Acute Effects of TASER X26 Discharges in a Swine Model

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Background: **Very little objective laboratory data are available describing the physiologic effects of stun guns or electromuscular incapacitation devices (EIDs). Unfortunately, there have been several hundred in-custody deaths, which have been temporally associated with the deployment of these devices. Most of the deaths have been attributed to specific cardiac and metabolic effects. We hypothesized that prolonged EID exposure in a model animal system would induce clinically significant metabolic acidosis and cardiovascular disturbances.**

Methods: **Using an Institutional Animal Care and Use Committee-approved protocol, 11 standard pigs (6 experimentals and 5 sham controls) were anesthetized with ketamine and xylazine. The experimentals were exposed to two 40-second discharges from an EID (TASER X26, TASER Intl., Scottsdale, AZ) across the torso. Electrocardiograms, blood pressure, troponin I, blood gases, and electrolyte levels were obtained pre-exposure and at 5, 15, 30, and 60 minutes and 24, 48, and 72 hours post-discharge. p values <0.05 were considered significant.**

Results: **Two deaths were observed immediately after TASER exposure from acute onset ventricular fibrillation (VF). In surviving animals, heart rate was significantly increased and significant hypotension was noted. Acid-base status was dramatically affected by the TASER discharge at the 5-minute time point and throughout the 60-minute monitoring period. Five minutes postdischarge, central venous blood pH (6.86 ± 0.07) decreased from baseline (7.45 ± 0.02; $p = 0.0004$). P_{CO_2} (94.5 mm Hg ± 14.8 mm Hg) was significantly increased from baseline (45.3 mm Hg ± 2.6 mm Hg) and bicarbonate levels significantly decreased (15.7 mmol/L ± 1.04 mmol/L) from baseline (30.4 mmol/L ± 0.7 mmol/L). A large, significant increase in lactate occurred postdischarge (22.1 mmol/L ± 1.5 mmol/L) from baseline (1.5 mmol/L ± 0.3 mmol/L). All values returned to normal by 24 hours postdischarge in surviving animals. A minor, nonsignificant increase in troponin I was seen at 24 hours postdischarge (0.052 ng/mL ± 0.030 ng/mL, mean ± SEM).**

Conclusions: **Immediately after the discharge, two deaths occurred because of ventricular fibrillation. In this model of prolonged EID exposure, clinically significant acid-base and cardiovascular disturbances were clearly seen. The severe metabolic and respiratory acidosis seen here suggests the involvement of a primary cardiovascular mechanism.**

Key Words: **Taser, Electromuscular incapacitation, Acidosis, Electrocardiograph.**

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Stun guns or electromuscular incapacitation devices (EIDs) generate between 25,000 and 250,000 V and can be discharged for as long as 5 to 10 minutes continuously. It is estimated that millions of people in the United States are at risk of exposure to EIDs daily and that thousands are exposed annually.[1] Amnesty International has compiled a list of more than 100 fatalities from cardiac arrest in the 2001 to 2004 period that were associated with EID use in the United States.[2] This growing list of fatalities has reinvigorated interest in the safety of EIDs and potential complications associated with their use, especially their ability to induce fatal ventricular dysrhythmia.[3–6] In the United States alone, more than 200,000 individuals have been exposed to discharges from the most common type of EID, the TASER. In training sessions, more than 24,000 law enforcement personnel have been exposed to brief TASER discharges but no deaths have been documented. Worldwide, TASERs are currently used by more than 9,100 law enforcement agencies and owned by more than 115,000 private citizens (www.taser.com). Despite the increasing usage of TASERs and other EIDs, there is no consensus in the biomedical community regarding their safety.

EIDs are very effective when used by law enforcement to incapacitate combative suspects while reducing the risk of injury to officers, suspects, and by-standers.[2] These devices utilize time-varying DC currents that evoke strong, repetitive contractions in most or all of the somatic musculature. The mechanism by which this occurs and the pathophysiologic effects of these discharges are poorly understood. There are several reasons for this lack of information including but not limited to (1) disagreement about the electrical output from these devices especially under resistive load, (2) disagree-

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ment about what load is appropriate or representative, (3) the lack of a standard model for study in vitro or in vivo, and (4) a general disinclination for academic scientists to study these devices because they are viewed as weapons.[3–5,7] Under no-load conditions, the TASER X26 delivers DC pulses at a voltage of about 50 kV, with a pulse duration of 140 μseconds, a frequency of 19 Hz, and power of 0.36 J/pulse (www.taser.com). In vivo, this type of discharge causes severe pain, strong muscle contractions, and incapacitation of volitional movement. However, Ruggieri[7] asserts that the peak currents achieved by the TASER under physiologic resistive loads can be many times higher than 2.1 mA and may easily exceed the ventricular fibrillation (VF) threshold. As a result of such disparities, the safety profile and effects of EID current exposure on the function and structure of living tissue cannot be extrapolated reliably.

