The results presenting at this PhD defense is based on the pathophysiological changes that occur in the coronary vasculature and surrounding myocardial tissue following experimental AMI and reperfusion. The results demonstrate that ischemia-reperfusion (I/R) injury leads to an increased stretch-induced basal tone of the affected coronary artery as well as increased contractile response mediated through smooth muscle endothelin B receptor subtype (ET\textsubscript{B}-Rs), which show an entirely different calcium handling than is observed with smooth muscle ET\textsubscript{A}-Rs. A shift in intracellular calcium handling may augment vasoconstriction from vasoactive peptides and alter the mechanisms of recovery, leading to further complications following AMI and reperfusion injuries. Endothelial-dysfunction manifests itself clinically in most ischemic cardiovascular diseases, and endothelium-derived vasodilation was investigated. NO bioavailability was found to be reduced following I/R; however H\textsubscript{2}O\textsubscript{2} appears to act as a compensatory endothelial-derived vasodilator in post-ischemic LAD coronary arteries only. These results need further attention, but might provide new insights into future treatment strategies for the sub-acute AMI patient.