

TASER X26 Discharges in Swine Produce Potentially Fatal Ventricular Arrhythmias

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Abstract

Objectives: Data from the authors and others suggest that TASER X26 stun devices can acutely alter cardiac function in swine. The authors hypothesized that TASER discharges degrade cardiac performance through a mechanism not involving concurrent acidosis.

Methods: Using an Institutional Animal Care and Use Committee (IACUC)-approved protocol, Yorkshire pigs (25–71 kg) were anesthetized, paralyzed with succinylcholine (SCh; 2 mg/kg), and then exposed to two 40-second discharges from a TASER X26 with a transcardiac vector. Vital signs, blood chemistry, and electrolyte levels were obtained before exposure and periodically for 48 hours postdischarge. Electrocardiograms and echocardiography (echo) were performed before, during, and after the discharges. p-Values < 0.05 were considered significant.

Results: Electrocardiograms were unreadable during the discharges due to electrical interference, but echo images showed unmistakably that cardiac rhythm was captured immediately at a rate of 301 ± 18 beats/min ($n = 8$) in all animals tested. Capture continued for the duration of the discharge and in one animal degenerated into fatal ventricular fibrillation (VF). In the remaining animals, ventricular tachycardia (VT) occurred postdischarge for 1–17 seconds, whereupon sinus rhythm was regained spontaneously. Blood chemistry values and vital signs were minimally altered postdischarge and no significant acidosis was seen.

Conclusions: Extreme acid–base disturbances usually seen after lengthy TASER discharges were absent with SCh, but TASER X26 discharges immediately and invariably produced myocardial capture. This usually reverted spontaneously to sinus rhythm postdischarge, but fatal VF was seen in one animal. Thus, in the absence of systemic acidosis, lengthy transcardiac TASER X26 discharges (2×40 seconds) captured myocardial rhythm, potentially resulting in VT or VF in swine.

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Stun guns or electromuscular incapacitation devices (EIDs) generate between 20,000 and 900,000 V and can be discharged for more than 10 minutes continuously. In the United States alone, more than 200,000 individuals have been exposed to

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discharges from the most common type of EID, the TASER (TASER International, Scottsdale, AZ). Worldwide, TASERs are currently used by more than 11,000 law enforcement agencies and owned by more than 115,000 private citizens.¹ Unfortunately, there have been more than 250 deaths in the past several years in the United States and Canada that have been temporally associated with the use of TASERs.^{2–4} Most of these deaths have been attributed to stimulant drug overdoses and intense agitation resulting in hyperthermia, extreme acidosis, and cardiac failure,⁵ but in more than two dozen deaths, TASER shocks have been cited as being contributory.⁴ With increasing use, the numbers of deaths that are temporally associated with exposure to TASER discharges are increasing at an alarming rate. As a result, there is great interest in EID safety and potential complications associated with their use, especially their ability to induce fatal ventricular dysrhythmia.^{6–9}

Early (pre-1999) studies of stun devices examined much less powerful first or second generation devices and their conclusions may not be relevant to the powerful present-day EIDs.¹⁰⁻¹² The recent peer-reviewed literature on fourth-generation EIDs (such as the TASER X26) is still emerging and the results are conflicting. Some studies in swine, utilizing a TASER-like device, showed no evidence of acute dysrhythmia and a large safety margin for the development of ventricular dysrhythmia.^{7,13} Similarly, neither acidosis nor hyperkalemia has been observed in healthy human volunteers exposed to brief (2–5 seconds) TASER X26 discharges.¹⁴⁻¹⁶ However, in swine models, TASER exposure has been shown to cause significant acidosis^{17,18} and the potential for fatal dysrhythmia.¹⁸⁻²⁰ Such conflicting results make it difficult to evaluate the hazards associated with the use of these devices.