Many of the initial studies on EIDs were performed on the much less powerful first or second generation devices.[8–10] The recent peer-reviewed literature on fourth generation EIDs (such as the TASER X26) is still emerging and the results are conflicting. Some studies, using a TASER-like device, showed no evidence of acute dysrhythmia and a large safety margin for the development of ventricular dysrhythmia in swine.[4,11] Similarly, neither acidosis nor hyperkalemia have been observed in healthy human volunteers exposed to brief (2.7 seconds on average) TASER X26 discharges.[12,13] However, in swine models, Jauchem et al.[14] showed the development of significant acidosis, and Webster et al.[15] and Nanthakumar et al.[16] have shown the potential for fatal dysrhythmias with TASER X26 exposure. Such conflicting results make it difficult to establish guidelines regarding the need for treatment or even monitoring of the increasing number of patients who arrive in emergency rooms after exposure to EID discharges.

In an attempt to reconcile some of the conflicting information about the effects of TASER discharges, we have studied the effects in a well-characterized swine model. Our working hypothesis was that subcutaneous discharges from the TASER X26 can produce significant cardiac effects including acute dysrhythmia or VF and that these effects may be exacerbated by concomitant acidosis or electrolyte and biochemical abnormalities.

MATERIALS AND METHODS

Animals and Groups

Three- to 6-month-old Yorkshire pigs (Michael Fanning Farms, Howe, IN) weighing between 22 kg and 46 kg were used. The experimental group (80-second thoracic discharge) and the negative or sham control group, were comprised of six and five animals, respectively. The size of the animals used in our study correlates with that of children, teenagers, and some adult humans with small frames. Other investigations of TASERs have used animals in a similar size range.[4,11,14,16] The Institutional Animal Care and Use Committee (IACUC) for the Hektoen Institute for Medical Research, LLC reviewed and approved this project.

All animals were deeply anesthetized during each monitoring session using intramuscular and intravenous 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). Ketamine/xylazine/glycopyrrolate (30/3/0.01 mg/kg) was administered intramuscularly for sedation and then ketamine and xylazine (5.6/0.8 mg/mL) in sterile saline were instilled intravenously using an infusion pump (Flogard 6200; Travenol, Deerfield, IL) through a 23-guage cannula placed into an ear vein at a rate of 3 $mL \cdot h^{-1} \cdot kg^{-1}$ (16.8/2.4 mg/kg). Animals were intubated using cuffed endotracheal tubes (5.0–6.5 mm, Rusch; Kernen, Germany) after anesthetizing the larynx with 0.25 mL to 1.0 mL of sprayed 20% benzocaine (Hurricaine; Beutlich Pharm., Waukegan, IL). Breathing was controlled (15 breaths per minute; tidal volume = 10 mL/kg; minute volume = 150 mL/kg). The TASER X26 was discharged (see below) in two separate 40-second intervals for a total of 80 seconds, during which time the ventilator was shut off but spontaneous breaths were permitted. Two ventilated breaths (within 10 seconds) were administered between the 40-second discharges. Breathing rate was adjusted after discharge according to demand. The purpose of this was to ensure that the ventilator was not the cause of any observed respiratory acidosis. Animals were maintained in dorsal recumbence for all electrical discharges and monitoring procedures. At the conclusion of each monitoring session, intravenous yohimbine (0.05–0.15 mg/kg; Yobine; BenVenue Labs, Bedford, OH) was used to reverse the effects of xylazine and to speed recovery from anesthesia.

Instead of using inhaled halothane or isoflurane anesthesia, ketamine/xylazine was used throughout this study (except for thoracotomized animals). The primary local electrical injury anticipated with these waveforms was membrane electroporation, particularly of nerve and muscle.[17,18] This effect is sensitive to the presence of lipids or highly lipid-soluble agents such as isoflurane, halothane, or barbiturates. The ketamine and xylazine combination used here has also been shown to be an effective general anesthetic in swine[19,20] and our data confirm this (see below).

Test Device

An unmodified, police-issue TASER X26 device was used to produce electromuscular incapacitation. Because it is illegal for civilians to possess the TASER X26 in Illinois, a member of the local law enforcement community trained in TASER use delivered the discharges. TASER lithium 6 V Digital Power Magazines (DPM) were used as the power source for all discharges. DPM charge state was monitored before and after each discharge and at no time was a DPM used with a charge state less than 70%.

