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Gating Current of the KCNQ1 Voltage-Gated Potassium Channel

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KCNQ1 pore-forming alpha-subunits are crucial to physiology, operating *in vivo* with KCNE1 and KCNE3 beta-subunits in the heart and stomach, respectively. Recently, the gating currents of KCNQ4 and KCNQ5 channels were characterized (Miceli, Channels, 2009); here, equivalent measurements are described for KCNQ1 channels formed in the absence of beta-subunits. Human KCNQ1 was studied in *Xenopus* oocytes with the cut-open oocyte voltage clamp technique. To record gating currents, cells were depleted of internal potassium ions and residual ionic current was blocked with tetraethyammonium and barium. At room temperature, gating currents were too small to resolve. At 28°C, ON gating current gave rise to peak charge movement at +40 mV of ~0.7 nC / μA of ionic current. The total charge movement at each test potential was conserved in the OFF-gating currents. Analysis of the charge-voltage (QV) relationship and conductance-voltage (GV) relationship showed a 10 mV hyperpolarizing shift of the half-maximal voltage of activation of the normalized QV curve with respect to the normalized GV curve. KCNQ1 ON-gating current decay constants were 4-fold slower than in KCNQ4 and required longer test pulses to fully resolve. Overlays of gating and ionic currents revealed that, as for KCNQ4, gating charges were still moving even after KCNQ1 channels started to open, indicating that charge movement was a rate-limiting factor in channel opening. These first characterizations of KCNQ1 gating currents and are an important step towards understanding mutations that lead to cardiac arrhythmias, such as long-QT syndrome, and the effects of beta-subunits on channel function. Supported by NIH GM030376, University of Chicago MSTP, and The Paul and Daisy Soros Fellowship for New Americans.