Adding to this uncertainty, there is disagreement about the electrical output from these devices (especially under resistive load) about what load is representative of human tissues and the lack of a standard model for study *in vitro* or *in vivo*.^{6-8,21} Under no-load conditions, the TASER X26 delivers DC pulses at 50 kV, with pulse durations of 140 μ seconds, at 19 pulses/second, and power of 0.36 J/pulse.¹ *In vivo*, this type of discharge causes severe pain, strong muscle contractions, and incapacitation of volitional movement. The manufacturer also states that, under a 400 Ω load, this device produces 90- μ second pulses at 1200 V with a peak current of 3 A and 2.1 mA time-averaged current. However, Webster²² and others^{21,23} assert that higher voltages and currents that exceed the ventricular fibrillation (VF) threshold may be produced. Such disparate opinions make the safety profile and physiologic effects of EID current exposure difficult to deduce reliably.

In an attempt to reconcile some of the conflicting information about the physiologic effects of TASER discharges *in vivo*, we have studied discharges in a well-characterized swine model. Our working hypothesis was that subcutaneous, transcardiac discharges from the TASER X26 could capture cardiac rhythm and produce fatal ventricular dysrhythmias, independent of systemic acidosis.

METHODS

Study Design

This was a laboratory investigation using a swine model. The primary goal of the investigation was to determine if such discharges could capture cardiac rhythm and produce fatal ventricular dysrhythmias, independent of metabolic or respiratory acidosis. The project was reviewed and approved by the Institutional Animal Care and Use Committee (IACUC) for the Hektoen Institute. The care and handling of animals were in accord with National Institutes of Health (NIH) guidelines and American Veterinary Medical Association (AVMA) standards for ethical research.

Animal Subjects

The musculoskeletal response to TASER discharges in swine and humans is very similar.^{7,17} This seems rea-

sonable, since their integumentary, musculoskeletal, and cardiovascular systems display numerous anatomic and physiologic similarities. The ratios of heart size to body mass, coronary arterial distribution, and susceptibility to VF are similar.²⁴ However, some important differences in the composition of the cardiac conduction system exist^{24,25} that warrant attention when comparing arrhythmogenic stimuli in swine and humans. There are also differences in thoracic geometry and the rotation of the heart within the thorax that may affect the thresholds for arrhythmia or VF in swine. Despite these differences, the swine has proven to be an effective model for ventricular arrhythmia and pacemaker testing²⁶⁻²⁹ and is the primary animal model for the study of EIDs, such as TASERs.^{7,13,17-20,22,30,31}

Before use, 3- to 6-month-old male Yorkshire pigs (Michael Fanning Farms, Howe, IN) weighing between 22 and 71 kg (mean \pm SD for all animals; 37.6 \pm 14.8 kg; 48–156 lb) were acclimatized for at least 1 week in the animal facility. Animals were sedated with IM ketamine (Ketaset; Fort Dodge Animal Health, Fort Dodge, IA) and xylazine (Anased; Lloyd, Shenandoah, IA) and respiratory secretions were inhibited using glycopyrrolate (Robinul; Fort Dodge Animal Health, Fort Dodge, IA) in the ratio 30/3/0.01 mg/kg. Animals in dorsal recumbence were intubated and breathing controlled with a respirator as described previously.¹⁸ Three types of controls were used: intraanimal internal (0 time), paralyzed control (Sch-CT), and untreated (Sham-CT) animals.

In the experimental group (Sch-TASER), the effects of TASER X26 discharges applied with a specific transcardiac vector in the presence of succinylcholine (Sch) were determined. The Sch-TASER group was paralyzed with Sch and received two 40-second TASER discharges, and then six of these animals were monitored for 48 hours. The masses of the animals in each group were as follows: Sch-TASER (25, 25, 28, 46, 58, and 71 kg), Sch-CT (22, 35, and 47 kg), and Sham-CT (22, 27, 31, 43, and 46 kg). The average mass for each animal in the combined control groups was 34.1 \pm 10.3 kg (48–103 lb), which was not significantly different from that of the experimental group. After 48 hours of monitoring, two surviving animals from the Sch-TASER group (the 58- and 71-kg animals) were again injected with Sch and then exposed to a second set of two 40-second discharges. Cardiac activity was assessed by echo and 60 minutes of further monitoring was performed. Immediately after this monitoring, thoracotomy was performed on these animals (see Study Protocol).

