Causal Relationship Analysis of Heart Rate Variability and Band Power Time Series of Electroencephalographic Signals

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Abstract

This study aimed to find whether there is a causal relationship between band power time series ($\boldsymbol{BP}_{ts}$) extracted from EEG and heart rate variability (HRV). Such relationships were explored during spontaneous and a controlled breathing tasks. Data analyzed were recordings obtained from 14 healthy subjects using one ECG lead and 21 EEG channels. The RR intervals from the ECG were used to obtain the HRV signal, which was decomposed with Empirical Mode Decomposition into components of different spectral content known as intrinsic mode functions (IMFs). Granger causality tests were run for the $\boldsymbol{BP}_{ts}$ of alpha, beta and gamma frequency ranges of the EEG signal and the HRV signals IMFs. G-causality increased for three different conditions: slower IMFs (IMF4), $\boldsymbol{BP}_{ts}$ of higher frequency (gamma) band and during task realization. Meaning, gamma's $\boldsymbol{BP}_{ts}$ G-caused HRV for a larger number of subjects and channels. Also there was a larger incidence on the number of channels that G-caused HRV during the controlled breathing task. The causal influence from the $\boldsymbol{BP}_{ts}$ of EEG signals to the HRV IMFs suggests there is an indirect or unobserved interaction between instantaneous changes on EEG band power and components of HRV which may explain changes in its dynamics.

1. Introduction

The central autonomic network (CAN), is an interconnected ensemble of structures that controls autonomic functions like heart rate, respiratory efforts, hormone secretion among others, aiming to preserve homeostasis [1, 2]. CAN structures are located on different levels of autonomous nervous system: insular and medial prefrontal cortices, amygdala, hypothalamus, periaqueductal gray matter, locus coeruleus, parabrachial nuclei, reticular formation, dorsal vagal, ambiguus, tractus solitarius and Raphe nuclei, and Rostral ventrolateral medulla [1, 3]. CAN has control over outflows of sympathetic and parasympathetic branches influencing heart rate variability (HRV). Observing HRV is therefore a useful tool to explore autonomic nervous system (ANS), nevertheless a full comprehension of the phenomenon has not been achieved [2, 4]. Yet, it is commonly accepted that spectral analysis allows to study effects of the two branches: *sympathetic*, on low-frequency band (**LF**, [0.04 – 0.15] Hz) and *parasympathetic*, on high-frequency band (**HF**, [0.15 – 0.4] Hz). Affectations on cerebral cortex have secondary effects on cardiovascular systems, for example, stroke could produce cardiac arrhytmias and even sudden death if affected region is neighboring CAN structures (insular or prefrontal cortices) [3]. Autonomic system disorders could have manifestation on HRV indices like tachycardia or fixed heart rate, producing cardiac autonomic neuropathy. Polisomnographic studies have shown an interaction between indices derived from EEG signal and HRV, Jurysta et al. [5] found a relationship between vagal activity and delta band of EEG. Kuo et al. [6], showed that beta power is a descriptor of autonomic activation during NREM sleep. Some authors have suggested analysis of EEG and HRV signals could be useful to explore CAN without an invasive intervention. Schieke et al. [7] and Piper et al. [8], analyzed data derived from children with temporal lobe epilepsy, both suggest an interaction between epileptogenic networks and CAN producing arrhythmias prior to seizure episode. Liou et al. have worked with autonomic dysfunction: Parkinson's Disease [9] and uremic patients [10]. Both works suggest a relationship between EEG's band power and **HF** and **LF** components of HRV, despite significant correlations were found they are below 0.5. In a previous work of our group, de la Cruz-Armienta et al. [11] demonstrated that for healthy subjects a Granger causality relationship exists from HRV to $\mathbf{BP}_{ts}$, with a channel distribution depending on respiratory tasks. On this work, a new set of experiments were realized, analysis includes separation of HRV components and extending analysis over gamma band, from $\mathbf{BP}_{ts}$ to HRV. The obtained results confirm those of previous work, suggesting cortical CAN centers have larger than recognized relationship with sympathetic activity.

 ISSN: 2325-887X DOI: 10.22489/CinC.2019.131

2. Materials and Methods

The experimental paradigm for this study was the same as the one used by de la Cruz-Armienta on 2017 [11].

