing alcoholics than in abstainers[5], which suggests that desynchronised beta activity may be a precious indicator of relapse in abstinent alcoholics. Given that, the augmentation of beta power in abstinent alcoholics is not correlated with the period of abstinence[22] and is also found in children of alcoholics at risk of alcohol addiction[21], the increased beta power is thought to be a marker of vulnerability rather than a trait or state variable (that's to say it may predate the development of alcoholism).

Beta activity has also been used as a predictor of relapse in alcohol addicts. It was found[5] that high-frequency beta activity can significantly distinguish between relapse-prone and abstinence-prone patients. Also, prediction [28] of relapse in chronic alcoholics using quantitative EEG (Q-EEG) was able to successfully classify 83-85% of patients, outperforming most previous efforts to predict relapse rates based on clinical assessments.

The results of some studies [5], and those reported by others, indicate that the value of rapid EEG power in predicting relapse can be generalised to all patients with a history of addiction to alcohol, cocaine, cocaine and alcohol or opioids. In summary, the EEGs of alcoholic patients clearly differ from those of both normal controls and patients with other psychiatric disorders, and the EEG can therefore be used for diagnostic reasons[32]. Therefore, EEG mapping can be used also as an objective method to predict relapse in chronic alcoholism[5].

2) Nicotine: A seminal work on smoking [33] reviewed previous research on the effects of smoking on the EEG. The immediate effect of smoking generates an "arousal" or "activation" EEG profile as smoking produces an increase in the beta band (14-30 Hz), an increase or decrease in the alpha band (8-13 Hz), a decrease in the delta (1-4 Hz) and theta (4-8 Hz) bands, and a passage to a higher dominant alpha frequency. EEG topographic representations show important regional spatial distributions and smoking induced changes in brain waves.

The administration of acute nicotine has been related to strong increases in brain activity from low frequencies (delta, theta, lower alpha) to high frequencies (alpha, higher beta), which reflects an excited state [10], [27]. Research has also revealed an increase in craving, a decrease in excitation and a deterioration in mood with a decrease in alpha frequencies during smoking abstinence [27].

Daily smokers had reduced resting delta and alpha EEG power and higher impulsiveness (Barratt Impulsiveness Scale) compared to nondaily smokers and non-smokers. Both daily and nondaily smokers discounted delayed rewards more steeply, reported lower conscientiousness (NEO-FFI), and reported greater disinhibition and experience seeking (Sensation Seeking Scale) than non-smokers. Nondaily smokers reported greater sensory hedonia than nonsmokers. [7].

It is very important to separate the different components of smoking, including psychological and pharmacological, by studying the EEG effects of fake and real smoking of a cigarette of the subject's preferred brand Nicotine-free cigarettes contain other components, such as tar and a very little quantity of nicotine. The inhalation of the fictitious cigarette concerns only the ambient air. EEG theta power was decreased when subjects smoked their cigarette in a telic (excitation avoiding) state. Beta 2 power was elevated when subjects smoked their cigarettes in a paratelic (excitation searching) state.

3) Cannabis: Cannabis is the most commonly consumed illicit recreational substance in the world, with an estimated annual prevalence of 3.8 per cent of the adult population having used cannabis in 2021 [34]. At present, cannabis use has been legally established in many countries, both as a recreational and medical drug [35].

Resting-state activity in cannabis users has been characterised as the inverted of typical frequency dynamics, because it reduced delta and augmented theta, beta and gamma

[25]. This neural oscillation tendency is generally observed in task-related EEG signals, indicating that cannabis users presented increased cortical activation, also during the resting state. Similar resting state patterns have been found in heroin users [14], alcohol users[5], [22] , and cocaine dependent users [9].

Smoking marihuana cigarettes [6] modify the strength and coherence of the alpha, theta and beta EEG bands. However, very few studies have analysed the influence of cannabis on the high frequency EEG bands, like gamma and high frequency oscillations (HFO) [8]. These oscillations have been related to a wide range of higher cerebral functions such as consciousness, working memory and perception [36].