The Sch-CT and Sham-CT groups were composed of three and five animals, respectively. Some of the values for the Sch-TASER group have an $n = 6$ (mean of animal mass; 24- and 48-hour blood values, electrocardiograms (ECGs), and vitals; echo EF data in Table 1), others have an $n = 8$ (pre-discharge to 60 minute blood values, ECG, qualitative echo data, and vitals; heart rates pre-, mid-, and postdischarge), and some have an $n = 2$ (thoracotomized animals including ECGs and qualitative observations).

Table 1
EF Calculated from Left Ventricular Diameter

	SCh-TASER	SCh-CT
Predischarge	0.44 ± 0.02	0.44 ± 0.02
Middischarge	0.09 ± 0.03*	0.50 ± 0.03
Postdischarge	0.42 ± 0.11†	0.48 ± 0.01

The table shows the means ± SD for LV diameter at each stage of the experiment.
SCh-TASER = succinylcholine + TASER discharges; SCh-CT = succinylcholine only control group.
* p < 0.001 vs. pre- and postdischarge within the SCh-TASER group and middischarge between groups.
† Excludes one animal in VF.

Study Protocol

An unmodified, police-issue TASER X26 device powered by TASER lithium 6-V digital power magazines was used. Discharges were delivered by a member of the local law enforcement community trained in TASER use.

TASER Discharge. The barbed darts were placed along a transcardiac vector (see Figure 1). Just before TASER discharge, 2 mg/kg SCh (Sandoz, Broomfield, CO) was given intravenously. When fasciculations ended, the TASER was discharged in two separate 40-second intervals for a total of 80 seconds, during which time the ventilator was shut off. Ventilated breaths were administered during the 10- to 15-second pause between the 40-second discharges to prevent respiratory acidosis.

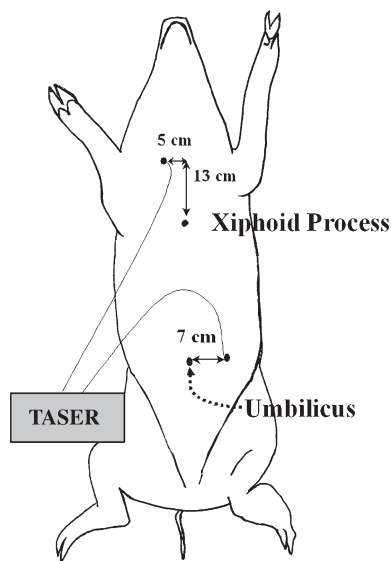


Figure 1. Dart placement on the ventral body surface of the swine. The superior dart was placed 13 cm superior to the xiphoid process and 5 cm to the right of the midsternal line. The inferior dart was placed 7 cm to the left of the umbilicus. Darts were inserted perpendicular to the skin to their maximum depth (12 mm) such that the dart tip was located in subcutaneous tissue and the current path between the darts was transcardiac.

Thoracotomy. As mentioned previously, left anterior thoracotomies were performed on two animals (58 and 71 kg) under inhaled 1.5%–2% isoflurane anesthesia. SCh was administered and the beating myocardium was then directly observed during a third set (2 × 40 seconds) of TASER discharges. Upon completion of this procedure, these anesthetized animals were euthanized.

Cardiac Rhythm and Echocardiography. Cardiac rhythm was monitored continuously using a five-lead surface ECG (Datex Instruments, Helsinki, Finland). Echocardiography (echo) was performed using a LOG-IQ 7 (GE Medical, Milwaukee, WI) with a 3-MHz transducer. ECGs and echo were performed on animals in all three groups.

