Effect of an Electronic Control Device Exposure on a Methamphetamine Intoxicated Animal Model

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Introduction

Electronic Control Devices (ECDs) are used primarily by law enforcement officers to subdue combative subjects. Detained combative subjects occasionally deteriorate for unknown reasons and suffer in-custody death (ICD).

Subjects suffering ICD often are in excited delirium, a condition associated with both stimulant intoxication and characterized by agitation and non-coherent behavior, elevated temperature, and excessive endurance without fatigue. It is hypothesized that both stimulant intoxication combined with ECD application may produce a deleterious effect.

Animal studies have demonstrated cardiac capture and ventricular fibrillation (VF) with ECD application directly over the cardiac apex. This was not reproduced in a similar study involving humans.1 Study of cocaine intoxication in pigs demonstrates a cardioprotective effect with regard to arrhythmia induction from ECD application (VT threshold is increased).2 Methamphetamine intoxication (MA) in combination with ECD exposure has not been studied. This is the first study to examine the cardiac effects of ECDs on MA sheep.

Objective

To determine cardiac and metabolic effects of an ECD exposure in the presence of methamphetamine intoxication (MA). The ECD is used to control violent or agitated subjects. These subjects may have MA present. Death has occurred in this population on occasion.

We present findings on ECD applications in combination with MA using an animal model.

Methods

16 New Zealand sheep (24-79 kg) had cardiac rhythm and arterial blood sampling at baseline and after each intervention. The sheep received 0.0, 0.5, 1.0, or 1.5 mg/kg IV methamphetamine (6 animals in each group). The sheep were observed for 30 minutes after methamphetamine administration.

ECD-darts were inserted to death at the sternal notch and the cardiac apex. All animals received ECD exposures in sequences 5, 15, 30, and 45 seconds. There was a 15-minute rest between applications.

Cardiac motion was monitored by echocardiography. After the 45 second exposure, a thoracotomy was performed. The ECD exposure was repeated with visualization of the heart in order to determine cardiac capture. ECD darts were inserted to death at the sternal notch and the cardiac apex. Blood samples were analyzed for artifacts at baseline, after MA and after each ECD exposure.

Results

All animals demonstrated signs of MA including atrial and ventricular ectopy before exposure. Small animals (n=8, <85 kg) had supraventricular dysrhythmias and large animals (n=8, >85 kg) had sinus tachycardia after exposures. One of the smaller animals had ventricular ectopy including a brief run of ventricular tachycardia after exposure that spontaneously resolved.

Five animals of varying size (26-74 kg) had negligible cardiac capture during exposure but no ventricular fibrillation (3 control animals, 2 animals with 1.5 mg/kg MA). There was immediate reversion to sinus tachycardia when the application was stopped. There was significant change in pHi and lactate from baseline when compared with MA. No significant differences in pHi or lactate were noted between MA and post ECD Exposure.

Conclusions

Methamphetamine intoxication (MA) caused evidence of cardiac irritability independent of ECD exposure. Animals of lower body weight demonstrated supraventricular dysrhythmias after ECD exposure.

After the initial 30 minutes of MA, larger animals had sinus tachycardia that generally increased after ECD exposure (but showed intra-exposure slowing of heart rate). There was variable cardiac capture that was not associated with MA and no induction of ventricular fibrillation.

Discussion

ECDs are classified by the department of defense as intermediate weapons and are devices that induce subject compliance due to pain or incapacitation. Similar weapons in this category include aerosolized chemical irritants, impact balls, and projectile beanbags. The most commonly used ECD in law enforcement is the TASER X26°.

No previous studies have examined the effects of ECDs on MA in mammals. We used doses of methamphetamine that, on their own, have been reported to be lethal in humans. No animals died during the experiment. Cardiac capture was observed, however it occurred independent of MA and not always in the same animal.

Supraventricular tachycardias were observed in the small animal group, but not in the large animals.

Previous animal studies were able to demonstrate cardiac capture, which our study did as well. However, VF, which was observed in some previous vertebrate studies, was not observed in ours. Critics of ECDs point to VF or cardiac capture as possible mechanisms of death in cases where an ECD is applied in proximity to an ICD. However, review of most ECDs associated with SD application reveals that collapse does not occur immediately after the ECD application, which, physiologically, is inconsistent with an induced VF mechanism for death.

Furthermore, cardiac capture has not been demonstrated in human studies using real-time echocardiography; suggesting capture is more likely related to anatomic differences between sheep, swine, and humans.

References
