The sympathetic nervous system is an important extrinsic mechanism that controls myocardial function. Sympathetic neurons within the stellate ganglion (SG) innervate the heart and they regulate both heart rate and contractility. Undesirable functional effects occur in cardiac tissue as a result of an alteration in sympathetic nervous system function. During ischemia, for example, sympathetic activity increases such that both cardiac workload and oxygen consumption are elevated. Under these conditions, cardiac injury may occur. Studies now show that opioid receptor activation appears to play a major cardioprotective role in the phenomenon known as ischemic preconditioning. Opioid receptors are widely distributed in the peripheral nervous system and are capable of eliciting potent cardiovascular and respiratory responses. The purpose of this talk is to present evidence demonstrating that SG neurons express ORL1 opioid receptors, while the other three “classical” receptors are not present. The functional data also shows that activation of ORL1 receptors with the endogenous ligand, nociceptin, results in Ca\textsuperscript{2+} channel inhibition. The presence of ORL1 receptors in the peripheral nervous system suggests that the cardiovascular effects produced by nociceptin are a result of the peptide’s ability to modulate autonomic neurotransmission at several levels.