Measurements

Ejection Fraction (EF). The method chosen for EF measurement was constrained by the experimental conditions and the model. The multiple, standard echo views that are used in humans to calculate cardiac output or the multidimensional views for EF could not be obtained in swine under the conditions used here. Differences in thoracic anatomy, such as the closeness of the rib spacing, the prominence of the sternum, and the rotation of the long axis of the heart to a more antero-posterior orientation in swine,^{26,32} made it difficult to obtain more than one unobstructed view of the left ventricle (LV). In addition, the brief time period available during the discharges (80 seconds) severely limited our ability to reposition the transducer and to capture multiple views reproducibly during the discharges. Thus, EF estimations were based on the diameter of paired (from the same heartbeat) circular cross-sectional profiles of the LV at end-systole and end-diastole.

Digital video was captured and frame grabs obtained using Studio QuickStart (v 9.3, Pinnacle Systems, Mountain View, CA). Left ventricular area was quantified from frame grabs using UTHSCSA ImageTool v3.00 (UTHSCSA, San Antonio, TX). Each value was calculated from triplicate measurements made on each frame grab for each animal in the SCh-TASER and SCh-CT groups. Images of the LV predischarge, middischarge, and postdischarge were analyzed and EF was calculated based on the formula $EF = (EDD^2 - ESD^2)/EDD^2$, where EDD is end diastolic diameter and ESD is end systolic diameter.

Blood Samples and Analysis. At eight time points (predischarge [Time 0], 5, 10, 15, 30, and 60 minutes and 24 and 48 hours postdischarge), central venous blood was drawn from the precaval venous complex (the confluence of the precaval vein with the cephalic, axillary, costocervical, and jugular veins),²⁴ and vital signs (tissue oxygen saturation, heart rate, and blood pressure) were taken. Blood pressure was monitored using a cuff. Central catheters were avoided to preclude the possibility that they might provide an artificial low-resistance path for current to penetrate into deeper tissues. Blood was tested using an iSTAT analyzer (Abbott Labs, Abbott Park, IL), and values were compared among all three animal groups. Animals

were humanely euthanized according to AVMA standards after the 48-hour data point.

Data Analysis

Paired t-tests or one-way analysis of variance (ANOVA) with Tukey multiple comparison tests were used to compare parametric data, and trends were analyzed using multiple regression analyses with InStat or Prism software (GraphPad Software, San Diego, CA).

RESULTS

Heart rate and blood pressure in the SCh-TASER group were similar to both control groups at all time points. One animal in the SCh-TASER group (28 kg) died from VF. In this animal, echocardiography showed a rapid cardiac rhythm and significant decline in systolic function that was consistent with ventricular tachycardia (VT) or ventricular flutter. When the discharge ceased, VF was noted on echo and confirmed by ECG (Figure 2). Two other animals showed VT/ventricular flutter on ECG subsequent to TASER discharge, which reverted spontaneously to sinus rhythm 10 or 17 seconds after cessation of the discharge, respectively.

Echocardiography and Direct Visualization

By echocardiography, all SCh-TASER animals showed rapid cardiac rhythm and significant decline in systolic function that was consistent with VT/ventricular flutter (301 ± 18 beats/min; $n = 8$) during discharges. This was consistent with the rate of 324 ± 66 beats/min reported by Nanthakumar et al.²⁰ Blood pressure dropped precipitously during the discharges. VT or ventricular

flutter occurred immediately upon starting the discharges and continued until the discharge terminated (Figure 3). This is seen to best advantage in the edited video clip associated with Figure 3 (available as an online data supplement at: http://www.aemj.org/taser_echo_video.html). For all SCh-TASER animals, EF decreased significantly from a baseline average of 44% to 9% at middischarge ($p < 0.001$) regardless of body mass (Table 1). In each of the five surviving animals, sinus rhythm was regained postdischarge and sinus tachycardia commenced within 1 minute. No changes in EF were seen in SCh-CT animals.

Similar results were obtained in animals that underwent thoracotomy. Before TASER discharge, with the heart exposed via left anterolateral thoracotomy, normal sinus rhythm was directly visualized and confirmed by ECG. When the TASER discharge started, direct visualization showed that sinus rhythm was immediately (within 1 second) disrupted. The rhythm observed was consistent with VT or ventricular flutter with rapid response. At the termination of the TASER discharge, sinus rhythm was regained, and shortly after this, sinus tachycardia ensued. Although VF was seen in one 28-kg animal, multiple regression analyses showed no significant correlation ($p = 0.06$) with mass, probably because there were two smaller animals that did not show VF under identical conditions.

