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

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Abstract

Objectives: Because of the prevalence of methamphetamine abuse worldwide, it is not uncommon for subjects in law enforcement encounters to be methamphetamine-intoxicated. Methamphetamine has been present in arrest-related death cases in which an electronic control device (ECD) was used. The primary purpose of this study was to determine the cardiac effects of an ECD in a methamphetamine intoxication model.

Methods: Sixteen anesthetized Dorset sheep (26–78 kg) received 0.0 mg/kg (control animals, $n = 4$), 0.5 mg/kg ($n = 4$), 1.0 mg/kg ($n = 4$), or 1.5 mg/kg ($n = 4$) of methamphetamine hydrochloride as a slow intravenous (IV) bolus during continuous cardiac monitoring. The animals received the following exposures in sequence from a TASER X26 ECD beginning at 30 minutes after the administration of the drug: 1) 5-second continuous exposure, 2) 15-second intermittent exposure, 3) 30-second intermittent exposure, and 4) 40-second intermittent exposure. Darts were inserted at the sternal notch and the cardiac apex, to a depth of 9 mm. Cardiac motion was determined by thoracotomy (smaller animals, ≤ 32 kg) or echocardiography (larger animals, > 68 kg). Data were analyzed using descriptive statistics and chi-square tests.

Results: Animals given methamphetamine demonstrated signs of methamphetamine toxicity with tachycardia, hypertension, and atrial and ventricular ectopy in the 30-minute period immediately after administration of the drug. Smaller animals ($n = 8$, ≤ 32 kg, mean = 29.4 kg) had supraventricular dysrhythmias immediately after the ECD exposures. Larger animals ($n = 8$, > 68 kg, mean = 72.4) had only sinus tachycardia after the exposures. One of the smaller animals had frequent episodes of ventricular ectopy after two exposures, including runs of delayed onset, nonsustained six- to eight-beat unifocal and multifocal ventricular tachycardia that spontaneously resolved. This animal had significant ectopy prior to the exposures as well. Thoracotomy performed on three smaller animals demonstrated cardiac capture during ECD exposure consistent with previous animal studies. In the larger animals, none of the methamphetamine-intoxicated animals demonstrated cardiac capture. Two control sheep showed evidence of capture similar to the smaller animals. No ventricular fibrillation occurred after the exposure in any animal.

Conclusions: In smaller animals (32 kg or less), ECD exposure exacerbated atrial and ventricular irritability induced by methamphetamine intoxication, but this effect was not seen in larger, adult-sized animals. There were no episodes of ventricular fibrillation after exposure associated with ECD exposure in methamphetamine-intoxicated sheep.

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Law enforcement officers utilize electronic control devices (ECDs), such as the TASER X26 (TASER International, Inc., Scottsdale, AZ), to incapacitate actively resisting and combative subjects. These devices discharge an electrical current through two metal contacts on the front of the device or through two probes, connected to the device by insulated wire, which are fired by compressed nitrogen. The primary mechanism by which these devices operate is through the electrical capture (depolarization secondary to the induced electric fields) of peripheral motor neurons. The peripheral motor neurons are stimulated at a subtetanic rate (19 pulses per second in the TASER X26) leading to involuntary muscle contraction and therefore incapacitation. The devices likely have some direct muscle tissue stimulation in the area immediately adjacent to the contacts or probes and likely stimulate reflex arcs through afferent pathways.[1] The devices also cause pain through the stimulation of sensory neurons. The theoretical safety construct of these devices lies in the difference in the strength–duration curves for excitability between motor and sensory neurons and cardiac tissues, the latter requiring a higher strength, longer duration electrical stimulus to cause depolarization (or capture) than the former.[2]

Because of the prevalence of methamphetamine abuse worldwide, and because of the nature of methamphetamine intoxication, it is not uncommon for subjects in law enforcement encounters to be methamphetamine-intoxicated. Methamphetamine (or amphetamine) has been present in arrest-related death cases, some of which included an ECD in the uses of force.[3–6] While the effects of these devices have been studied in human volunteers, there has been no study on the effect of these devices in subjects intoxicated by methamphetamine (or amphetamine). The primary purpose of this study was to examine the cardiac effects of an ECD discharge in a methamphetamine intoxication model by studying the ability of the device to electrically capture the myocardium and to induce ventricular arrhythmias, as well as to affect postexposure vital signs. A secondary objective was to determine the metabolic effects of the device exposures in the context of methamphetamines.