The association between cannabis use and resting EEG is still ambiguous. Researchers discovered [6] that the theta resting state, with eyes closed, had a correlation with performance on a working memory task after acute cannabis intoxication, reflecting that the resting EEG can be related to cognitive performance. They also found effects in the dose-induced theta and beta bands, suggesting that these particular frequencies could be more sensitive to cannabis-related changes in cortical activity [6].

The relationship between resting EEG with eyes closed in cannabis and alcohol users [11] reported a positive correlation between delta power and cannabis dependence. Other researchers [26] have consistently found an increase in alpha and theta power and a decrease in delta and beta power at rest with eyes closed in long-term cannabis users and that cumulative exposure to marijuana over a very long period of time may be associated with slower cognitive processing; however, other studies have found a decrease in alpha and beta frequencies in posterior regions in abstinent cannabis users [16].

In addition, delta-9-tetrahydrocannabinol (THC) in the acute phase can disturb gamma oscillations via inhibitory inter-neurons [37]–[39], which suggests that cannabis users may have impaired gamma oscillations. Furthermore, a link between disruption of neuronal γ-band oscillations and cannabinoid-induced psychosis has been reported [8]. These results add to a large literature which proposes some overlapping of the acute effects of cannabinoids with the behavioural and psycho-physiological abnormalities identified in the psychotic diseases.

4) Cocaine: Cocaine's effects on human EEG [17] have been described as increasing beta-band activity. This has been repeated in more recent researches with larger sample sizes [4], [9], [19]. Excessive alpha activity and decreased delta activity [4], [7], [19] were observed, however others have found increased beta power[17] in cocaine addicts during resting conditions with eyes closed. Alterations in the EEG, predominantly in the anterior cortical regions, have been found to be associated with the quantity of cocaine previously consumed [19]. The EEG has been widely applied to characterise the effects of withdrawal in cocaine addicts. Many researches have shown that during prolonged cocaine abstinence, EEG effects are characterised by sustained increases in the alpha and beta bands and decreased activity in the delta and theta bands. [4], [19].

Recently, qEEG profiles [23] have been studied in cocaine addicts in reaction to acute self-administered smoked cocaine (50 mg) versus placebo. Theta, alpha and beta absolute potentials were elevated in prefrontal cortical areas for up to 25 minutes after cocaine use. Increased theta power was related to a positive subjective effect of the drug (high), and elevated alpha was correlated with nervousness. Cocaine also induced a Delta Coherence increase over the prefrontal cortex, which was related to nervousness. The placebo produced only a small alpha power elevation over the prefrontal cortex. These qEEG data reveal the implication of the prefrontal cortex in acute cocaine and suggest that the slow waves of qEEG activity, delta and theta, are implicated in the processes associated with experiencing the gratifying properties of cocaine [23].

The notion of linking baseline EEG activity to cocaine dependence in subjects in treatment programmes shows that cocaine addicts have a durable change in brain function as measured by qEEG, present at baseline assessment 5 to 14 days after the latest report of cocaine use, and persisting at one-month and six-month follow-up assessments [4], [19], [40]. A number of recent QEEG studies have shown an increased beta activity in the EEG to be related to relapse to cocaine abuse [5]. A reduction in the delta and theta bands of the EEG can be interpreted as a strong indicator of brain disturbances.

5) Heroine: A limited number of investigations have examined QEEG changes in heroin addicts. In more than 70% of heroin users, changes were seen at the beginning of the abstinence period (acute withdrawal), and they included low-voltage activity in the background with decreased alpha activity, increased beta rhythm, and a large quantity of delta and theta waves of low amplitude in the central regions [18]. Abstinent heroin addicts[14] have increased rapid beta power when compared to normal controls, and this result is consistent with several other EEG findings in cocaine and alcohol users [4], [17], [21]. Most studies have shown considerable, if not complete, normalisation of EEG spectral power in formerly dependent heroin users who have been abstinent for at least three months [9], [18].

