Central pattern generators (CPG) are small neuronal networks that endogenously produce stereotyped motor output, controlling behaviors such as breathing, swallowing and walking. Flexible internal and external modulations of rhythmic motor behaviors controlled by CPG are key to maintaining homeostasis. The neurogenic heart of the American lobster is controlled by a simple CPG, the cardiac ganglion (CG), which consists of five motor neurons and four small premotor neurons that are all electrically and chemically coupled. Four small interneurons provide the excitatory input that initiates driver potentials in the five anterior motor neurons. The action potentials generated by the five motor neurons drive heart contractions. The CG receives feedback from the myocardium. Nitric oxide inhibits and slows the heartbeat, while feedback from stretch sensitive dendrites is thought to exert positive feedback and increases burst frequency. The lobster heart is also modulated by hormonally delivered peptides, which act at multiple sites, including the CG and the periphery. The two feedback pathways and the modulation by neuropeptides, together with the CG itself, control the heartbeat.

The goal of my study is to elucidate the mechanisms underlying stretch feedback in the cardiac neuromuscular system of the American lobster, Homarus americanus, and to determine whether and how stretch feedback interacts with extrinsic neuromodulators. To do this, I have been examining the effects of stretch and current injection in the motor neurons of the cardiac ganglion in the presence and absence of a neuropeptide, SGRNFLRFamide (SGRN).

After isolating the cardiac ganglion along with muscles surrounding and underlying the pacemaker cells, I recorded intracellularly from cardiac motor neurons and extracellularly from one motor nerve of the CG, while the muscle was stretched. Simultaneous intracellular recordings from two motor neurons showed that the motor neurons generate driver potentials with the same burst frequency, duration, and cycle periods. Stretch elicits identical effects on both cells. During tonic stretch, motor neurons with relatively long intrinsic burst durations showed significant decreases in both cycle duration and burst frequency. However, although extension caused significant decreases in burst duration, release caused significant increases in burst duration of the CG motor neurons. Moreover, as the force increased, the effect of the stretch increased.

Repeated consecutive phasic stretches were applied to the CG to mimic heartbeats. The ability of the CG to be entrained by these stretches appears to be dependent on the intrinsic burst duration. CGs with longer intrinsic burst duration tend to be entrained to a range with shorter burst period and CGs with shorter intrinsic burst durations tend to be entrained to a range with longer burst period. A brief stretch applied on the CG could cause either phase advance or phase delay. The positive and negative extremes of phase shifts determine the entrainment range of the CG. Application of SGRN at 10^{-6}M decreased the entrainment range of the CG and the extremes at both ends of the phase shift. By injecting short current pulses into the motor neurons of the CG, similar phase shifts and entrainment ranges of the CG can be generated, enabling us to correlate the effects of potential ion movements through stretch-activated channels and stretch. Depolarizing, but not hyperpolarizing, currents can entrain the CG similarly to stretch. As the magnitude of the current increases, the entrainment range of the CG increases. The application of SGRN at 10^{-6}M decreased the range of frequencies over which current injections could entrain the CG.

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**References**