Troponin-I (Figure 4) showed a minor increase in the SCh-TASER animals ($n = 6$) at 24 hours postdischarge (0.020 ± 0.010 ng/mL) but this was not significant compared to baseline values (0.000 ± 0.002) or to SCh-CT animals (one-way ANOVA, $p > 0.05$).

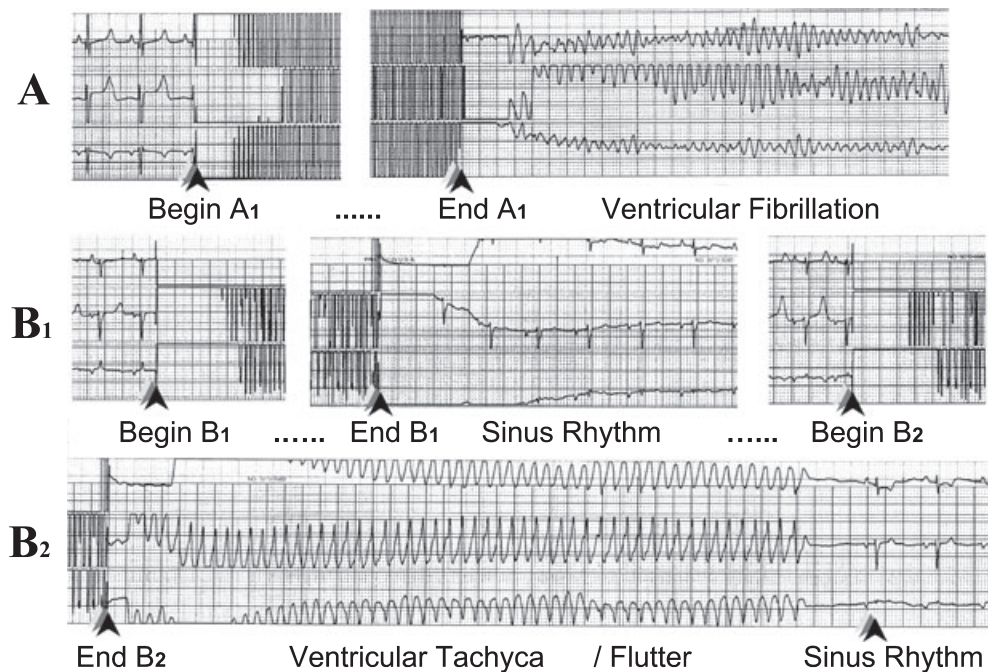


Figure 2. Electrocardiograms from two animals (A and B) exposed to two 40-second TASER discharges. After the first 40-second discharge (End A₁) animal A showed fatal VF. After the first 40-second discharge (End B₁), the ECG for animal B reverted to normal sinus rhythm. After the second 40-second discharge (End B₂), VT/ventricular flutter ensued for several seconds until sinus rhythm was regained.

