RESPIRATORY SINUS ARRHYTHMIA: A MARKER OF DECREASED PARASYMPATHETIC MODULATION AFTER SHORT DURATION SPACEFLIGHT.

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ABSTRACT

We investigated the hypothesis that the effects of a short duration spaceflight on the autonomic nervous system can be reflected in the respiratory component of heart rate variability (HRV), the respiratory sinus arrhythmia (RSA).

1. INTRODUCTION

Results from previous space missions have shown that early in microgravity (µg) cardiac filling and stroke volume increase, and that there is a decrease in resting heart rate (HR). These changes are the onset of complex adaptation mechanisms in the cardiovascular and cardiopulmonary systems, which play a role on mechanisms of orthostatic intolerance, one of the most important physiological problems endured by astronauts after spaceflight. Although several studies have shown evidences that µg causes adaptation of the autonomic control of the cardiovascular and cardiopulmonary systems, the underlying mechanisms are still unclear. We hypothesized that alterations of the pre-load conditions of the heart could alter RSA. Because RSA is known to be mediated by the parasympathetic nervous system [1], autonomic adaptation and recovery period following space flight should be reflected in the RSA.

2. SUBJECTS AND MEASUREMENTS

Measurements were performed on three healthy male subjects (means ± SD: age 39.3 ± 1.7 yr., weight 75.3 ± 8.0 kg, height 178.3 ± 3.9 cm), the crew members of the 11 days odISSea mission launched on October 30 2002. Pre-flight data collection consisted of 3 sessions performed in the 2 months before launch. Inflight experiments were performed on days 5 and 8 after the launch. Post-flight data were recorded 1, 2, 4, 9, 15, 19 and 25 days after landing. Testing was performed during the normal working period for a subject, but due to the logistical constraints associated with spaceflight, we were unable to fix testing to a particular time in the circadian phase. Electrocardiogram (ECG) and respiratory movements were recorded during a predefined sequence of 5 imposed and controlled breathing protocols, each lasting 3 min and preceded by 1 min resting period. For each protocol the subjects followed on a computer screen a signal which imposed a breathing period of 4, 5, 6.7, 8 and 10 s respectively. The subjects were trained to adapt the depth of their breath, and to breath as normally as possible with a constant ventilation. On ground the same protocols were performed in the supine and active standing postures, the subjects' arms resting at their sides. Between the standing posture protocols, the resting period was increased to 2 min in the sitting posture.

3. METHODS

Heart beat intervals (RRI) and breathing period (Tresp) were measured as time differences between automatically detected heart-beats and onset of inspirations respectively. RSA amplitude and phase were determined breath by breath through the fitting of a model: the polar representation of RSA (see Fig. 1), which is a method employed for the analysis of uncontrolled breathing experiments performed during the Euromir-95 mission [2] and for controlled breathing experiments of the Neurolab mission [3].

![Fig. 1](image_url)
between 50 and 100 % respectively. Heart-beats from adjacent respirations are pooled together to form a graph of RRI versus their phase in breath (see Fig. 1). Then a cosine fitted curve allows breath by breath measurement of the amplitude and phase of the RSA.

4. RESULTS

Our analysis shows that the amplitude of the RSA showed a linear relationship with the breathing period.

![Graph showing RSA amplitude vs. imposed breathing period. Data points are average value (±95 % CI) during the pre-flight baseline data collection for 3 subjects. For the supine data the slope is smaller (p=0.066) and the intercept is larger (p=0.025) than for the standing data.]

In-flight, the slope of this relationship was slightly decreased, with a difference more pronounced for the standing than the supine posture (see Fig 3). For the first days after return, a significant decrease of the slope was observed compared to both supine and standing pre-flight slopes (p<0.05). Between the 15th and 25th days after return, there was a progressive return to normal values of the RSA relationship with Tresp (p>0.05).

5. DISCUSSION

Our results are in agreement with the notion that RSA is due to the gating of vagal activity which is inhibited during inspiration and can express its influence during expiration. The longer the expiration period, the larger the decrease of HR and the larger the amplitude of RSA: this explains the observed linear relationship with Tresp which is preserved in-flight and post-flight. As the amplitude of RSA is a marker of the parasympathetic, vagal mediated, modulation of HR [1], our results are consistent with a decrease in parasympathetic activity during and after spaceflight.

6. CONCLUSION

Our results of decreased amplitude of RSA and decreased slope of the relationship between RSA and Tresp during and after exposure to μg support the hypothesis that the parasympathetic activity of the autonomic nervous system is decreased. We showed also that simultaneous knowledge of HR and Tresp is required for the analysis of RSA as a tool for following-up the progressive re-adaptation to normal gravity of HR autonomic control. This result stresses the importance of the influence of respiration on the interpretation of changes in HRV [4], and the necessity for its control.

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8. REFERENCES