Data recording implied 2 stages:

1. Idle state: the subject was awake, sat down with eyes closed, listening to a mild story (*The Origin of Evil* by Fiodor Dostoyevski) during 8 minutes.

2. Controlled breathing task: the subject was awake, sat down with eyes closed, listening to a guiding sound that indicated the duration and rhythm of each inspiration and expiration he/she must take, during 8 minutes.

Data analyzed were extracted from a database of 14 healthy subjects (age 21±2 years, 7 females); according to inclusion criteria, subjects were non-smokers, did not take any alcoholic beverages nor medication for at least 24 hours prior the study, did not practice any cardiovascular exercise, got at least 6 hours of sleep the night before the study and had normal BMI.

21 EEG channels and one ECG lead were recorded using Neuroscan 4.5 amplifier at a sampling rate of 1000 sps. From the international 10-20 system, the following were used: Fp1, Fp2, F3, Fz, F4, FC3, FCz, FC4, C3, Cz, C4, CP3, CPz, CP4, T5, P3, Pz, P4, T6, O1 and O2. The ECG lead was obtained by placing the precordial electrodes V_1 and V_2. Fpz corresponded to ground for both EEG and ECG and the common references were A1 and A2.

The ECG signal was extracted by subtracting the signal recorded by V_2 from the V_1 signal. Joachim Behar's QRS detector based on the P&T method was used to obtain the indexes of the RR peaks [12]. The HRV signal was then interpolated at a sampling frequency of 10 sps using a cubic spline. Lastly, the HRV signal was decomposed into IMFs using the Empirical Mode Decomposition method, a typical register during breathing control task is shown on Figure 1.

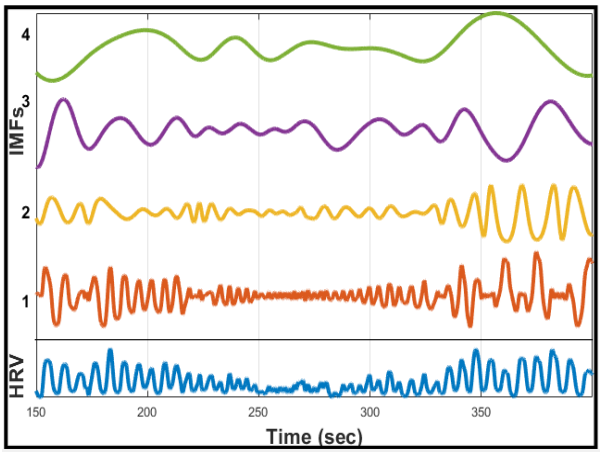

Figure 1. An example of HRV signal and HRV IMFs, scaled for better appreciation

The band power time series ($\mathbf{BP}_{ts}$) were estimated for alpha ([8-12]Hz), beta ([12-30]Hz) and gamma ([30-100]Hz) bands with a sliding window of 2 seconds and a sliding step of 0.1 seconds using Welch periodogram method achieving a 10 sps frequency synced with HRV. The $\mathbf{BP}_{ts}$ estimation consists on obtaining the signal's frequency spectrum and calculating the relative power for each frequency band, therefor the $\mathbf{BP}_{ts}$ show the distribution of power according to the components composing the signal. Figure 2 shows the $\mathbf{BP}_{ts}$ obtained for alpha, beta and gamma frequency ranges from a register during controlled breathing task.

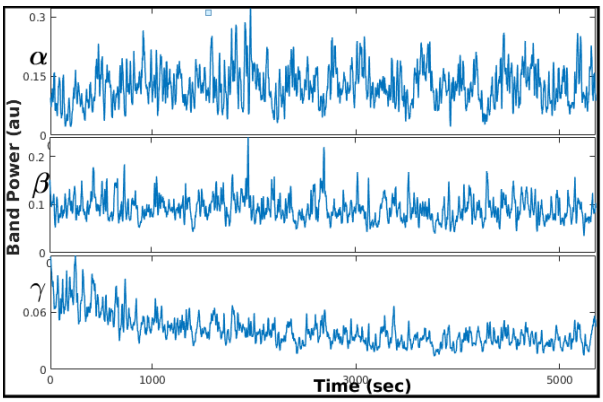

Figure 2. An example of $\mathbf{BP}_{ts}$ of the EEG

Finally, a Granger causality test was run from the $\mathbf{BP}_{ts}$ of each EEG frequency range to the HRV IMFs, for each of the stages recorded. A causal correlation found between two signals does not imply a physical connection, but detects causality relationship from the first signal to the second. This method is based on a linear reggresion model of stochastic processes and was obtained using Seth's Matlab toolbox [13, 14].

