Effect of nitric oxide on strophanthidin-induced ventricular tachycardia

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Abstract

Nitric oxide (NO) has been demonstrated to have several effects on the heart. Through the stimulation of guanylate cyclase, NO increases cyclic GMP and decreases intracellular calcium. The purpose of this study was to evaluate the effects of NO on ventricular arrhythmia induced by strophanthidin in guinea pigs and dogs. In experiment 1, after strophanthidin-induced ventricular tachycardia, guinea pigs received different doses of L-arginine (0, 25, 50, 100, 200, and 400 mg/kg; n = 10 for each dose), 200 mg/kg L-arginine combined with 100 mg/ kg N-G-nitro-L-arginine methylester (L-NAME, n = 10), or 200 mg/kg D-arginine (n = 10). In experiment 2, after strophanthidin-induced ventricular tachycardia, dogs (n = 7) received 200 mg/kg L-arginine. By 12-lead EGG, monophasic action potentials in left and right ventricles were recorded throughout the study. In experiment 1, guinea pigs which received 200 mg/kg or 400 mg/kg L-arginine had greater incidences of ventricular tachycardia termination (60 and 80%, respectively) than those which received 0, 25, 50, and 100 mg/kg L-arginine (0, 0, 20, and 30%, respectively), those which received L-arginine with L-NAME (0%), and those which received D-arginine (0%). In experiment 2, 5 (71%) of the dogs had successful termination of ventricular tachycardia. These findings suggest that L-arginine was effective in treating strophanthidin-induced ventricular tachycardia in vivo and that the underlying mechanism is th rough a NO pathway. Copyright (C) 2001 S.Karger AG, Basel.