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RESPIRATORY MODULATION OF HEART RATE EXPLAINS THE MAGNITUDE BUT NOT THE PHASE OF RESPIRATORY MODULATION IN ARTERIAL BLOOD PRESSURE

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Introduction: Slow deep breathing (SDB) exercises may be a therapeutic intervention for wellness and wellbeing and can have measurable effects on cardiovascular activity. The mechanisms of action and putative therapeutic benefits of SDB are controversial. During SDB, the blood pressure decreases and the modulation of the blood pressure on the timescale of respiration increases [1]. These changes could be mediated by sympathetic input to the vasculature via sympathetic motor output controlling vasoconstriction or by respiratory modulated vagal input to the heart. Here, we focus on the second mechanism and test the hypothesis that respiratory modulation of the heart rate is sufficient to induce respiratory modulation in the arterial blood pressure.

Methods: We developed a simple computational model of arterial blood pressure in order to analyze the effects of SDB on cardio-respiratory coupling. Model blood pressure incremented at the time of each heartbeat and relaxed between heartbeats. We analyzed ventilation, ECG, and blood pressure from ten volunteer participants during restful breathing, SDB, and a recovery epoch. For each participant, we captured R-peak times from ECG data and input each heartbeat time to the model. The output of each model instance—a surrogate blood pressure trace—was analyzed to compute the depth of respiratory modulation of mean arterial pressure (MAP) as a percent of pulse pressure (PP; the difference between end-systolic and end-diastolic pressures) and the phase offset between the respiratory cycle and the respiratory modulation of MAP.

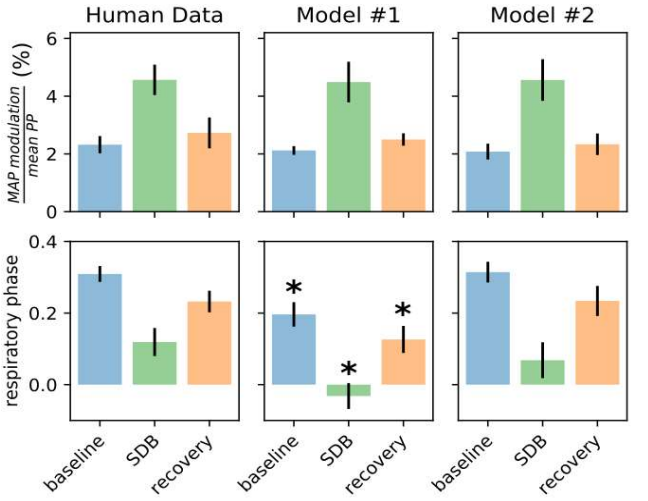

Figure 1. * indicates significant difference from epoch in Human Data ($p < 0.05$).

Results: Our modeling results showed that respiratory modulation of MAP emerged based solely on respiratory modulation of the RR-interval (Top Row, Fig.1). Moreover, the increase in respiratory cycle duration during SDB was sufficient to induce an increase in respiratory modulation of MAP. Surprisingly, this model failed to capture the difference in phase between the respiratory cycle and respiratory modulation of MAP (Bottom Row, Fig. 1). The computational model was extended to incorporate respiratory modulation of PP, which was sufficient to correct the error in phase difference between the respiratory cycle and respiratory modulation of MAP.

Conclusions: We used a simple computational model of arterial blood pressure to show how respiratory modulation of the heart rate was sufficient to induce respiratory modulation in MAP using real human heartbeat times. To correct the phase difference, respiratory modulation of PP was required. These results support the hypothesis that respiratory modulation of MAP emerges from respiratory modulation of vagal tone to the heart, but phase modulation may require baroreceptor input. We demonstrated how this mechanism supports increased cardio-respiratory coupling during SDB.

References:

Dick et al. *Respir Physiol Neurobiol.* 204, 2014.