Causality in the Cardio-Postural Interactions during Quiet Stance

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Abstract

Cardiovascular and postural systems are understood to interact with each other to maintain equilibrium. Prolonged standing is known to induce postural sway, pre-syncope symptoms and muscle fatigue. Prior work has shown the presence of interactions between the cardiovascular and postural systems during quiet stance. The cause-and-effect relation between the representative signals of the two systems remains to be established. This preliminary work presents a study to identify the causal relationship between the blood pressure waveform (BP) and resultant center of pressure (COPr) as well as between systolic arterial pressure (SAP) and COPr signals during quiet stance. A 5 minute sit-to-stand experiment was conducted for 7 healthy young participants to acquire data for the SAP and COP signals.

The statistical framework of Granger causality was applied to test for the bidirectional causal relation between the BP-COPr and SAP-COPr signal pairs. The algorithm computed the F-statistic and critical value from an F distribution for null hypothesis of no causality between the signals. Data were extracted for a window of 15 seconds length, translated with an overlap of 5 seconds over the last 4 minutes of the quiet stance phase to obtain 22 to 23 time windows.

Data for subjects rejected the null hypothesis for both BP-COPr and SAP-COPr signal pairs. The results from this study suggest that during quiet stance bidirectional interaction exist between BP and COPr as well as between SAP and COPr.

1. Introduction

It is known that the cardiovascular and postural systems interact together to maintain homeostasis and stability [1]. This interaction between blood pressure, centre of pressure and electromyogram have been studied using classical as well as wavelet based coherence analysis [1, 2]. A strong coherence using a wavelet based approach was reported between medio-lateral postural sway and blood pressure [2] and SAP and EMG [3]. Although coherence represents the degree of linear coupling between two time series data, it does not indicate the direction in which the information flow occurs [4]. Finding an answer to the question of directionality of information flow is critical, as, there have been cases with reported fainting and falling in elderly, astronauts and patients with neurodegenerative diseases [1].

The existing knowledge of bi-directional causal interaction can be used in predicting functionality of physiological system. For example in case of heart rate (HR) and SAP, it is well established that both HR and SAP interact in a closed loop (bidirectional interaction) [5, 6] where the dominant reflex is in the direction of SAP changing HR via the arterial baroreflex. In a physiological sense, SAP is the variable in need of regulation (homeostasis) and HR is one of the mechanisms through which SAP can be changed. A similar closed loop interaction is yet to be established between blood pressure and postural sway.

Granger causality is a statistical technique proposed by C.W.J. Granger in 1969 for studying causality between two time series data [7]. In literature Granger causality has been applied extensively in the fields of economics, neuroscience and climate science for studying the causality between two time series data. Motivated by above, we aim to find the cause-and-effect relationship between the cardio-postural time series data during orthostatic challenge under quiet standing.

2. Materials and Methods

2.1. Experimental Data Acquisition

Data were acquired from 7 young individuals (Age 24.7±2.2 years, Weight 62.1±6.9 Kg, Height 1.71±0.4 m) during a sit to stand test. The study was approved as minimal risk by the research ethics board of the Simon Fraser University. Written and informed consent was obtained from each participant prior to any experimentation. Participants with documented or reported cardiovascular disease or postural complications were excluded from the study. All participants refrained from exercise and caffeine 24 hours prior to the experiment. The experiments were performed in a sensory input reduced

environment within an enclosed space. The sit-to-stand test required participants to be seated for 5 minutes, passively (assisted) transfer to standing, and maintain a quiet stance for the next 5 minutes. The 10 minute procedure was performed with eyes closed with an imaginary eye level gaze. This choice of removal of visual input was motivated by the observation where, increased postural sway, elevated muscle activation and change in blood pressure were recorded with eyes closed in comparison to eyes open during the experiment [1].

During the 10-minute period, continuous blood pressure measurement was acquired from finger photoplethysmography (Finapres, Ohmeda USA) from which beat-to-beat systolic arterial pressure (SAP) was obtained. The postural sway data in terms of vertical projected coordinates (medio-lateral: COPx; and anterior-posterior: COPy) of the center of gravity were obtained from the force and moment data acquired with a force platform (Accusway Plus, AMTI USA. Resultant centre of pressure (COPr) was obtained from COPx and COPy as $\mathrm{COP}_r = \sqrt{{COP_x}^2 + {COP_y}^2}$. All signals were sampled at sampling rate of 1000 Hz. Of the 10 minutes of data acquired during a sit to stand test, we used last four minutes of quite standing for processing.

