Role of SAK_{Ca} channels in stretch-induced extrasystoles

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We have previously reported the electrophysiological properties of stretch-activated BK_{Ca} (SAK_{Ca}) channels cloned from cultured chick embryo ventricular myocytes. This type of channel is a voltage-sensitive K^+ channel, which is activated by intracellular Ca^{2+} ($[Ca^{2+}]_i$) and membrane stretch. Despite its unique electrophysiological characteristics, the physiological role of SAK_{Ca} channels in the mature *in situ* heart is not well known. In the present study, we investigated their role in acute left ventricular stretch-induced extrasystoles (SIE) using an isolated 2-week-old Langendorff-perfused chick heart. A thin latex balloon was fitted into the left ventricular (LV) chamber. The ventricular wall was rapidly stretched by application of a volume change pulse to the balloon. As the speed of the stretch increased, the probability of SIE also significantly increased; significantly shortening the delay between SIE and the initiation of the stretch. Application of 100 nM of *Grammostola spatulata* mechanotoxin 4, a cation-selective stretch-activated channel (SAC) blocker, significantly decreased the probability of SIE. However, the application of Iberiotoxin, a SAK_{Ca} channel blocker, significantly increased the probability of SIE, suggesting that a K^+ efflux via a sarcolemmal BK_{Ca} channel reduces SIE by balancing out stretch-induced cation influx via SACs.