Effects of Exercise on SCG Signals in Healthy Subjects

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Heart diseases are a leading cause of mortality globally with cardiovascular disease (CVD) accounting for around 17.9 million deaths as of 2019 [1]. Limited access to healthcare services in low- and middle-income countries may be a main reason of the high mortality. The financial burden associated with CVD is also high and may reach $70 billion in USA by 2030 [2]. Early detection of heart diseases can reduce adverse health events and lower related costs. Regular monitoring of these conditions can alert patients and healthcare providers about life-threatening abnormalities, which can reduce hospitalization rates. Use of a reliable, simple-to-use and cost-effective non-invasive techniques to detect heart conditions can expedite the diagnosis and treatment processes, improving patient management and quality of life.

Seismocardiography (SCG) is a non-invasive method that measures the vibration of the chest wall surface induced by cardiac events such as valve closure, myocardial contraction, blood flow turbulence, and flow momentum changes [3,4]. SCG is measured by accelerometers which are placed at the chest surface. Several studies extracted features from SCG that were found to correlate with heart diseases and heart failure readmission [3,4]. Hence, it has the potential to be utilized in a clinical setting as a non-invasive diagnostic and monitoring tool for patients with heart diseases. But the variability of SCG signal may limit its clinical utility. The aim of this study is to better understand the respiration induced variability of SCG signals and to investigate the effect of exercise on SCG variabilities.

SCG signals were acquired from 5 healthy subjects, aged 21-29 years old (4 male, 1 female). A triaxial accelerometer placed on the 4th intercostal space (ICS) at the left lower sternal border (LSB) was used to acquire the SCG signal. The accelerometer was attached to the chest surface using double-sided medical-grade tape. A signal conditioner (Model: 482C, PCB Piezotronics, Depew NY) was used to amplify the signals from the accelerometer. Amplified signal was acquired on a computer using a data acquisition system (IX-TA-220, iWorx Systems Inc., Dover, NH 03820, USA) and associated software at 10 kHz sampling frequency. Electrocardiogram (ECG) and galvanic skin response (GSR) signals were also acquired simultaneously using a recording module (IX-B3G Biopotential & GSR Recorder). In a previous study, GSR signal was found to best match the lung volume signal when one electrode was attached in the mid clavicular line just below the right clavicle and another on the middle of the lower left abdominal quadrant [5]. The experiment setup is shown in Figure 1.

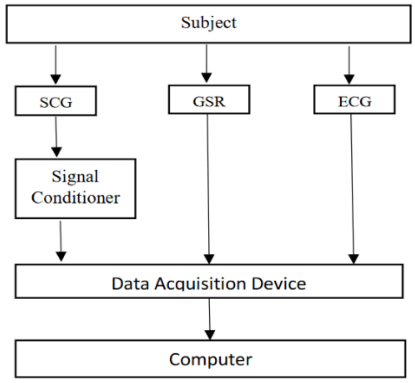

Figure 1. Experiment Setup

The protocol consisted of the following steps (also shown in Figure 2): (a) Subjects sat on a chair and waited for 10 minutes before data acquisition. This helps to reach a stable resting heart rate for subjects. Sensors were placed on subjects (accelerometer on 4th ICS at LSB) and data was acquired for 3 minutes at rest [Figure 2(i)]. (b) Subjects exercised by cycling on a stationary bike until the target heart rate for moderate exercise level achieved (Equation 1) [Figure 2(ii)]. Typically, it took 10-15 minutes for the subjects to reach to their respective target heart rate. The equation of the target heart rate followed a previous study [6].

$$Target\ heart\ rate = 220 - subject's\ age * 0.7 \quad (1)$$

(c) After achieving the target heart rate, subjects rested again on the chair and data was acquired for another 3 minutes at the sitting position [Figure 2(iii)].