2.2. Granger Causality

For two time series variables say, X and Y, the variable Y is said to Granger cause X if the error in predicting the future of X can be reduced by using the past information of Y along with that of X [4, 8]. The application of Granger causality method is limited to time series data that are wide sense stationary, meaning, that the mean and variance of these data do not change over the period of analysis [8]. Mathematically, Granger causality can be explained in terms of restricted and unrestricted models. A restricted model of X uses only past information of X to predict the future of X, whereas unrestricted model of X incorporates past information of another variable Y along with that of X to predict the future of X [8]. A restricted model of wide sense stationary time series X is given as;

$$X_t = \sum_{l=1}^{\infty} a_{1l} X_{t-l} + w_{1t} \qquad (1)$$

Where, w_1 is the error in predicting the future of X using past information of X. Now, the unrestricted model of X is given as;

$$X_t = \sum_{l=1}^{\infty} a_{2l} X_{t-l} + \sum_{l=1}^{\infty} b_{2l} Y_{t-l} + w_{2t} \qquad (2)$$

Where w_2 is an error in predicting the future of X using past of both X and Y. If w_2 is significantly less than w_1 then according to the definition of causality by Granger, Y is considered to have a causal influence on X [4, 8]. In this research the Granger causality test was conducted using an F statistics. In this method, the value of F statistics is first computed and then followed by critical value from an F distribution for a user defined significance level. The null hypothesis of no causality between the two time series is rejected for values of F greater than the critical value. The F statistics is computed as;

$$F = \frac{\frac{RSSr - RSSur}{k}}{\frac{RSSur}{L - 2k - 1}} \qquad (3)$$

Where, RSSr and RSSur are residual sum of squares of restricted and unrestricted models respectively, k is model order and L is the total number of observations used in estimating unrestricted model. The residual sum of squares for restricted and unrestricted models can be computed as discussed in [8]. The F statistic computed above has an F distribution, the degree of freedom for numerator and denominator being k and L-2k-1 respectively. The critical value of an F was determined using an F inverse cumulative distribution function which requires a knowledge of significance level of a test, degree of freedom in numerator and denominator. The model used for estimating Granger causality is very sensitive to model order, choosing a model order too low can cause a poor representation of the data while choosing a model order too high can cause problems in estimating the model [8]. Typically, model specification is chosen based on Akaike information or Bayesian information criteria (BIC) [9, 10]. BIC was used in this research.

2.3. Data Processing

Data acquired during the five minutes of quiet standing was band pass filtered between DC (0.001 Hz) and 20 Hz frequency cutoff and resampled to 100 Hz. Next, last four minutes of data was processed to eliminate any motion artefacts incorporated into signal during the transition from sit to stand. A window of 15 seconds was chosen for estimating the Granger causality between a pair of physiological signals. The window was then translated over the 4-minute period with 5 seconds of overlap, this step resulted in the Granger causality being computed over multiple time windows throughout the final 4 minutes of standing. The small window size ensured that the segments of signals under analysis were in alignment with the stationarity assumption of the Granger causality method; small window size helps resolving non-stationarity issue [8]. Granger causality test was conducted at a significance level of 0.05 and number of lag was chosen to be 2 for all pairs and all subjects. Processing to test causality is

explained in Figure 1.

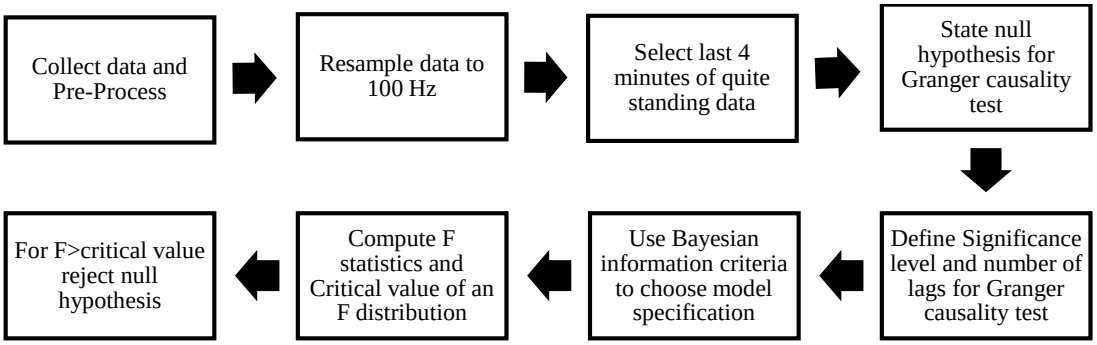

Figure 1. Step by step processing to conduct a Granger causality test between cardiovascular and postural dataset.

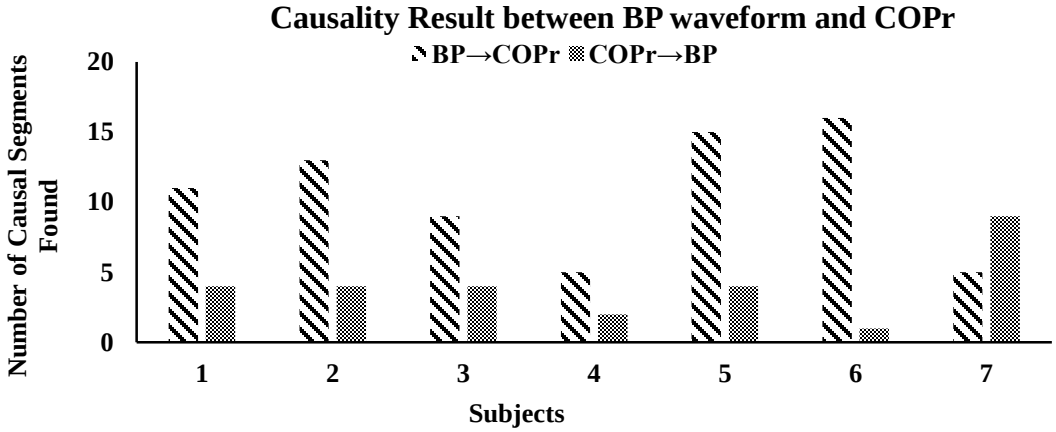

Figure 2. Causality results computed between BP and COPr signal pair for 7 subjects; showing the existence of bidirectional interaction between BP and COPr signal pair.