METHODS

Study Design

This was a laboratory study using sheep. The Hennepin County Medical Center Institutional Animal Care and Use Committee approved this study, and the study was conducted at an animal laboratory at Hennepin County Medical Center (Minneapolis, MN).

Animal Subjects

Sixteen Dorset sheep (weight range = 26–78 kg) received intramuscular xylazine (0.01 mg/kg) sedation for physical control, followed by intravenous (IV) sodium pentothal (5 mg/kg) induction (once an IV line was established), endotracheal intubation or cricothyrotomy, and then 1.5% isoflurane maintenance anesthesia. Initial ventilator settings were at the discretion of the laboratory animal technician. These settings were adjusted, as needed, based on vital signs as determined by the technician. There was some time delay from the start of maintenance anesthesia to the beginning of the experiment (methamphetamine administration) as it took time to do the initial set-up procedures described below. The weight range in this study was due to an ordering error. The primary intent of the authors had been to study adult-sized animals. However, due to an ordering error, the initial group of animals was small-sized. Given the financial and personnel investment in the study, the authors decided to use the smaller animals and then do a later test with the planned adult-sized animals.

Study Protocol

Animals were positioned in the dorsal recumbent position with each limb restrained. After the establishment of an arterial line, continuous blood pressure, heart rhythm (one-lead), pulse oximetry, and capnography were monitored. Arterial blood sampling was performed at baseline, 30 minutes after the administration of the methamphetamine, and after each exposure from a TASER X26. The TASER X26 was modified to deliver variable duration exposures (as outlined below). The electrical output was not different from a standard TASER X26 as verified by oscilloscope prior to testing.

The sheep received 0.0 mg/kg (control animals, $n = 4$), 0.5 mg/kg ($n = 4$), 1.0 mg/kg ($n = 4$), or 1.5 mg/kg ($n = 4$) methamphetamine hydrochloride (as Desoxyn tablets, Ovation Pharmaceuticals [Lundbeck Inc., Deerfield, IL], dissolved in normal saline to 1 mg/mL) as a slow (5- to 10-second) IV bolus during continuous cardiac monitoring. Dosing was chosen sequentially; i.e., the first sheep was a control, the next sheep received 0.5 mg/kg, etc. Other authors have reported that the street dose of IV methamphetamine ranges from 0.1 to 0.8 mg/kg and that doses in this range result in immediate effects with peak intoxication ratings reported as early as 10 minutes and lasting about 2 hours.[7] Other authors have reported that street abusers usually inject over a period of 5–10 seconds.[8]

Measurements

Arterial blood was sampled for pH, lactate, potassium, bicarbonate, pCO_2, and pO_2. Blood samples were immediately run on the Abbott Point-of-Care i-STAT (East Windsor, NJ) device. The animals received the following ECD exposures 30 minutes after the administration of the drug, in sequence, with a minimum of 3 minutes of rest between exposures (up to 15 minutes if ventricular fibrillation occurred): 1) 5-second continuous exposure; b) 15-second intermittent exposure, delivered as 5 seconds on, 5 seconds off, 5 seconds on, 5 seconds off, 5 seconds on; 3) 30-second intermittent exposure, delivered as 15 seconds on, 10 seconds off, 15 seconds on; and 4) 40-second intermittent exposure, delivered as 20 seconds on, 10 seconds off, 20 seconds on. The device discharges for 5 seconds with a single trigger pull, but officers can hold down the trigger for longer exposures. Anecdotally, the longer exposures are less likely to be continuous, but rather intermittent as the officer assesses for effectiveness. Our timing was somewhat arbitrary, but was meant to capture both

