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<http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?db=PubMed&cmd=Display&dopt=pubmed_pubmed&from_uid=8726602> Related Articles, <javascript:PopUpMenu2_Set(Menu8726602,",",",",",");> Links

The effect of cocaine on Ventricular fibrillation threshold in the normal canine heart

Tisdale JE, Shimoyama H, Sabbah HN, Webb CR.
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We determined the effect of cocaine on ventricular vulnerability to fibrillation, as measured by ventricular fibrillation threshold (VFT), and cardiac electrophysiology in 20 anesthetized dogs with normal hearts. Animals were randomized in blinded fashion to receive a continuous 3-hour infusion of cocaine 0.11 mg/kg/minute (total dose 20 mg/kg) or placebo (lactose dissolved in normal saline). The VFT, systolic and diastolic blood pressures, ventricular effective refractory period (ERP), and electrocardiographic intervals were measured at baseline and every 30 minutes during infusion. Baseline mean +/- SE VFT in cocaine and placebo groups was 57.0 +/- 7.8 and 51.8 +/- 7.6 mA, respectively ($p = 0.64$). Cocaine did not significantly decrease VFT, but actually increased it (i.e., reduced ventricular vulnerability to fibrillation) compared with placebo (84.6 +/- 10.4 vs 55.8 +/- 7.2 mA, respectively, at 150 minutes, $p = 0.04$). Cocaine prolonged ERP and PR, QRS, QT, QTc, JT, and JTc intervals. Cocaine does not increase ventricular vulnerability to fibrillation in anesthetized dogs with normal intact hearts. Its electrophysiologic effects are similar to those of class I antiarrhythmic agents in this model.

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