The Frank-Starling relationship describes the ability of the heart to increase the power of contraction when there is an increase in the blood received by the heart (preload). During heart failure, the Frank-Starling relationship loses effect as the cardiac muscle has decreased ability to respond to an increase in preload, primarily due to decreased strength of the cardiac muscles. The body responds by activating the sympathetic nervous system, specifically the Beta-Adrenergic system, to increase the contractile strength of the heart. Recent work in our laboratory has shown that phosphorylation of cardiac TnI (cTnI), via the beta adrenergic pathway, increases the effect of the Frank-Starling relationship. It is also apparent that phosphorylation of cTnI was both necessary and sufficient to mediate this response. The goal of this study was to investigate whether these results translate to the organ (heart) level. The hypothesis was tested by comparing the amount of cTnI phosphorylation to the contractile strength of the same isolated rat heart. The finding that these two parameters were highly correlated suggests that modification of cTnI by phosphorylation translates to an increase in heart function and may be a key role in reviving the Frank-Starling relationship in heart failure patients.