longer duration and intermittent exposures. In a study by Bozeman et al.,[9] 55% of 1,201 field exposures were 5 seconds, 27% were 10 seconds, 12% were 15 seconds, and 7% were 20 seconds or more, but the study did not break down whether these were continuous or intermittent. Darts were inserted to a depth of 9 mm, at the sternal notch, determined by palpation, and the cardiac apex (left chest), determined by ultrasound. This was a similar positioning as used by other authors and was chosen to produce a worst-case transcardiac vector.[10] Skin-to-heart (apex) depths ranged from 20 to 46 mm.

Cardiac motion was determined by thoracotomy (smaller animals ≤ 32 kg, with an additional 15-second ECD exposure after the assigned sequence of exposures was completed) or echocardiography (larger animals > 68 kg). In the smaller animals, the ultrasonographer was unable to obtain reliable views secondary to narrow rib spacing, sharp chest angles, and motion, so this technique was abandoned, and thoracotomy, after the last sequential exposure, was used instead. Electrical capture was determined by an abrupt change in heart motion with tachycardia with the start of the discharge. The investigator was not blinded and had to watch closely and be aware when the electrical discharge was begun. All animals were euthanized with injection of potassium chloride at the conclusion of the protocol.

Data Analysis

Data were placed into an Excel spreadsheet (Microsoft Corp, Redmond, WA) and then exported into STATA 10.0 (StataCorp, College Station, TX) for analysis. Data were analyzed using descriptive statistics. Vital signs were compared using Wilcoxon rank sum tests. The rate at which ventricular fibrillation will be induced in sheep using an ECD is unknown. If there is a 50% increase in the number of sheep that will experience ventricular fibrillation after a standard ECD exposure in the context of methamphetamine intoxication, we will be 90% likely to detect it using 12 sheep (power analysis with alpha of 0.05, power of 90%). This number of sheep will allow us to detect only large changes in the threshold for ventricular fibrillation in the context of methamphetamine intoxication.

RESULTS

The surface electrocardiogram results are shown in Table 1, and the vital sign results are shown in Tables 2A and 2B. All animals given methamphetamine demonstrated signs of methamphetamine toxicity with tachycardia, hypertension, and atrial and ventricular ectopy (some including runs of nonsustained ventricular tachycardia) in the 30-minute period immediately after administration of the drug.

One smaller animal (animal 8, 30 kg, at 1.0 mg/kg methamphetamine) had a supraventricular tachycardia (SVT) shortly (7 minutes) after methamphetamine administration requiring cardioversion. One animal (animal 11, 78 kg, at 1.5 mg/kg methamphetamine) had apparent seizure-like activity shortly (4 minutes) after methamphetamine administration that resolved spontaneously.

At 30 minutes, most methamphetamine-intoxicated animals had continued hypertension and tachycardia. The median systolic pressure at 30 minutes was 105 mm Hg for the methamphetamine-exposed sheep versus 75 mm Hg for the control sheep ($p = 0.002$). The median heart rate for the methamphetamine-exposed sheep was 152 beats/min versus 97 for the control sheep ($p < 0.001$).

Smaller animals ($n = 8$, ≤ 32 kg, mean = 29.4 kg) had supraventricular dysrhythmias immediately after the ECD exposures (including SVTs and frequent premature atrial contractions). Larger animals ($n = 8$, > 68 kg, mean = 72.4 kg) had only sinus tachycardia after the ECD exposures.