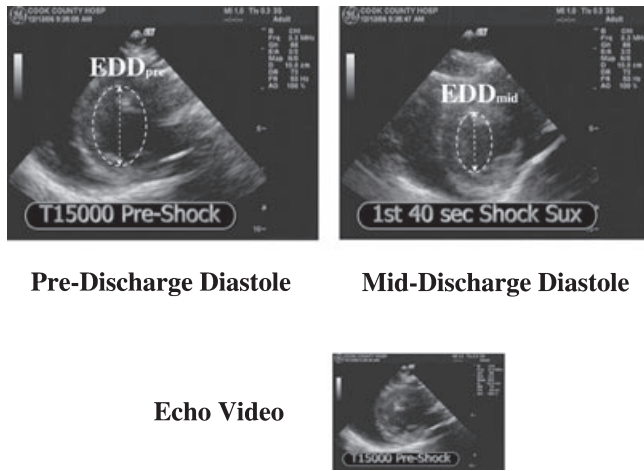


Figure 3. Echocardiograph frame grabs (upper left and right) showing end-diastolic diameter of the LV in the pre-discharge period (left, EDD_{pre}) and during the discharge (right, EDD_{mid}). The dashed ellipses show the outlines of the LV and the dashed lines show the long axes. Owing to difficulties in obtaining the LV view in this animal, the cross-section was not circular. A video link illustrating the capture of LV rhythm by TASER discharge using echo in this animal is at the bottom of the figure. This animal received two 40-second TASER discharges. The video was edited to focus on cardiac rhythms at transition points between rests and discharges. The resolution was decreased to reduce file size.

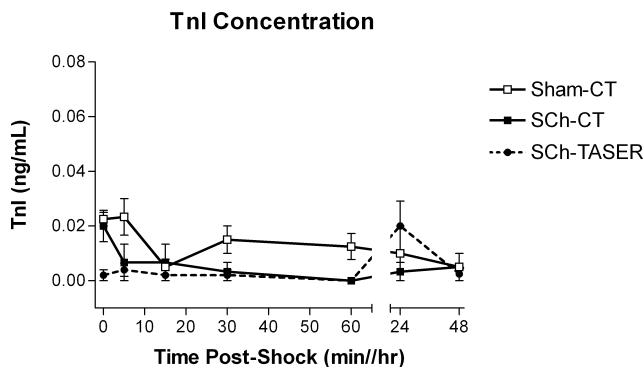


Figure 4. Troponin-I showed an increase at 24 hours postdischarge, but no significant differences were seen between the Sch-TASER and control groups (one-way ANOVA, $p > 0.05$).

Acidosis

Central venous blood pH (Figure 5A), pCO_2 , bicarbonate, and lactate (Figure 5B) showed minor changes from baseline values in the Sch-TASER group ($n = 8$). For pH, the differences between the Sch-TASER group and the control groups were not significant (one-way ANOVA, $p > 0.05$). Lactate was elevated significantly in the Sch-TASER group until 60 minutes postdischarge.

Potassium Levels

As expected, potassium values increased transiently³³ in all animals given Sch, but potassium values were not significantly different between the Sch-TASER and Sch-CT groups (one-way ANOVA, $p > 0.05$). Since potassium values never fell outside the normal range,

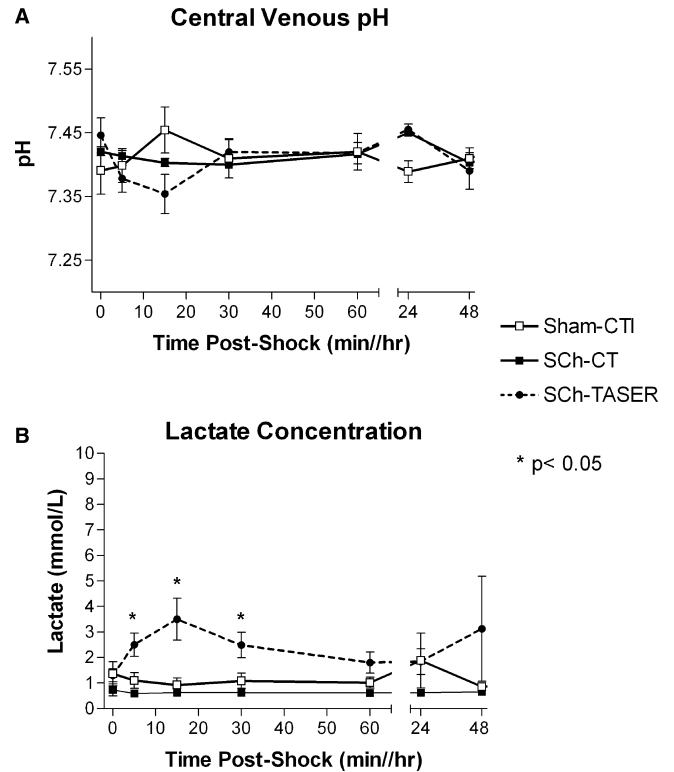


Figure 5. In the Sch-TASER group, central venous blood pH (A) decreased slightly postdischarge reaching a nadir at 15 minutes ($p > 0.05$) and then recovered to normal at 30 minutes postdischarge. Lactate values (B) increased significantly for 30 minutes postdischarge ($p < 0.05$). Blood pH and lactate levels in the control and experimental groups did not change significantly (one-way ANOVA, $p > 0.05$).

cardiac arrhythmias could not be attributed to altered potassium levels.

