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Changes in Intracellular Ca^{2+} Concentration Induced by L-Type Ca^{2+} Channel Current in Guinea-Pig Gastric Myocytes

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We investigated the relationship between the voltage-operated calcium channel current and the corresponding $[Ca^{2+}]i$ change (Ca^{2+}-transient) in guinea-pig gastric myocyte. Fluorescence microspectroscopy was combined with conventional whole-cell patch clamp technique and fura-2 (80 μM) was added into the CsCl-rich pipette solution. Step depolarization to 0 mV induced inward Ca^{2+}-current (I_{Ca}) and concomitantly raised $[Ca^{2+}]i$. Both changes were abolished by nicardipine, an L-type calcium channel antagonist, and the voltage dependence of Ca^{2+} transient was similar to the current-voltage relation of I_{Ca}. Peak Ca^{2+} transient increased with the increment of pulse duration up to 900ms and reached a steady state for longer stimulation. The calculated fast Ca^{2+} buffering capacity(B-value), determined as the ratio of the time integral of I_{Ca} divided by the amplitude of Ca^{2+} transient was usually above 100. The time course of the measured increase in $[Ca^{2+}]i$ followed the time-integrated I_{Ca}. Treatment with caffeine and ryanodine decreased both Ca^{2+} transient and I_{Ca} but the changes in B-values were not statistically significant. However, cyclopiazonic acid (CPA, an inhibitor for Ca^{2+}-ATPase in SR membrane) significantly decreased the B-value. With K-rich pipette solution, fast transient oscillatory outward currents ($I_{K(Ca)}$) were induced by depolarization with Ca^{2+}transient. $I_{K(Ca)}$ were sensitively suppressed by CPA while the change in the peak amplitude of Ca^{2+} transient was small. Above results suggest that the Ca^{2+} transient is tightly coupled to I_{Ca} and SR of guinea-pig gastric myocyte play a role as a Ca^{2+} buffer barrier rather than as a source of Ca^{2+} via Ca^{2+} induced Ca^{2+} release process in Ca^{2+} transient.