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 illicit stimulant intoxication and characterized by agitated and non-coherent behavior, elevated temperature, and excessive endurance without fatigue.[1] It is hypothesized that illicit stimulant intoxication combined with an ECD application may produce a deleterious effect.

Animal studies have demonstrated cardiac capture and ventricular fibrillation (VF) with ECD application directly over the cardiac axis.[2] This was not reproduced in a similar study involving humans.[3] Study of cocaine intoxication in swine demonstrates a cardioprotective effect with regard to arrhythmia induction from ECD application (the VF threshold is increased).[4] Methamphetamine intoxication (MI) in combination with ECD exposure has not been studied. This is the first study to examine the cardiac effects of ECDs on MI sheep.

Objective

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

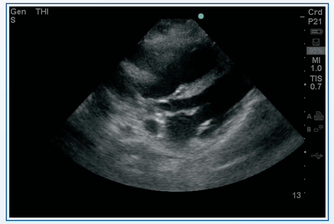
We present findings on ECD applications in combination with MI using an animal model.

Methods

16 Dorset sheep (26-78 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 of IV methamphetamine (4 animals in each group). The sheep were observed for 30 minutes after methamphetamine administration.

ECD darts were inserted to depth at the sternal notch and the cardiac apex. All animals received ECD exposures in sequence: 5; 15; 30; and 45 seconds. There was a 3-minute rest between applications.

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

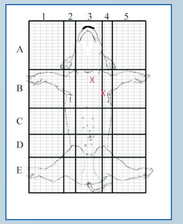


Animal	Dose	Weight	Skin to Heart Distance	Pre-exposure Heart Rate	Post Methamphetamine Heart Rate	Post 5-second ECD Exposure Heart Rate	Post 15-second ECD Exposure Heart Rate	Post 30-second ECD Exposure Heart Rate	Post 45-second ECD Exposure Heart Rate	Notes
1	0	31 kg	23 cm	68		102	163	119	155	Control; PACs post 30-second exposure; capture
2	0	30 kg	24 cm	84		125	145	87	85	
3	0	72 kg	30 cm	55		190	191	273	151	Capture
4	0	74 kg	34 cm	76		77	77	240	180	
5	0.5 mg/kg	27 kg	21cm	75	147	144	140	130	138	
6	0.5 mg/kg	68 kg	35 cm	77	142	195	145	134	137	
7	0.5 mg/kg	78 kg	34 cm	69	160	101	95	120	112	Ectopy post methamphetamine
8	0.5 mg/kg	27 kg	22 cm	80	115	110	104	99	95	Ectopy post methamphetamine
9	1.0 mg/kg	32 kg	21 cm	103	202	155	159	186	191	Delayed ventricular ectopy post ECD exposures
10	1.0 mg/kg	32 kg	21 cm	85	179	109	124	151	142	SVT post 30 second exposure
11	1.0 mg/kg	68 kg	31 cm	68	199	212	130	134	110	
12	1.0 mg/kg	68 kg	29.5 cm	82	230	196	170	168	102	
13	1.5 mg/kg	78 kg	46 cm	91	220	200	207	220	208	Ectopy post methamphetamine
14	1.5 mg/kg	73 kg	38 cm	77	220	163	173	112	149	
15	1.5 mg/kg	26 kg	20 cm	92	194	155	159	134	160	SVT post 30 second exposure; capture
16	1.5 mg/kg	30 kg	22 cm	80	174	167	163	150	157	Ectopy post methamphetamine; intermittent capture

Results

All animals demonstrated signs of MI including atrial and ventricular ectopy before exposure. Small animals (n=8, <38.5 kg) had supraventricular dysrhythmias and large animals (n=8, >68 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 reliable cardiac capture during exposure but no ventricular fibrillation (3 control animals, 2 animals with 1.5 mg/kg MI). There was immediate reversion to sinus tachycardia when the application was stopped. There was significant change in pH and lactate from baseline when compared with MI. No significant differences in pH or lactate were noted between MI and post ECD Exposure.



Conclusions

Methamphetamine intoxication (MI) 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 MI, 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 MI 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 batons, 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 MI 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 MI, 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 swine model 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 ICDs. However, review of most ICDs associated with ECD 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

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