Analysis of in vivo electropharmacological effects of vanoxerine

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Introduction: While a dopamine re-uptake inhibitor vanoxerine suppresses I_{Kr}, I_{Na} and $I_{Ca,L}$ in vitro, its electropharmacological information in vivo is limited. **Methods:** Vanoxerine dihydrochloride was intravenously administered at 0.03 and 0.3 mg/kg to the halothane-anesthetized dogs (n=4) under the monitoring of cardiovascular variables. **Results:** The low dose increased the heart rate and cardiac output, whereas no significant change was observed in the mean blood pressure, ventricular contraction or pre/afterload. It prolonged the ventricular effective refractoriness without any change in ECG variables. The high dose decreased the heart rate, increased the afterload, but it did not alter the other cardiohemodynamic variables. It delayed the early as well as late repolarization, and equally prolonged the atrial and ventricular effective refractoriness. No significant change was detected in the intra-atrial, atrioventricular-nodal or intra-ventricular conductions. **Conclusions:** Cardio-stimulatory responses after the low dose could be explained by the dopamine re-uptake inhibitory mechanism. In vivo electropharmacological effects of vanoxerine may largely depend on the I_{Kr} and I_{Na} inhibition, whereas $I_{Ca,L}$ suppression may play a minor role.