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Mechanisms of Isoflurane- and Halothane-Induced Decrease of Single Potassium Channel Current in Isolated Coronary Smooth Muscle Cells

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Aim. To determine and compare the effects of isoflurane and halothane on the large-conductance K+ channel current from the canine coronary artery smooth muscle cells.

Methods. We used cell-attached and inside-out patch-clamp techniques to record K+ current through individual K^+ channels and the effects of volatile anesthetics on this current.

Results. In the cell-attached mode, both anesthetics decreased the probability of K+ channel opening in voltage-independent manner. At the resting membrane potential, isoflurane (2.6%) and halothane (1.5%) decreased the probability of channel opening from 0.0053±0.0001 to 0.0030±0.0001 (p£0.05) and 0.0018±0.0001 (p£0.05), and increased the mean closed time from control of 384±98 to 531±113 ms (p£0.05), and from 302±80 to 636±56 ms (p£0.05), respectively. Halothane, but not isoflurane decreased the mean open time from control of 11±1.5 to 8±1.4 ms (p£0.05), shifted current-voltage curve upward, and changed reversal potential toward less negative pipette potential. Channel conductance in cell-attached patches was 100 pS in control condition, and was not altered by either anesthetic. Similarly, in the inside-out mode, the probability of channel opening and mean open time decreased, mean closed time increased, while channel conductance was not altered in the presence of inhalational anesthetics.

Conclusion. The increased mean closed time and decreased probability of channel opening by isoflurane and halothane, and decreased mean open time by halothane, are the mechanisms by which volatile anesthetics decrease macroscopic K+ current in the canine coronary artery smooth muscle cells.

Key Words: *coronary vessels; halothane; potassium channels*

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