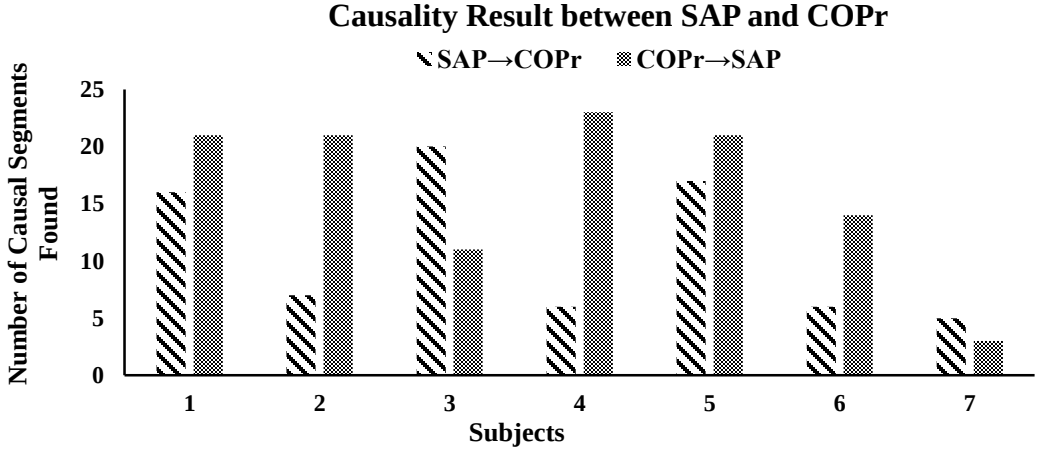

Figure 3. Causality results computed between SAP and COPr signal pair for 7 subjects; showing the existence of bidirectional interaction between SAP and COPr signal pair.

3. Results

The number of time-window-segments having a causal relationship between various signal pairs were obtained through the implementation of the steps shown in Figure 1.

Bidirectional causal interactions between the BP and COPr as well as between SAP and COPr signal pair was observed. For causality analysis between BP and COPr signal pair, BP→COPr (11±4 windows) was dominant compared to reverse causality of COPr→BP (4±3 windows). For 7 subjects total of 74 causal segments were found for BP→COPr compared to 24 for COPr→BP (Figure 2).

Causality analysis between SAP and COPr signal (Figure 3) for all subjects resulted into a total of 77 causal segments for SAP→COPr (11±6 windows) while a total of 114 causal segments were found for COPr→SAP (16±7 windows).

4. Discussion

In this research we have applied the Granger causality method in an attempt to establish a possible cause-and-effect relationship between the cardiovascular and postural systems during the orthostatic challenge of quiet standing. In general, the data show a bi-directional causal relationship between BP and postural sway; however, similar to baroreflex, blood pressure appears to be the regulated variable, as, on average the directional causality of BP→COPr was thrice as prevalent as COPr→BP. Causal segments found for BP→COPr were consistently higher in number compared to reverse causality for all subjects except for subject 7.

When causality of COPr was studied with SAP (upper value of BP peak) on an average COPr→SAP was higher than reverse causality of SAP→COPr. Causal segments found were higher in number COPr→SAP for all except for subject 3 and 7. This may suggest that postural sway initiates alterations in the SAP to maintain equilibrium.

The results from this preliminary research highlight the presence of a bidirectional causal interaction between the cardiovascular and the postural system under orthostatic challenge induced by quiet standing (Figure 2 and 3). These results further suggest a compensatory interaction between the systems to maintain homeostasis. Such information is critical as an impairment in one system may cause disruption in functioning of the other, leading to injury due to falls or fainting. Clinical application of our work would need thorough analysis of other representative signals from the cardiovascular and postural systems. Additionally, analysis of data from bigger cohorts would be required to gain statistical power and obtain population wide inferences.

5. Conclusion

Our work presents a study where we performed a Granger causality test to investigate the cause-and-effect relation between the cardiovascular and postural system during orthostatic challenge. Based on the results obtained from 7 subjects, we demonstrate the existence of a bi-directional Granger causal relationship between cardiovascular and postural system in which BP predominantly drives changes in postural sway, whereas postural sway drives changes in SAP. This further indicates a blood pressure control reflex closed loop, a possible postural muscle pump baroreflex, to maintain homeostasis.

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