An organism's ability to flexibly modulate patterned behavior is essential for maintaining homeostasis amidst changing external conditions. Central to this model is the central pattern generator (CPG), which creates predictable output that controls rhythmic behaviors such as walking, breathing, and chewing. One such CPG is the crustacean cardiac ganglion (CG), located within the neurogenic heart, which controls the lobster’s heart rate and amplitude. The cardiac ganglion consists of 5 large motor neurons that project directly onto the cardiac muscle as well as 4 smaller interneurons that have been shown to initiate activity of the motor neurons through electrical and chemical coupling (Alexandrowicz, 1932).

The output of this CPG is subject to extensive modulation by a variety of neuromodulators that act upon the cardiac ganglion and periphery. Two other feedback pathways, distinct in that they do not utilize peptides, have also been found to have a significant modulatory effect. Of these, muscle stretch is thought to allow the cardiac ganglion to process information on relevant changes, such as the degree of filling in the heart, and subsequently adjust motor output.

Though there has been much work done on the CPG’s responses to tonic stretches in the whole heart (Dickinson, 2014; Chin-Purcell, 2014), as well as phasic stretches on the semi-isolated CG (Harmon, 2014), no significant work has been done on the CPG’s responses to phasic stretches in the whole heart. It has been found that it is possible to entrain the CPG in a semi-isolated CG preparation by imposing repeated phasic stretches at a frequency out of tempo from the natural rhythm, albeit in a small range of frequencies (Harmon, 2014). Based on this study, I seek to test whether phasic stretches can also cause entrainment in the whole heart preparation, and if so, what range of frequencies this entrainment is limited to.

Because the CG is located within the lumen of the heart, and most cardiac modulators are delivered to the heart hormonally, the cardiac ganglion and adjacent muscles are typically exposed to modulators simultaneously. Few studies have examined the ways in which this concurrent modulation integrates to produce a singular effect. I seek to utilize two peptides GYS (GYNRSFLRFamide) and SGRN (SGRNFLRFamide) that are known to modulate the output of the heart, affecting contraction amplitude and frequency in the whole heart, as well as the output of the cardiac ganglion itself (Dickinson, Calkins, and Stevens; 2014). In Homarus, the effects of GYS and SGRN are known to be dependent on dosage and location of application.

I am characterizing the effects of phasically stretching the whole heart, then identifying the effect that neuropeptides GYS and SGRN have on responses to these phasic stretches. This was accomplished by emulating Chin-Purcell and Dickinson’s methods, which involve isolating the intact heart and cannulating the large posterior artery with small tubing allowing saline to perfuse inwards. I then hook the anterior arteries using an Aurora dual-lever system and apply quantifiable phasic stretches while checking for entrainment. Lastly, I apply neuropeptides GYS and SGRN to observe how contrasting modulators affect the whole-heart CPG system. In doing so, I had noticed that the amplitude of the force applied had an effect upon the range of entrainment. As such, I searched for an amplitude of imposed stretch that would best cause entrainment.

Preliminary results indicate that the range of contraction periods is very small for our control group, which could be why previous attempts had not succeeded. However, we notice an overall increase in the entrainment range by the application of peptide, with a significant increase for SGRN $10^{-9}$ M from control. In addition, application of peptide also increases the range of imposed stretch amplitudes that cause entrainment.

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References