3. Results and Discussion

A G-casual relation was found between the $\mathbf{BP}_{ts}$ of alpha, beta and gamma waves and the HRV IMFs; in contrast with Schieke and Piper works', who found a larger correlation with delta band. Tests with surrogate data from HRV were run achieving no G-causal relation, which suggests a real inference from $\mathbf{BP}_{ts}$ to HRV sympathetic components. As seen on Figure 3, G-causality increased significantly for slower IMFs (IMF4) as well as for higher EEG frequency (gamma) bands. Row A on the figure corresponds to idle state and row B to the breathing control task, showing there was a larger incidence on the number of channels that G-caused HRV during the controlled breathing task.

Table 1 shows the proportion of subjects that had a G-causal relation from each frequency band $\mathbf{BP}_{ts}$ to the different HRV IMFs, for the channel(s) with the highest in-

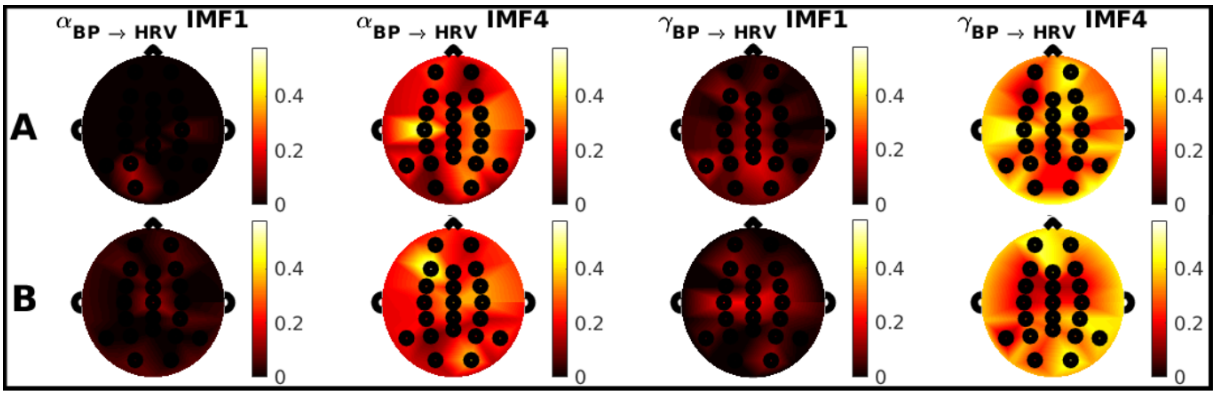

Figure 3. G-causality from each channel of alpha's and gamma's $\mathbf{BP}_{ts}$ to HRV IMFs 1 and 4. Row A corresponds to idle state and row B to the breathing control task.

Table 1. Channel(s) where the highest number of subjects had G-causality from EEG $\mathbf{BP}_{ts}$ to HRV IMFs. The first set of values corresponds to the results during idle state whereas the sets in bold correspond to the controlled breathing task.

	IMF1	IMF2	IMF3	IMF4
α	P3,O1 (2/14)	F3,T5,P4,O1 (3/14)	Fp2,Fcz,Cz,O2 (6/14)	C3 (8/14)
	Cz (2/14)	**F3,Fc3,Fc4 (5/14)**	**Fp1,T5 (6/14)**	**F3 (8/14)**
β	O2 (2/14)	C3 (6/14)	O1 (8/14)	Fc3 (9/14)
	Fp2,F4,Fc4,C3,C4 (1/14)	**Fp1 (7/14)**	**Fp1 (8/14)**	**Cz (8/14)**
γ	T5,Pz (3/14)	Fc4 (6/14)	F4 (6/14)	Fp2,C3 (7/14)
	C3,Cz (3/14)	**Fp1 (7/14)**	**Fp1 (7/14)**	**Fp1,O2 (7/14)**

cidence. The first set of locations and values in each row corresponds to the results during idle state whereas the sets in bold correspond to the breathing control task results.