Table 1
Surface Electrocardiographic Results

Methamphetamine Dose (mg/kg)	Weight (kg)	Skin to Heart Distance (cm)	Rhythms After ECD exposure
Control	31	23	PACs, single PVC after 30-second exposure
Control	30	21	Sinus tachycardia
Control	72	30	Sinus tachycardia
Control	74	34	Sinus tachycardia
0.5	27	22	Sinus tachycardia
0.5	27	21	Sinus tachycardia
0.5	68	35	Sinus tachycardia
0.5	78	34	Sinus tachycardia
1.0	32	21	Ventricular ectopy, nonsustained Vtach after 30- and 40-second exposures
1.0	32	21	SVT after 30-second exposure
1.0	68	29.5	Sinus tachycardia
1.0	68	31	Sinus tachycardia
1.5	26	20	SVT after 30-second exposure
1.5	30	22	Sinus tachycardia
1.5	78	46	Sinus tachycardia
1.5	73	38	Sinus tachycardia

ECD = electronic control device; PAC = premature atrial contraction; PVC = premature ventricular contraction; SVT = supraventricular tachycardia; Vtach = ventricular tachycardia.

Table 2A
Vital Signs*

Animal Weight (kg)	Methamphetamine Dose (mg/kg)	Baseline Vital Signs	5 Minutes After Methamphetamine	30 Minutes After Methamphetamine	1 Minute After 5-Second Exposure†	1 Minute After 15-Second Exposure†	1 Minute After 30-Second Exposure†	1 Minute After 40-Second Exposure
31	0	68, 97/71	NA	NA	102, 75/56	163, 78/58	119, 70/47	155, 58/42
31	0	96, 69/43	NA	NA	122, 62/36	82, 57/34	92, 54/33	83, 59/34
72	0	112, 70/37	NA	NA	112, 70/37	80, 58/41	150, 103/81	136, 50/38
74	0	76, 82/52	NA	NA	77, 54/28	77, 47/28	72, 74/51	164, 82/44
27	0.5	98, 81/52	89, 99/52	115, 99/42	110, 84/M	104, 117/M	99, 60/M	95, 100/51
27	0.5	75, 77/44	84, 187/132	147, 106/67	144, 68/M	140, 81/M	130, 80/M	138, 58/36
68	0.5	77, 83/55	142, 90/74	95, 215/162	89, 188/146	145, 64/51	130, 80/58	131, 92/63
78	0.5	111, 91/45	121, 237/175	109, 95/59	105, 92/59	83, 54/38	95, 72/56	83, 78/60
32	1.0	103, 70/44	161, 171/129	158, 126/89	155,130/97	159, 119/87	186, 77/43	191, 49/30
32	1.0	85, 86/48	94, 190/137	134, 119/78	109, 55/32	124, 87/49	151, 55/33	142, 76/45
68	1.0	100, 117/95	138, 276/208	174, 120/88	196, 85/59	100, 80/60	85, 126/80	84, 122/86
68	1.0	68, 66/45	131, 196/190	148, 130/108	140, 115/92	129, 153/119	133, 114/72	110, 93/57
30	1.5	80, 70/43	133, 213/159	163, 141/97	167, 178/133	163, 110/76	150, 107/62	157, 74/52
26	1.5	86, 84/56	184, 97/30	160, 96/27	155, 124/M	159, 85/M	134, 60/M	148, 100/47
78	1.5	91, 108/78	113, 223/173	197, 139/111	105, 92/59	83, 54/38	95, 72/56	83, 78/60
73	1.5	70, 59/37	130, 185/96	220, 125/116	190, 180/29	179, 146/101	112, 130/126	103, 55/46

NA = not applicable (see text).
*Heart rate (beats/min); blood pressure (mm Hg).
†M = missing data.

