The scheme of ionic currents within a cardiac cycle for a mammalian pacemaker cell (i.e., SA node) is generally described with the background $[Ca^{2+}]_i$ continually increasing and decreasing. Starting in diastolic depolarization with a slow release of Ca^{2+} by ryanodine receptors (RyR), from the SR, leads to a rise in $[Ca^{2+}]_i$. The SERCA pumps Ca^{2+} back into the SR and the NCX removes $[Ca^{2+}]_i$ in exchange for Na^+ ions across the plasma membrane of the cell. The influx of Na^+ ions can lead to a depolarization of the plasma membrane. Thus, opening low voltage-gated T-type Ca^{2+} channels (V_{Ca}) (Hüser *et al.* 2000) and potentially voltage-gated Na^+ channels. The influx of Ca^{2+} acts on the RyR to cause the ER to dump Ca^{2+} which results in a calcium induced inhibition of the RyR. Until the $[Ca^{2+}]_i$ is reduced by the SERCA and NCX, the RyR stay inhibited but will start leaking Ca^{2+} as $[Ca^{2+}]_i$ returns to a low level to then repeat the cycle (Subramani and Subbanna, 2006). In the mammalian heart, the pace making sinus node cells do not contain a K^+ current (I_{K1}) which is thought to be one reason the pacing cells do not show a resting membrane potential (Opthof, 2007).