Cardiac function is regulated in part by the sympathetic branch of the autonomic nervous system via the stellate ganglion (SG). Neuropeptides and neurotransmitters regulate the excitability of sympathetic neurons by modulating ion channel function. There is evidence that circulating levels of opioids—produced in sympathetic nerve terminals—help to reduce sympathetic activity via pre- and post-synaptic opioid receptors. To better understand the neuronal cellular processes by which the endogenous opioid peptide nociceptin regulates sympathetic neuron excitability and consequently heart rate and contractility, it is necessary to identify the components involved in this transduction pathway and how this information is transduced into electrical excitability. Nociceptin activates ORL1 opioid receptors, members of the G protein coupled receptor (GPCR) superfamily. In this lecture, we will describe studies that examine the signaling mechanisms by which activated ORL1 receptors modulate N-type Ca$^{2+}$ channels in rat SG neurons that innervate cardiac muscle.