One of the smaller animals had frequent episodes of ventricular ectopy after two ECD exposures, including runs of delayed-onset (2–5 minutes after the exposure), nonsustained, six- to eight-beat unifocal and multifocal ventricular tachycardias that spontaneously resolved. This animal had significant ventricular ectopy after the methamphetamine administration and prior to the exposures as well. Thoracotomy performed on three smaller animals demonstrated cardiac capture during ECD exposure consistent with previous animal studies.[10–12] It was noted that capture seemed to depend on the respiratory cycle. Capture seemed to occur when the heart was most anterior in the chest. While not measured, the appearance of the heart was similar to ventricular fibrillation or even cardiac standstill, noted by other authors.[11]

Table 2B
Summary Vital Signs by Exposure Duration and Methamphetamine Dose

Methamphetamine Dose (mg/kg)	Exposure Time (Seconds)	Heart Rate (Beats/Min)	Comparison to Control Group	Systolic Blood Pressure (mm Hg)	Comparison to Control Group
0	Baseline	104 (68–112)		76 (69–97)	
0	Postmethamphetamine	NA		NA	
0	5-second exposure	112 (102–122)		68.5 (57–78)	
0	15-second exposure	122.5 (82–163)		67.5 (57–78)	
0	30-second exposure	105.5 (92–119)		62 (54–70)	
0	40-second exposure	119 (83–155)		58.5 (58–59)	
0.5	Baseline	87.5 (75–111)	0.201	82 (77–91)	0.754
0.5	Postmethamphetamine	100, 84–142		143 (90–237)	
0.5	5-second exposure	127 (110–144)	0.233	76 (68–84)	0.843
0.5	15-second exposure	115.5 (91–140)	0.532	75.5 (70–81)	0.456
0.5	30-second exposure	113.5 (97–130)	0.350	76, (72–80)	0.537
0.5	40-second exposure	116.5 (95–138)	0.435	69.5 (58–81)	0.438
1.0	Baseline	92.5 (68–103)	0.629	78 (66–117)	0.719
1.0	Postmethamphetamine	134.5 (94–161)	0.014	193 (171–276)	<0.001
1.0	5-second exposure	155 (109–165)	<0.001	70 (55–130)	0.249
1.0	15-second exposure	162 (159–165)	<0.001	102 (55–126)	0.035
1.0	30-second exposure	159 (85–186)	<0.001	71.5 (55–126)	0.638
1.0	40-second exposure	171 (142–191)	<0.001	63 (49–76)	0.751
1.5	Baseline	83 (70–91)		77 (59–108)	
1.5	Postmethamphetamine	131.5 (113–184)	<0.001	199 (97–223)	<0.001
1.5	5-second exposure	167 (155–195)	<0.001	124 (85–110)	0.020
1.5	15-second exposure	161 (159–163)	<0.001	97.5 (85–110)	0.046
1.5	30-second exposure	142 (134–150)	<0.001	83.5 (60–107)	0.067
1.5	40-second exposure	168 (80–219)	<0.001	74 (55–100)	0.152

Heart rate and systolic blood pressure values are median (range). Comparisons found using Wilcoxon rank sum.

Table 3
Echocardiography Results in Larger Animals (Heart Rates, Beats/Min)

Methamphetamine Dose (mg/kg)	Exposure			
	5 Seconds	15 Seconds	30 Seconds	40 Seconds
0	190 (55, 112)*	191 (73, 80)	273 (79, 105)	NO
0	NO	NO	240 (72, 99)	188 (84, 93)
0.5	NO	NO	NO	NO
0.5	78 (105, 95)	NO	120 (95, 93)	111 (83, 112)
1	NO	105 (116, 170)	92 (110, 85)	102 (168, 84)
1	133 (148, 140)	94 (212, 129)	66 (129, 78)	91 (134, 108)
1.5	102 (197, 195)	207 (200, 200)	171 (135, 145)	NO
1.5	NO	125 (179, 130)	111 (173, 112)	103 (105, 115)

NO = not obtained.
*The numbers in parentheses represent the monitor heart rate before and after the exposure, respectively.