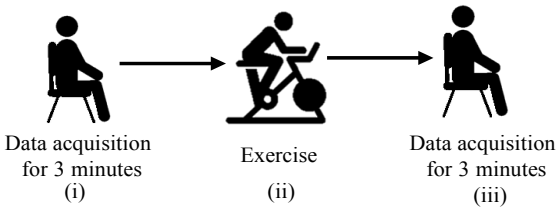

Figure 2. Schematic representation of study protocol

A commercial software package (Matlab, Mathworks, Natick, MA) was used to perform data analysis. To demonstrate the effect of exercise on the SCG signals, 1st minute of pre- and post-exercise recordings were considered for analysis. The analysis in this study was limited to the dorsal-ventral direction of the SCG signals. Raw SCG and ECG signals were filtered to remove environmental and electronic noises. GSR signals were filtered using a bandpass filter with passband of 0.1-8 Hz. Signals were then downsampled to 1 kHz for faster analysis. ECG R-waves were detected using Pan-Tompkin algorithm [7]. ECG and SCG signals were segmented into cardiac cycles (also called beats) which started 0.1 seconds before the R peak of the corresponding ECG signal and ended at 0.1 seconds before the next R peak. All the SCG and ECG beats were grouped into 4 respiratory phases. The respiratory phases were defined using GSR signal (representing lung volume) and its derivative, which is equivalent to airflow rate [Figure 3].

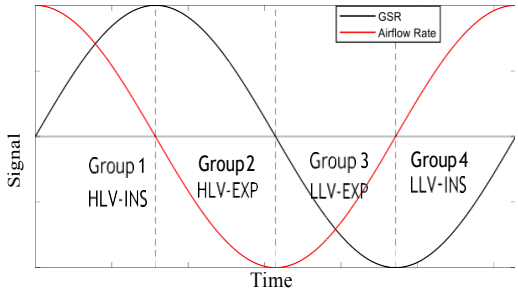

Figure 3. Representation of the 4 respiratory phases based on GSR and airflow rate signal. SCG and ECG events were categorized into the 4 shown groups where HLV: High lung volume, LLV: Low lung volume, INS: Inspiration, EXP: Expiration.

After grouping ECG and SCG beats in the 4 groups shown in Figure 4, the R-peak of each ECG beat was detected using the Matlab function 'findpeaks'. A window starting at 100 ms and ending at 150 ms was set for each SCG beat to capture the SCG1 peak as the maximum within that window using the same 'findpeaks' function which marked the SCG1 event. The time interval between R-wave and the corresponding SCG1 peak approximates the pre-ejection period (PEP) and was calculated for each beat. Figure 4 shows R-SCG1 time interval for the four respiratory phases (i.e., Group 1-4 of Figure 3) in the pre- and post-exercise states.

