Dissociated States in Fibromyalgia

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Abstract

Fibromyalgia (FMS) is a chronic syndrome of widespread pain and fatigue; its aetiology is still a matter of debate. Some authors suggest that it is a psychosomatic response to psychological distress, but there are other organic factors and symptoms that are still under research. To our knowledge, this disorder is explained by the Dissociated States (DS) concept. An example of that is the alpha-delta sleep pattern. In FMS, these two features coexist reflecting a “sleeping-awake” state. Considering that FMS patients may suffer from “dissociation”, it is important to characterize their awake and sleep brain microstates.

Our goal is to identify sleep DS, both in NREM and REM as well as to provide data for sleep electroencephalogram (EEG) mapping abnormalities both in specific sleep stages and across sleep stages. This will be achieved by computing sleep EEG mapping in the conventional frequency of bands (δ, θ, α₁, α₂, σ and β).

1. Introduction

Fibromyalgia (FMS) is a disorder of unknown aetiology which occurs in 3-4% of the general population, 80-90% are females (ACR). FMS patients suffer from severe fatigue, sleep disturbances, morning stiffness, cognitive problems, affective disturbances, multiorgan dysfunctions and other symptoms (anxiety, depression, sensory hypersensitivity) [1,2].

Discussion also concerns its aetiology. Some authors argue that it is a psychosomatic or a psychiatric disorder related to major depression [3]. In our experience, the polysomnography (PSG) features of FMS differ strongly from those of major depression. Alternatively, FMS is a psycho-neuro-endocrine-immune disorder, so it has an organic basis [4,5]. However the biological markers are still not found.

Considering this, we hypothesize as a basic general mechanism, the concept of DS, which can take several forms. A clear example of that is the dissociation of wake/sleep states, the alpha-delta sleep pattern.

Whenever dissociation is present, there are consequences upon timing or switching errors in the normal process of the dynamic reorganization of the brain, as it moves from one state to another. Consequences can be dramatic, if elements of one state persist, or are recruited erroneously, into another state. The construct of dissociation, characterized by disruptions of the usual functions of memory, consciousness, identity, or perception [6] provides therefore a useful theoretical framework for understanding medically unexplained symptoms in FMS. Clinical assessment of FMS brings to light the inconsistency between the severity of complaints and the lack of explanatory biological markers, which in our opinion can be explained by a central nervous system (CNS) dysfunction, via DS, and must be investigated.

The presence of alpha rhythm pattern is typical from awake, disappearing at sleep onset, while delta EEG patterns are characteristic of sleep. In FMS these two features coexist reflecting a “sleeping-awake” state (see Fig. 1).

Figure 1 – Alpha-Delta pattern

Our recent data [7], demonstrate that among the higher electroencephalogram (EEG) frequencies, sleep spindles frequency appears as a not yet described or “hidden” component of the brain activity. As the thalamus is the main spindle generator and the main filter of higher frequencies to the cortex, we hypothesize on a thalamo-cortical dysfunction...
in FMS and can constitute specific markers of an abnormal pain processing in FMS, expressed, at least during the sleep. Sleep is a local and use dependent phenomenon subserving synaptic homeostasis. Many stress disorders have a dysfunction or suppression of the hypothalamus, which controls sleep, hormonal function, temperature regulation, and the autonomic nervous system. Even more, FMS has an enhancement of cardiovascular sympathetic activity while recumbent but during the tilt test, a lack of increased sympathetic discharge and decreased cardiac vagal activity is observed [8].

In our experience (unpublished results) dissociation is present not only in NREM but also in REM sleep. Recent data suggest that these patients have specific topographic changes and abnormal patterns and hidden abnormalities in the sigma band, suggesting a cortical dysfunction [7].

To our knowledge, despite brain mapping studies in FMS have been rarely performed, they are very useful to determine brain connectivity changes in FMS and therefore to evaluate the hypothesized dissociations in this syndrome. Our main goal is to identify the presence of dissociated states during sleep (both in NREM and in REM). Using conventional mapping techniques, we will then try to provide data for Sleep EEG mapping abnormalities both in specific sleep stages and across sleep stages and try to identify which possible structures are involved in these brain mechanisms.

2. Methods

Twelve women, aged 30 to 58 years, with FMS and 12 healthy controls were recruited to perform a nocturnal polysomnography (PSG) at our sleep laboratory, using a 19 EEG channels system plus conventional PSG data. The sleep studies comply with the recommendations of the ESRS (2007). The patients have then completed several questionnaires including the FIQ and others about sleep quality. All FMS subjects have at least 11 tender points (TP) from the 18 eligible.

PSG will then be analyzed by us and the DS will be characterized. The dissociations that we will assume as relevant are: the alpha-delta sleep; the presence of rapid eye movements of saw tooth like activity in stage 2NREM; the presence of alpha and spindle like activity in REM; the presence of slow eye movements in stage 2NREM (typical of stage 1NREM).

Sleep parameters collected will be introduced in tables for statistical analysis. Through spectral analysis (FFT), power maps will then be computed for all EEG frequency bands at all the channels, at specific times. The mapping techniques used will be the standard ones, based on frequency components and spectral analysis.

3. Results

After the data analysis we expect to find dissociated states both during REM and NREM which, by a general view of dissociation, will contribute to a final model of dysfunction. We expect to identify differences between the topographic brain mapping concerning FMS and normal controls. These differences should be more marked in the alpha and spindle range during sleep.

Our preliminary analysis seems to show that the evolution of the EEG activities in FMS reveals a consistent reduction of delta during 2NREM and a persistent dominance of more rapid frequencies, particularly α1, α2 and sleep spindles. EEG Mapping across sleep cycles looks like there is a reduction of delta activity and an increase in alpha activity throughout the night, which are in favour of a deficient homeostatic process for sleep. Furthermore abnormal topography of the alpha band, spreading towards the frontal regions in 2NREM and in REM, may explain the subjective symptom of being both asleep and awake.

4. Conclusions

The presence of DS seems to be significantly higher in FMS and almost inexistent in controls. These data may reveal that the difficult boundaries between stages are in line with other symptoms of dissociation, namely in what concerns pain and disease severity evaluation.

References