Another study on heroin[18] found that heroin users who had been using heroin for at least 18 months showed frequency changes in the fast alpha band at frontal and central sites and a slowdown in the average frequency of slow alpha at central, temporal and posterior recording sites. Acute heroin withdrawal is typically characterised by marked desynchronisation, but as noted above, studies [9], [18] show that EEG spectral power tends to return to near-normal after several weeks of abstinence. The most consistent changes in the EEG of heroin users were observed in the alpha and beta frequencies. In early heroin withdrawal, there was a lack of alpha activity and overactivity in fast beta. This latter abnormality, which can be considered an acute withdrawal effect, appears to be significantly reversed when heroin use is discontinued for several months.

The increased power of beta, alpha and theta rhythms in people with a history of opiate use suggests potential deficits in cognitive function, according to a quantitative study of resting EEG changes after methadone treatment in opiate users[15]suggests that the increased power of beta, alpha and theta rhythms in people with a history of opiate use signals potential deficits in cognitive function. In fact, studies have shown that chronic opioid users are impaired in visual memory, perception, motor function and inhibitory control [41], [42]. However, it seems that these deficiencies are not so severe in patients undergoing methadone maintenance treatment, as their EEG measures are less impaired than those of healthy controls.

IV. Discussion

Electroencephalography (EEG) has revealed for the first time how addiction affects the brain and how it is diagnosed. For example, acute exposure to nicotine is associated with a large increase in scalp activity from lower (Delta, Theta, Low Alpha) to higher (Alpha, High Beta) frequencies, which is indicative of excitement. [10], [27]. Theta power was reduced when the participants in the telic state were smoking, whereas beta 2 power was increased when the participants in the paratelic state were smoking; this finding was only true for men. On the other hand, changes in the beta and theta frequency bands have been shown in EEG studies to be caused by doses of alcohol. In alcohol-dependent subjects, an increase in absolute power was detected at all scalp locations in Beta 1 (12.5-16 Hz) and Beta 2 (16.5-20 Hz), the increase was most pronounced in the central region. Beta 3 (20.5-28 Hz) power increased frontally in alcoholics. Male alcohol dependent subjects had clearly higher beta power in all three bands but Female alcoholics did not show a statistically significant increased. [22].

In the same sense, the augmentation of absolute theta power was also observed in all locations of the scalp in alcohol addict subjects; this increase in theta power was significantly greater in the central and parietal cortex in male alcoholics and in the parietal cortex in female alcoholics. Increased theta power in the EEG may represent an impairment of the central nervous system's information processing capacity in alcoholics [20]. Therefore the EEG of alcoholics differs significantly from those of normal controls, so the EEG can be used for diagnostic purposes. Therefore, the EEG of alcoholics is considerably different from that of normal controls, and the EEG can be used for diagnostic purposes. Furthermore, EEG may also have predictive utility because relapsers differ from abstainers in that they present with significantly more pronounced CNS hyperexcitability [24].

Cannabis users showed increased resting-state cortical activation, reflecting changes in the timing of neural oscillations that may be associated with cognitive impairment in cannabis use [25], with increased alpha and theta power and decreased delta and beta power in long-term cannabis users [26]. Therefore, the impact of THC on theta power and memory performance was correlated. [6].

In the same way, heroin induces: (a) a marked reorganisation of cerebral waves, with an augmentation in the proportion of fast beta and mainly in alpha frequency segments, (b) Prolonged temporal stabilisation for alpha and beta brainwaves and brief temporal stabilisation for polyrhythmic theta activity, and (c) dominance of the right hemisphere (greater presence of certain spectral characteristics in the EEG channels) than in the left hemisphere [12]. Similarly, there is some evidence that heroin addicts have an increase in relative beta 2 power and an increase in gamma coherence on intra-hemispheric and left hemisphere. In addition, coherence measures have shown significant correlations with clinical variables [14].