For most cases of the controlled breathing task, Fp1 was the channel with the largest number of subjects for which G-causality was stated; this may show the $\mathbf{BP}_{ts}$ of the waves (most pertaining to gamma's frequency range) found near the left hemisphere's portion of the prefrontal cortex, influence HRV dynamics. This correlates with the distribution of CAN centers at the cortex as well as with the fact that all of the channels with the highest number of G-causalities found for the controlled breathing task were located on the anterior cortex. During idle state, the locations of the highest incident channels were more scattered than those on the controlled breathing task and were possibly not as exacerbated due to a lower activity on those centers. Nevertheless, for both conditions G-causality was found on several channels located on the right hemisphere of the cortex, this lateralization phenomenon agrees with findings of Piper, where patients with right temporal lobe epilepsy present major alterations on HRV dynamics.

Table 1 shows how, for every condition and frequency range, the channels influencing IMF4 had the highest incidence of subjects with a true G-causality. As mentioned before, IMF4 corresponds to the slowest frequency mode from the HRV decomposition; lower frequencies are associated with sympathetic nervous system activity, meaning changes on EEG power due to the sympathetic activity of the ANS alter the low frequency HRV components.

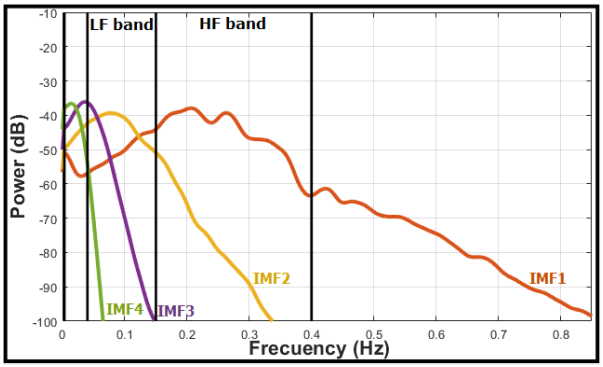

Figure 4. HRV IMFs' mean relative power during idle state

Figures 4 and 5 show the population mean HRV spectral components' relative power for **LF** and **HF** ranges according to their IMF. During idle state IMFs were clearly separated, whereas for the controlled breathing task, IMFs overlapped mainly at **LF** and were not clearly divided onto the frequency ranges defined. Table 1 shows how, during

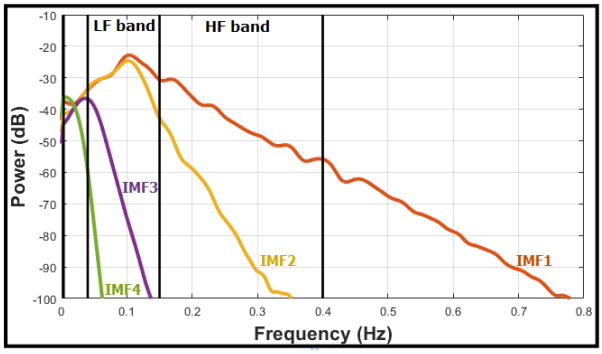

Figure 5. HRV IMFs' mean relative power during breathing control task

idle state, faster IMFs (IMF1) were G-caused by channels located on the posterior part of the cortex, while the slower IMFs (IMF4) were caused by channels found on the anterior cortex. Channels that G-caused IMFs during the controlled breathing task did not show this changes in distribution and, as mentioned before, were all found on the frontal lobe, this leads to believe there is relation between activity on the anterior cortex and low frequency or sympathetic activity in the heart. Also, these may help explain the dynamics of the CAN activity as well as its regulation of the parasympathetic and sympathetic branches while adapting heart rate to changes in breathing.

4. Conclusion

There is a causal influence from the $\mathbf{BP}_{ts}$ of EEG signals to the HRV IMFs that leads to believe there is an indirect or unobserved interaction between instantaneous changes on EEG band power and components of HRV. Knowing this interaction might be useful for a better understanding of HRV analysis in different applications, since it may help explain or predict changes in its behavior. This tool could be used to explore relationships in different contexts such as hemodyalisis, diabetes, Parkinsons or other systemic diseases that have affectations on nervous systems and homeostatic condition.

Acknowledgments

Authors gratefully acknowledge to Research Office of Universidad Iberoamericana CDMX, for financial support for this work.

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