Central pattern generators (CPGs) control rhythmic behaviors such as breathing and heartbeat in invertebrates (Calabrese and Marder 1996). Malfunctions in a CPG may have a variety of detrimental effects such as abrupt disruption of breathing, as has been implicated in sudden infant death syndrome (SIDS) (Paydarfar et al. 2005; Moon et al. 2007). In the United States SIDS has been responsible for approximately 2,300 infant deaths annually since 2001 (Moon and Fu 2012). SIDS may cause the unexpected stop of breathing of an infant. While the causes of SIDS are largely unknown, mutations in the channels conducting the hyperpolarization-activated current ($I_h$) can predispose an infant to SIDS (Mitterauer et al. 2000). Inspiration is controlled by the CPG in the pre-Bötzinger complex (Smith et al. 1991). Mathematical models of single neurons of the CPG display tonic spiking, bursting, or silence (Butera et al. 1999), they have also been shown to exhibit a coexistence between bursting and silence (Malashchenko 2012). This type of bistability has been intensely studied in leech heart interneurons and has been shown to occur within a specific range of leak conductance values (Malashchenko et al. 2011). The propensity index for bistability was defined by this range (Malashchenko 2012). In this study increases in the conductance of $I_h$ ($g_h$) caused increases in the propensity index. Our hypothesis was that increases in $g_h$ under variations in kinetics of $I_h$ would expand the propensity index to bistability. We investigated how changes in voltage of half-activation and steepness of the steady state activation function affect the propensity index. In contrast with studies done on the leech heart interneuron the propensity index in our study exhibited a resiliency to variations in $I_h$. This suggests that resiliency may be intrinsic to these neurons. Future studies should aim to investigate the response of the propensity index to other currents’ variations.