The echocardiographic results for the larger animals are shown in Table 3. In the larger animals, none of the methamphetamine-intoxicated animals demonstrated capture (although an occasional extrasystole was noted in a few animals). Heart rates could be determined by echocardiography but not specific rhythm due to difficulty with resolution in the M-mode with the muscle contraction. The animals tended to show a bradycardic response during exposure (particularly initially) and then tachycardia once the exposure ceased. Two of the larger control sheep showed evidence of capture by echocardiography similar to the smaller animals. No ventricular fibrillation occurred after the exposures in any of the animals (including the smaller animals). There was immediate reversion to sinus tachycardia in the larger animals when the application was stopped. Note that no heart rate results were obtained in one animal (due to ultrasonographer error), but there was no visual evidence of capture. The other heart rates not obtained were due to similar errors.

The laboratory results are presented in Table 4. These results are similar to those of other animal studies with prolonged exposures.[13]

DISCUSSION

The results of this study may be important in the debate of the use of ECDs in the context of drug intoxication, specifically methamphetamine intoxication. Methamphetamines are synthetic amines that have significant

Table 4
Laboratory Results

Exposure Time	Methamphetamine Dose (mg/kg)	pH	pCO2 (mm Hg)	Bicarbonate (mmol/L)	Potassium (meQ/L)	Lactate (mg/L)
Baseline	0	7.61, 7.49–7.68	33.8, 29.4–39.7	32.5, 30.1–37.1	3.7, 3.1–4.5	1.16, 0.82–2.92
Baseline	0.5	7.64, 7.60–7.68	32.3, 30.8–33.3	34.9, 33.6–36.1	3.5, 3.3–3.7	0.45, 0.41–0.48
Baseline	1.0	7.64, 7.60–7.67	30.7, 29.5–32.1	33.0, 26.7–33.4	3.7, 3.4–3.9	0.30, 0.24–0.78
Baseline	1.5	7.72, 7.41–7.84	25.9, 23.3–51.6	33.8, 32.9–39.9	3.4, 3.0–3.6	0.43, 0.36–1.52
30 minutes postmethamphetamine	0.5	7.56, 7.51–7.62	38.7, 33.3–44.8	35.2, 32.9–37.6	3.4, 3.0–3.9	1.64, 0.44–5.09
30 minutes postmethamphetamine	1.0	7.43, 7.35–7.55	49.3, 43.3–55.7	32.3, 29.9–33.1	3.6, 3.4–3.9	0.43, 0.36–0.89
30 minutes post methamphetamine	1.5	7.63, 7.55–7.72	32.8, 30.2–35.4	35.0, 31.0–38.9	3.1, 2.9–3.2	0.44, 0.39–1.02
5 seconds	0	7.66, 7.64–7.67	30.9, 29.5–32.2	34.5, 31.7–37.3	4.0, 3.5–4.5	3.49, 3.0–3.99
5 seconds	0.5	7.56, 7.51–7.62	37.6, 41.4–49.8	33.6, 25.5–36	3.4, 3.2–3.5	0.66, 0.44–0.87
5 seconds	1.0	7.44, 7.42–7.64	42.3, 33–52.2	34.2, 28.6–35.9	3.8, 3.6–4.3	3.81, 0.91–6.70
5 seconds	1.5	7.52, 7.40–7.63	37.6, 36.2–58.6	36, 30.9–38	3.6, 3.4–4.3	0.77, 0.73–1.51
15 seconds	0	7.56, 7.47–7.65	37.7, 28.5–37.9	31.3, 27.5–33.8	4.6, 3.5–5.1	6.56, 2.29–6.98
15 seconds	0.5	7.42, 7.35–7.55	46.2, 41.4–49.8	32.1, 30.0–36.2	4.0, 3.2–4.5	5.08, 3.68–5.19
15 seconds	1.0	7.43, 7.35–7.44	48.1, 45.4–61.9	32.8, 30.9–34.4	4.3, 4.2–4.5	4.50, 2.51–6.62
15 seconds	1.5	7.58, 7.46–7.70	37.9, 32.5–43.2	35.2, 30.6–39.7	3.5, 3.1–3.9	2.20, 2.13–2.26
30 seconds	0	7.40, 7.32–7.49	49.5, 43–55.9	30.9, 28.8–32.9	4.4, 3.9–4.9	9.22, 5.35–11.47
30 seconds	0.5	7.43, 7.35–7.51	45.1, 42–48.1	30.0, 26.5–33.5	4.0, 3.5–4.5	5.21, 3.59–6.83
30 seconds	1.0	7.43, 7.32–7.52	46.2, 38.1–61.9	31.1, 30.6–31.9	4.8, 4.4–5.0	4.62, 4.46–7.68
30 seconds	1.5	7.45, 7.27–7.63	47.9, 28.2–56.3	32.7, 31.1–34.2	4.1, 4.0–4.2	3.77, 3.76–3.79
40 seconds	0	7.30, 7.22–7.38	54.2, 52.6–55.7	27, 22.8–31.1	4.45, 4.4–4.5	8.49, 4.46–12.52
40 seconds	0.5	7.49, 7.41–7.56	38.1, 28.8–47.3	27.9, 25.8–29.9	4.3, 4.1–4.5	5.22, 3.59–6.83
40 seconds	1.0	7.33, 7.22–7.35	58.2, 53–73.2	30.2, 29.2–30.5	4.8, 4.5–4.8	7.57, 7.3–9.01
40 seconds	1.5	7.31, 7.19–7.84	56.3, 23.3–84.9	28.2, 24.7–39.9	4.3, 3.4–5.1	5.28, 3.78–11.86