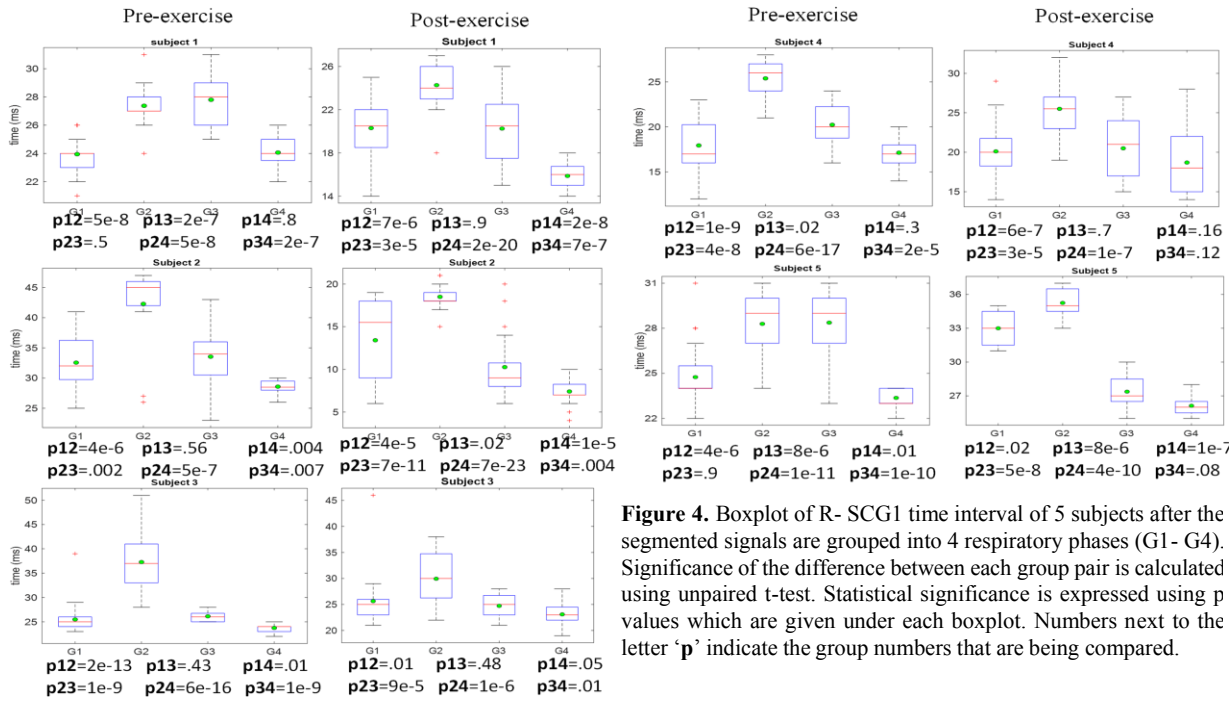

Figure 4. Boxplot of R- SCG1 time interval of 5 subjects after the segmented signals are grouped into 4 respiratory phases (G1- G4). Significance of the difference between each group pair is calculated using unpaired t-test. Statistical significance is expressed using p values which are given under each boxplot. Numbers next to the letter '**p**' indicate the group numbers that are being compared.

For the pre-exercise state, group 2 had highest R- SCG1 interval in 3 out of 5 subjects (subject 2,3,4). In the other 2 subjects, group 2 and 3 signals have similar R- SCG1 interval which were significantly higher than the other 2 groups. On the other hand, for the post-exercise case, all the subjects had their maximum R-SCG1 interval in group 2.

The longer R-SCG1 interval of group 2 and 3 may be explained by discussing the potential effects of respiration on cardiopulmonary interactions. During ventricular systole, the left ventricular (LV) pressure exceeds left atrial (LA) pressure causing mitral valve to shut and contribute the generation of SCG1 event, similar to M1 of first heart sound. Therefore, LV-LA pressure gradient may affect R-SCG1 interval over respiratory phases. A previous study found that the pulmonary capillary wedge pressure (PCWP), which represents the LA pressure, increases during expiration [8]. The increased LA pressure will tend to delay mitral valve closure and lengthen the R-SCG1 interval during expiration, which coincides with Group 2 and 3 in the current study.

The increase in intrathoracic pressure during expiration may also tend to increase this time interval. Here, as the pressure increases to deflate the lungs, it also squeezes out the blood in the inter-alveolar region and increases the left heart filling and left ventricular end diastolic volume [9]. This increases the stroke volume, which would tend to delay the mitral valve closure. The intrathoracic pressure may be higher during early (vs. late) expiration. This may explain the longer R-SCG1 interval observed in group 2 (HLV-EXP) than group 3 (LLV-EXP) for all post exercise subjects and 3 of 5 pre-exercise subjects. The more consistent trend in the post exercise state may be because both breathing rate and tidal volume increase with exercise which may augment the intrathoracic pressure effects.

Although the observations of the study are currently limited to healthy subjects, this may be helpful in diagnosis of various lung and heart conditions. For example, in conditions that can cause airflow obstruction (such as like atelectasis, pulmonary edema or chronic obstructive pulmonary disease (COPD)), the expected changes in the intrathoracic pressure may result in diagnostic SCG. For conditions

like heart failure (HF) with preserved ejection fraction, PCWP was found to be significantly elevated with exercise [10]. This can alter the R-SCG1 interval, which may provide a metric that correlate with HF severity. To confirm the results of the current study and further explore the utility of SCG as a diagnostic tool, more subjects are needed including patients with different cardiopulmonary conditions.