DISCUSSION

Case reports, autopsies, and retrospective analyses have suggested that TASER discharges may be associated with fatal dysrhythmia in humans, although the frequency of this complication is extremely low.^{2,6-9,34} We show for the first time using echocardiography and ECG that TASER discharges administered with a transcardiac discharge vector in swine have dramatic effects on myocardial function including rhythm capture, VT, or ventricular flutter and sometimes fatal VF. These effects are independent of body mass within the range tested here (25–71 kg), or of the coexistence of systemic acidosis. Our observations are in agreement with those of Nanthakumar et al.²⁰ who used electrically shielded intracardiac ECG and intraaortic manometry in swine in conjunction with discharges from an unmodified TASER X26. They showed that discharges can capture myocardial rhythm, resulting in high rates of ventricular stimulation and potential dysrhythmia with a sharp drop in blood pressure during discharge.²⁰

Using a “custom-built TASER-like device,” others⁷ have reported that the threshold for VF was directly

proportional to body mass for animals ranging from 30 to 117 kg. The output power of their device had to be increased by a factor of at least 15 to induce VF with a 5 second discharge in a 30 kg animal. The power of a police-issue TASER X26 cannot be adjusted by the user. If an unmodified TASER X26 had the same "safety factor," then VF should never have occurred in this study. However, in our previous study,¹⁸ which did not employ SCh, and in this study, cardiac rhythm capture occurred in all animals exposed to TASER discharges, and fatal VF was even observed. Differences in animal sizes (30–117 kg there vs. 25–71 kg range here), discharge durations (5 seconds vs. two 40 seconds), or other experimental conditions may account for the disparities in the results of the studies, yet during the entire duration of each discharge, cardiac rhythm capture and degraded cardiac function were seen in every animal studied here, and in our previous experiments,¹⁸ regardless of animal size. Regression analysis showed no significant correlation between body mass and any other parameter measured in this study.

To the best of our knowledge, this study is the first to use a paralytic agent to isolate the effects of TASER electrical energy on the myocardium independent of skeletal muscle contractions. It is also the first to show that acid–base disturbances that have previously been associated with TASER discharges^{17,18} are primarily a result of intense skeletal muscle contractions, not apnea or depressed cardiac function. SCh, a depolarizing muscle relaxant, can by itself cause bradycardia and junctional myocardial rhythms,³³ so we must consider the possibility that SCh contributed to the arrhythmias seen here. However, bradycardia was not seen here in response to SCh injection, and we¹⁸ and others²⁰ have reported arrhythmias very similar to those seen here in swine exposed to TASER discharges in the absence of SCh.¹⁸ In addition, the results of this study are in accordance with other published animal studies^{19,20,30,31} that have employed standard, law enforcement–grade TASER X26 devices and show that discharges can alter cardiac rhythm in swine. These findings, however, are at variance with those obtained using "custom-built TASER-like devices."^{7,13}

In this swine model, using a transcardiac vector, TASER X26 discharges invariably produced myocardial capture that usually reverted spontaneously to sinus rhythm postdischarge, but in one instance (of six animals and twenty 40-second discharges) degenerated into fatal VF. The extreme acid–base disturbances previously seen after lengthy TASER discharges were independent from these cardiac dysrhythmias and from possible discharge-related apnea. They are primarily the result of intense skeletal muscle contractions. Clearly, the TASER X26 captured cardiac rhythm during discharges, but these aberrant rhythms usually resolved immediately or within several seconds after the discharge ended. As a result, such events would go undetected if they occurred in humans exposed to TASER discharges in the field. In human volunteers, several groups have reported finding no significant ECG abnormalities aside from mild tachycardia after TASER X26 discharges.^{14,16,35}