Values are expressed as median, range.

sympathomimetic effects. Common street names for methamphetamine include speed, crank, ice, crystal, and go.[14] While the target effects in abusers are euphoria, motivation, and extroversion, these drugs can have significant cardiovascular, cerebral, metabolic, and psychiatric effects and can predispose the abuser to sudden death.[14–16]

According to the Drug Abuse Warning Network statistics for 2006, there were 107,575 emergency department visits for amphetamines, comprising 11% of the visits for illicit drug abuse (cocaine accounted for 57% of the visits).[17] An Australian autopsy review showed an increase in amphetamine-positive deaths from 27 cases in 2001 to 46 cases in 2005.[15] A recent study estimated that nearly 25 million people worldwide have used amphetamines in the past 12 months, making it the most widely used illicit drug after cannabis.[18] In a study by Cartier et al.,[19] use of the methamphetamine was significantly predictive of self-reported violent criminal behavior. Intoxication with methamphetamine is often accompanied by disinhibition, agitation, and paranoia and these effects may lead to violent behavior. In a New Zealand study of law enforcement officer perceptions, 40% of those reporting a change in the level of violence committed by methamphetamine abusers reported noticing ''more serious violence.''[20]

Law enforcement officers have contact with methamphetamine-intoxicated subjects who may require physical control and arrest. In addition, some subjects may ingest methamphetamine at the time of arrest to avoid detection of the drug, resulting in massive overdoses.[14] ECDs are commonly used by law enforcement officers in the United States and, increasingly, around the world, to control resisting and violent subjects.

A prior study by Lakkireddy et al.[12] examined the effects of acute cocaine intoxication on the ventricular fibrillation threshold for the TASER X26 in a swine model. In this study, the authors reported that cocaine increased the threshold from 1.5 to 2 times (also increasing the capture threshold). The authors are not aware of any study that has examined the effects of methamphetamine. We chose to study sheep because much of what is known about the effects of drugs of abuse on the heart has been done in sheep.[21] Furthermore, swine are known to have lower fibrillation thresholds than most other animals on a kilogram-for-kilogram basis. A study by Dalziel and Lee[22] of regressing fibrillation thresholds found swine to be least associated with the regression of reported fibrillation thresholds in other animals including dogs, guinea pigs, cows, sheep, and humans.