ACKNOWLEDGMENTS

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Abstract

- Heart disease is the leading cause of global mortality. Early detection and regular monitoring of cardiovascular diseases can reduce death rates [1].
- Current methods used to detect and monitor heart diseases are costly and not easily accessible worldwide.
- Seismocardiography (SCG) is a cost-efficient and noninvasive alternative method which measures the vibrations of the chest wall surface induced by mechanical cardiac events [2,3].
- In this study, the effects of exercise on SCG signal are investigated.
- Exercise intolerance is a clinical hallmark of many cardiovascular diseases. But how cardiopulmonary interactions contribute to exercise intolerance is not well understood.
- The aim of this study is to focus on the fundamental mechanisms of cardiopulmonary interaction using Seismocardiography (SCG) signal in healthy subjects.

Methodology

- 5 healthy subjects were recruited for the study.
- Subjects sat on a chair and waited for 10 minutes before data acquisition. This helps to reach a stable resting heart rate for subjects.
- Sensors were placed on subjects and data was acquired for 3 minutes at rest.
- Subjects exercised by cycling on a stationary bike until the target heart rate for moderate exercise level achieved.

  Target heart rate = 220 – *subject's age* ∗ ***0.7***

- After achieving the target heart rate, subjects rested again on the chair and data was acquired for another 3 minutes at the sitting position .