Electrical noise interfered with ECG during these discharges so that cardiac capture, if it occurred, would have been obscured. A key factor that sets this animal study apart from those human studies is the use of a transcardiac vector, whereas human studies to date have used discharges applied to the back. The discharge times used in those studies ranged from 5 to 15 seconds, so it is possible that the transcardiac discharge vector or longer discharge times are necessary to produce the transient postdischarge ECG abnormalities seen here. However, using echocardiography, we have seen similar abnormalities and even VF in animals exposed to transcardiac vectored discharges of 10 seconds' duration (unpublished data of Walter, Dennis, Valentino, Margeta et al.).

If similar capture of myocardial rhythm occurs in humans exposed to transcardiac discharges, it seems reasonable to speculate that this could be a factor in some of the TASER-associated sudden deaths that have been reported. Although body mass does not appear to be a factor in the cardiac capture seen here, our findings may be most relevant to individuals of smaller mass who have died after lengthy or repeated TASER discharges.⁴

LIMITATIONS

For ethical reasons, anesthetized animals were used and used in limited numbers. Anesthesia precludes pain perception, which is one of the two principal effects of TASER discharges in conscious humans. Although the blood chemistry and echocardiographic findings were highly consistent in this small sample (six animals; sixteen 40-second discharges), an accurate assessment of the incidence of VF requires further study.

Unstressed, resting, sedated animals were studied. However, TASERs are generally used in the field to subdue highly agitated individuals who may also be under the influence of stimulatory drugs. Under those conditions, the effects of TASER discharge might differ from those seen here.¹³ In the future, we will extend this study to anesthetized animals stimulated by cocaine or methamphetamine.

Animal size may have been a limiting factor here. Other investigations of TASER effects have used animals of similar average size to those used here (25–71 kg; 42.2 ± 19.4 kg; 55–156 lb; Lakkireddy et al.,¹³ 34 ± 8.7 kg; Nanthakumar et al.,²⁰ 45–55 kg; Jauchem et al.,¹⁷ 49–58 kg; McDaniel et al.,⁷ 30–117 kg). The size of the animals used here compares most closely with that of children, teenagers, and adult humans with medium frames. It is possible that ventricular capture and fibrillation may be more likely in this size range than in individuals with greater mass.

One vector (dart location) was employed for all discharges. The discharge vector is very important when considering the effects of TASER discharges in animals or in humans, and alternate vectors will result in different amounts of myocardial capture.^{20,30} We are currently completing experiments examining the effects of a wide range of different discharge vectors on cardiac capture.

CONCLUSIONS

To the best of our knowledge, this study is the first to demonstrate several significant effects of TASER discharges in swine using an unmodified law enforcement-grade TASER X26. Using echocardiography and ECG, we have shown that TASER discharges administered with a transcardiac discharge vector in swine have dramatic effects on myocardial function including rhythm capture, VT, or ventricular flutter and sometimes fatal VF.

In contrast to several previous studies in humans^{16,35} and animals,^{7,13,19} we have shown that VT or ventricular flutter occur immediately upon starting the discharge and invariably in all animals tested regardless of body mass (in the 25–71 kg range), using darts that are more than 5 cm from the heart. Thus, there is no safety factor for VT, and a moderate likelihood of fatal VF (one of eight animals tested) with the transcardiac vector used here.

Using the paralytic agent, SCh, TASER electrical discharges in swine capture the myocardium in the absence of extreme systemic acidosis.

The acid–base disturbances previously seen after lengthy TASER discharges in swine^{17,18} are largely the result of intense skeletal muscle contractions and are not caused by apnea or depressed cardiac function occurring during or after the discharge.

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Supplementary Material

The following supplementary material is available for this article online:

Video S1. <http://www.aemj.org/cgi/content/full/j.aem.2007>.

Please note: Blackwell Publishing are not responsible for the content or functionality of any supplementary materials supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.

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