Heart Rate Variability Associated with the Influence of Mood

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Abstract

To understand the control of hemodynamic events elicited by positive and negative induction in mood, we studied heart rate continuously in 10 normal healthy volunteers. Each subject underwent a task in which they encoded positive and negative words, listening music for sad or happy induction for 3 min accompanied by the presentation of series of standardized emotional faces. The words, music and faces were matched in valence. Heart rate variability measured from the standard deviation significantly reduced for the positive mood compared with the negative mood. Power spectrum analysis showed distinctive change in frequency components. Low and high frequency components increased for positive mood. Very low frequency component and total frequency power increased relatively for negative mood. We attribute the increase in low and high frequency components to the response of autonomic arousal mechanism.

1. Introduction

Cardiovascular adaptation to the emotional change depends on the proper interplay of the hemodynamic system and the reflex mechanisms that maintain optimal blood pressure for the particular physiological state. Failure of one of the subsystems to adapt to the emotional stress and circulatory needs may cause insufficient psychosocial activity.

While heart rate response to the emotional state has been studied in the past for both positive and negative mood, there remains a lack of consensus whether the two mood elicit dissociable heart rate variability (HRV) features. Research done in our laboratory aims at using the information contained in the fluctuation response of electrocardiogram (ECG) heart rate to emotional stress. To achieve this goal, it is necessary to understand better the mechanisms underlying the control of hemodynamic events through reciprocal autonomic activation elicited by mood manipulation. Specifically, we are interested in quantifying the degree of similarity between ECG inter-beat interval fluctuation responses seen during positive and negative inductions in mood.

2. Methods

We recruited ten healthy volunteers (5 males, 5 females) to participate in this study. The mean age was 27.5 years. Participants had no sign of cardiovascular disease, emotional disturbance or psychological problem. Each participant was instrumented with a standard clinical ECG monitor. ECG were recorded throughout the duration of the experiment.

Each mood induction procedure took 3 min consisting of presentation of words, music and faces simultaneously. 4 inductions accompanied by 4 intermissions were designed to avoid sequential bias.

Valence matched words were presented every 3 s for 1 s each. Words were translated into Japanese from the ANEW list [1], a database of English words with standardized ratings of valence from 1 to 9. Words with a valence rating 8 and 9 were shown during positive mood induction, and valence rating 1 and 2 were shown during negative mood induction. 60 words for each induction with total of 240 words were chosen. For example, ‘lovely’ was translated into Japanese ‘kawaii’ and used for positive induction, and ‘frightening’ was translated into Japanese ‘kowai’ which was used for negative induction.

Music was played during each induction. We played Spring (from Four Seasons) by Vivaldi for positive mood induction, and Tchaikovsky’s Swan Lake for negative mood induction.

We chose 10 happy and 10 sad faces from standardized emotional faces [2] which were presented every 10 s for 5 s each (Figure 2). Words, music and faces were matched in valence.

Total of 4 inductions were allocated alternatively for positive and negative mood with 5 min of intermissions between and after each induction. The inter-beat intervals from ECG during these intermissions were used for analyses. Subjects rated mood levels (from −10 to +10) using analog scale immediately after each mood induction (post) and just before the subsequent mood induction (pre), i.e. before and after each intermission (Fig 1).
Figure 1. Sequence of events. 4 inductions were allocated alternatively for positive and negative mood with 5 min of intermissions between and after each induction. The inter-beat intervals from ECG during these intermissions were used for analyses.

Figure 2. Example of emotional faces. The two rows of this illustration contain morphed continua ranging between happiness – surprise (top), fear – sadness (bottom).

3. Results

We performed 2 by 2 ANOVA on subjective mood ratings to determine the influence of mood induction, which revealed a main effect of both mood (p=0.01) and timing of subjective mood ratings (post/pre mood induction, p=0.005) without significant interactions. This indicates the difference between subjective mood ratings after positive and negative inductions, as well as the difference between subjective mood ratings taken immediately after the mood induction and just before the subsequent mood induction (Fig.3).

Figure 3. Mean subjective mood ratings taken immediately after each mood induction (post) and just before the subsequent mood induction (pre), i.e. immediately after each intermission. Using analog scale, ratings are scored between −10 and +10, where zero indicates a neutral mood.
ECG data were analyzed for 10 subjects to test for autonomic states in the induced positive and negative mood. Population averaged results for inter-beat intervals were not significantly different between positive and negative mood. Standard deviation of corresponding heart rate variability significantly reduced for the positive mood compared with the negative mood (133ms to 149ms respectively; P=0.03). Most notably, power spectrum analysis using Lomb method showed distinctive change in frequency components. Low frequency component (LF; 0.04Hz to 0.15Hz) and high frequency component (HF; 0.15Hz to 0.40Hz) increased for positive mood (p=0.02). Very low frequency component (VLF; 0.003Hz to 0.04Hz, p=0.01) and total frequency power (TF; p=0.05) increased relatively for negative mood (Fig. 4).

4. Discussion and conclusions

This study was designed to elucidate the heart rate response to the emotional state, namely positive and negative mood. As can be seen from Figure 3, the mood induction was successful for both mood which tends to diminish as time passes after the end of the induction.

We could not find any difference in mean heart rate between positive and negative mood. Significant differences exist, however, between the two moods in frequency components of the heart rate power spectrum. Since high frequency component corresponds to respiratory sinus arrhythmia, the frequency of this component is around the frequency of respiration. Though sympathetic nervous system cannot transfer fluctuations higher than 0.15Hz, parasympathetic nervous system can transfer as high as 1Hz where respiratory sinus arrhythmia is transferred. High frequency component is modulated by parasympathetic nervous system, but not by sympathetic nervous system [3-5]. Mayer wave [6] in arterial blood pressure reflect itself to heart rate through arterial baroreflex which generates low frequency component of the heart rate variability [7,8]. Recently this low frequency component has also been found in heart rate fluctuations under the artificial heart control suggesting central origin of this autonomic nervous rhythm [9]. Since the low frequency component is within the transferable frequency of sympathetic nervous system, this component is modulated by both sympathetic and parasympathetic nervous system. As mentioned above, low frequency component and high frequency component increased for positive mood. We attribute the increase in high frequency component to the response of parasympathetic neural enhancement, and the increase in low frequency component to the response of both parasympathetic and sympathetic arousal mechanism. This result suggests the possibility of heart rate variability as a handy and quantitative mood detector which could be used in evaluating mood.
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References


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