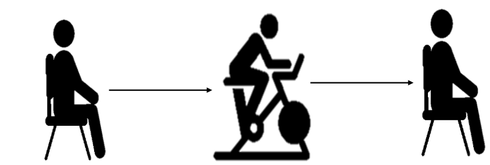

Figure 1 Schematic representation of study protocol

Experiment Setup

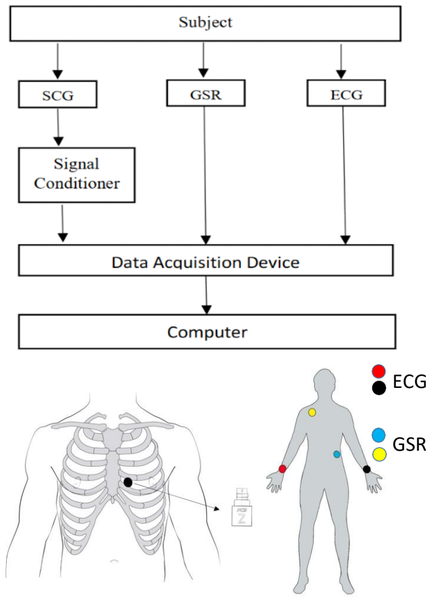

Figure 2 Experiment Setup

Data Analysis

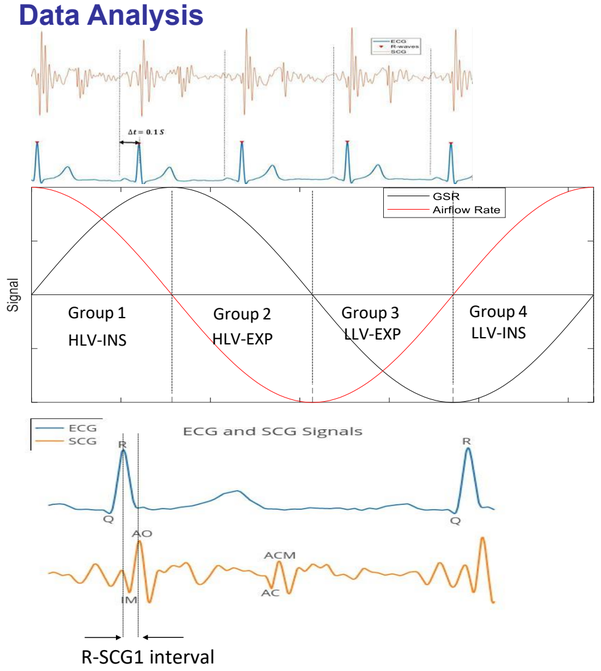

Figure 3 Data Analysis

Results

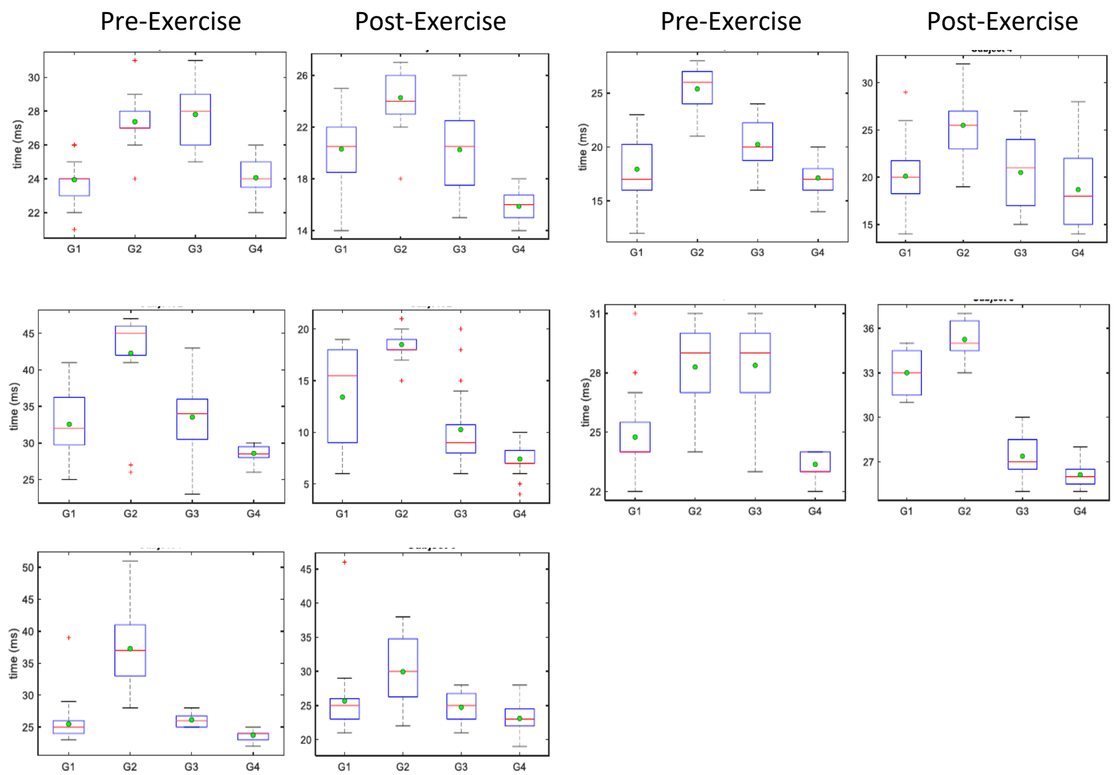

Figure 4 Boxplot of R-SCG1 interval of 5 subjects.

Conclusions

- R-SCG1 interval is higher in G2 and G3 (i.e., expiration)
- SCG1 occurs when left ventricle (LV) pressure exceeds left atrium (LA) pressure and mitral valve closes
- A study found that LA pressure increases during expiration [4]
- This may delay the mitral valve closure and consequently lengthen the calculated interval.
- Moreover, during expiration intrathoracic pressure (ITP) increases to deflate the lungs. While deflating, blood from inter-alveolar region may be squeezed out to LV increasing its stroke volume [5]. This may further increase the interval in G2 and G3.
- ITP is expected to be more in early expiration (G2) than late (G3).
- Due to increased breathing rate and tidal volume after exercise, ITP effect may become more prominent. Hence, higher R-SCG1 interval was found in G2 for all subjects at post-exercise.
- More investigations are needed in larger number of subjects to confirm the results.

Acknowledgments

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