In the larger animals, only the control animals showed evidence of capture. The methamphetamine-intoxicated animals did not show capture, but rather an intraexposure slowing in many of the animals. The control animals had preexposure heart rates that were less than 100 (and were mildly hypotensive, probably an anesthesia affect). The methamphetamine-intoxicated animals had high preexposure heart rates. It is possible that the tachycardia itself made the myocardium more resistant to electrical capture. Pacing thresholds can be increased with higher heart rates.[23] A less likely alternative is that methamphetamine made the myocardium less likely to capture. In the study by Lakkireddy et al., it was felt that the sodium blockade effect of cocaine made the myocardium more resistant to electrically induced ventricular fibrillation.[12]

Our results suggest that there is a weight-based effect with smaller weight animals having more cardiac dysrhythmias after exposure. Since dart-to-heart (apex) distance was correlated with weight (mean in the smaller animals was 21.1 mm and in the larger animals 35.6 mm), this would be consistent with the work of Wu et al.[24] that showed a depth effect. In our larger animals (>68 kg) with methamphetamine intoxication, we found neither capture nor ventricular fibrillation.

While prior work in animals has shown cardiac capture with ECDs,[10–12] in work in humans with mean body mass indexes (BMIs) of 29.8 and 27.8 kg/m^2, there was no evidence of capture.[25,26] These BMIs match the BMIs of arrest-related death subjects. In Stratton et al.[3] the mean BMI was 30 kg/m^2. In Strote and Hutson[4] the mean BMI was 30.8 kg/m^2. In Swerdlow et al.,[6] the mean BMI was 30 kg/m^2.

The discrepancy between the animal findings and human findings may be weight-related (although we did have capture in some large control animals), species-related (differences in susceptibility, chest shape and charge distribution, cardiac anatomy), related to experimental conditions (anesthetized and mechanically ventilated sheep vs. awake humans), or related to very specific positioning of the probes. The human experimental findings are supported by field use studies (3,188 uses in five studies) that have not demonstrated sudden ventricular dysrhythmias associated with the use of the devices.[27–31] In the study by Bozeman et al.,[29] 22% of the field uses included chest deployments with probes in a possible transcardiac vector. The human experimental findings are also supported by studies of arrest-related death temporal to ECD use that have reported a low incidence of ventricular fibrillation.[3,6] However, because these field studies did not measure cardiac effects during exposure (for obvious reasons) and did not detail subject behavior (e.g., syncope) during exposure, it is still possible that some subjects in these studies did have capture during the exposure. They just did not have ventricular fibrillation.

The discrepancy between animal and human studies (prospective and retrospective) certainly merits further investigation and may suggest caution in the use of these devices in low-weight individuals and children without a greater legal justification and may suggest the need for officers to always reassess the subject's resistance in longer exposures. However, based on our current study, while we did get cardiac capture in the sheep model, methamphetamine intoxication did not seem to be a worsening factor with regard to cardiac safety in the larger animals. These results suggest that in suspects of adult weight, methamphetamine intoxication does not preclude the use of an ECD.

LIMITATIONS

The original design of the study was not to do a weight-based comparison. The authors had planned to only study animals approximating the size of human

adults but, as explained under Methods, an ordering error by the laboratory personnel caused the authors to include these smaller animals. In hindsight, it was an important result because there was a difference in the groups. However, our study was not designed to examine this difference.

The relevance of animal models to human subjects is always a limitation. As has been demonstrated with previous ECD studies, animal studies have sometimes been contradicted by human studies. This does not mean that these animal studies are irrelevant, but means their results must be interpreted with caution. For some questions, particularly with regard to intoxication with illegal drugs, such as cocaine and methamphetamine, animal models may be the only ethical study model.

Our study was also limited in that our results only apply to single device exposures. The potential for additive charge in closely spaced multiple exposures needs to be kept in mind and should be studied. This probably needs additional study, especially to see if this effect is seen in humans and whether other use-of-force options may cause similar changes.

CONCLUSIONS

Methamphetamine intoxication was associated with both supraventricular and ventricular irritability. In small sheep, electronic control device exposures did exacerbate this irritability, but this did not occur in the larger sheep. There was no ventricular fibrillation after electronic control device exposure.

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