Increased absolute and relative alpha power have seen with quantitative EEG correlates of crack cocaine addiction [4]. Cocaine also markedly increased beta at the front and centre, and increased alpha at the front and temporal parts of the brain. Cocaine produced a similar cortical distribution of increases in EEG beta power to that induced by benzodiazepines and barbiturates. [17]. Cocaine's acute effects[23] produce rapid increases in absolute, alpha and beta-theta power over prefrontal cortical areas (FP1 and FP2) that persist for up to 25 minutes after exposure. Increased theta power was associated with feeling good effect and increased alpha power was associated with nervousness. There is a similar increase of delta coherence measured in the prefrontal lobe, which were a positive correlation with plasma cortisol and a negative correlation with nervousness. These findings provide evidence for the prefrontal cortex being implicated in the qEEG response to the acute administration of cocaine.

EEG measurements may also have a predictive and biomarker role. For example, in the prediction of alcohol and drug abuse relapse, is related to an increase in high frequency beta activity (19.5 to 39.8 Hz) with the coexistence of two pre-morbid factors, paternal alcoholism and the childhood conduct disorder. [5]. This prediction of alcohol relapse was found [28] and it was noted that, compared to abstainers, relapsers had more desynchronised EEGs in frontal regions, which was explained as a prefrontal cortex dysfunction. The elevation in EEG beta power can therefore be seen as a probable risk marker for the development of alcohol addiction and can be considered as an endo-phenotype predictor [21]. This suggests that subjects with a family history of alcohol abuse show alpha decreases and beta over-activity and have reduced relative and absolute alpha power in the posterior (01; 02) and frontal (F3; F4; Fz) and elevated relative beta in both areas when compared to controls who do not have a family history of alcohol dependence. [13]. EEG recordings are also useful for monitoring the evolution of the addictive state and in case of withdrawal or abstinence.

Grace Y. Wang (2015) [15] examined the electro-physiological activity related with methadone maintenance treatment (MMT) in opiate addicts and found that patients taking MMT or active opiate consumers showed a considerable increase in beta and theta band power compared to controls and that the abnormal electrical activity of the nervous system, which is present in people who are still illegal opiate users, can be reduced after MMT. This increase in beta power was considerably higher in the inpatient groups addicted to alcohol and cocaine and in the 1-6 months abstinent group [9].

A pronounced hyperactivity of alpha relative power and deficits in absolute and relative delta and theta power demonstrate the presence of anomalies in brain function in withdrawal cocaine dependent subjects (not dependent on any other substance), These changes tended to be higher in the anterior than in the posterior cortex, and inter-hemispheric relations were also disrupted [19]. The cumulative evolution of the effect of heroin on brain function shows that a significant desynchronisation is characteristic of acute heroin withdrawal, and there is an almost complete return to normal over several weeks of abstinence, with the mean alpha 1 frequency decreasing, most markedly in the central, temporal and occipital lobe, and frequency changes in the alpha 2 frequency, most pronounced in the frontal and central lobe [18]. This slowing of alpha frequency is similar in smoking cessation [27]

V. CONCLUSION

Although the literature on EEG in addictions is highly heterogeneous, some authors agree that it is a relevant technique for discerning the phenomenology of addictions. EEG is sensitive to how addiction affects the brain and has shown changes in brain electrical activity during addiction. However, the clinical value of EEG recording in addictions is not yet clearly established. However, several studies argue that this non-invasive technique has an undeniable contribution to the understanding, prediction, diagnosis and monitoring of addictions. But future EEG studies are needed to identify other robust brain-electrophysiological specificities in addiction. The collection of more comparative data between EEG findings and clinical changes in addicts could also be useful and be improved by methodological insights.

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