Experimental Set-Up and EID Discharge

While in dorsal recumbence, all four limbs of the animal were restrained to the table. The TASER cartridge was fired

into a towel and the darts were disentangled from the cloth without disrupting any of the fine wires, insulation, or connections. The barbed darts were placed along a line parallel to the cardiac axis. The superior or noncurrent-emitting dart was placed 13 cm superior to the xyphoid process and 5 cm to the right of the midsternal line. The lower or current emitting dart was placed 7 cm to the left of the umbilicus. This dart configuration produced a diagonal separation of approximately 30 cm in each animal and is similar to that used by Jauchem et al.[14] For two of the six animals in the experimental group, the superior dart was the current emitting dart. All darts were manually inserted perpendicular to the skin and to the maximum depth allowed by the length of the barbed end (3/8 of an inch) such that the dart tip was located in subcutaneous tissue. For TASERs discharged from distances less than 11 feet, skin penetration has been shown to occur for both darts in approximately 65% of TASER strikes in the field.[21]

The TASER X26 was discharged in two separate 40-second intervals for a total of 80 seconds, during which time the ventilator was shut off but spontaneous breaths were permitted. Two ventilated breaths were administered during the 10-second pause between the 40-second discharges. The discharge times of stun devices as used in the field vary greatly. Often short bursts (~5 seconds) are sufficient to subdue most subjects, but the devices are capable of delivering very prolonged, continuous discharges. The only practical limit on the discharge duration is the amount of battery power available, so continuous discharges could be administered for more than 10 minutes and instances of discharges longer than 90 seconds have been reported for TASERs.[22] Subjects are usually incapacitated almost immediately upon exposure to TASER discharges but they regain muscle control very rapidly after discontinuation of the discharge. Highly combative subjects may receive prolonged or repeated discharges, during which time officers can approach and restrain them.

Cardiac Rhythm and Echocardiography

Cardiac rhythm was evaluated and monitored continuously during anesthesia using a five-lead electrocardiogram (EKG) and monitor (Datex instruments, Helsinki, Finland) and at each experimental time point 10- to 15-second tracings were printed and retained. EKGs were also recorded throughout the duration of the discharge. Because of the amount of electrical interference created by the TASER discharge, EKGs done during the discharge were unreadable. To adequately assess the rhythm and function of the myocardium, echocardiography was performed using a Sonosite 180 with a 2-MHz probe (Sonosite Inc. Bothell, WA) on four of the six experimental animals. Echo images were first obtained predischarge to establish a baseline for each animal in the left parasternal axis. Echocardiography was then continued during and after the TASER discharge to assess, in real time, any changes that occurred in myocardial rhythm and function. Video records of each echo were digitally recorded for further analysis.

Controls

Five sham control animals were studied for 72 hours using the same paradigm as that used for animals exposed to TASER discharges except that they were not exposed to any discharges during the monitoring period. At the completion of the 72-hour blood sampling and monitoring period, two of these sham animals underwent thoracotomy. Each of these animals then received two 40-second TASER discharges while direct visual monitoring was performed. Each of these animals was physiologically normal before these discharges according to all blood chemistries, vital signs, and EKG. These animals had received no previous TASER discharges. In addition, baseline intra-animal data were obtained for all 11 (6 experimentals and 5 sham controls) animals studied.

Thoracotomy

To further document cardiac activity, two 40-second discharges were administered to two of the five control animals (31 kg and 46 kg) just before being euthanized (see above). Left anterior thoracotomies were performed under inhaled anesthesia with 1.5% to 2% isoflurane. Electrocautery was avoided to eliminate any non-EID electrical exposure. An incision approximately 10-cm long was made over the left anterior thorax in the fifth or sixth intercostal space. Sharp dissection was carried down to access the left thorax. A rib spreader was used to expose the heart and lungs. The rib spreader was placed outside the current path between the darts and it was not in direct contact with the heart. The left lung was retracted out of the field with gauze sponges. The pericardium was opened sharply facilitating a direct view of the beating myocardium. The TASER darts were then placed in the manner previously described and two 40-second discharges were administered. The 31-kg animal received the second 40-second discharge with the superior dart as the current emitting dart. Cardiac activity was directly monitored and recorded before, during, and after TASER discharge for subsequent analysis and comparison with echocardiographic data.

Blood Samples and Analysis

There were eight time points at which central venous blood was drawn from the precaval venous complex, and vital signs (tissue oxygen saturation, heart rate, and blood pressure [BP]), and additional EKGs were recorded. The sampling time points included predischarge (time 0) and 5, 10, 15, 30, 60 minutes, 24, 48, and 72 hours postdischarge. Animals were euthanized according to American Veterinary Medicine Association standards after the 72-hour time point by switching the anesthesia to 5% inhaled isoflurane and injecting 3 mol/L KCl into the heart.

Immediately after being drawn, each blood sample was placed into heparinized and plain vacutainer tubes. The heparinized blood was tested using an iSTAT analyzer (Abbott Point-of-Care, Abbott Park, IL) using CG8+, CG4+, creatinine, and troponin I (TnI) cartridges. These cartridges return

data on pH, P_{CO_2}, bicarbonate, lactate, potassium, TnI, and creatinine. Blood samples were stored on ice for a maximum of 2 hours, centrifuged (3,000*g* for 15 minutes at 4°C), plasma and serum aliquoted into 400 μL microcentrifuge tubes, and samples stored at -85°C until use. Serum from each time point was thawed and assayed for creatine kinase-MB isoform (CK-MB) and myoglobin using microplate enzyme-linked immunosorbent assays.

When whole blood lactate values exceeded the CG4+ maximum value of 20.0 mmol/L, the aliquoted serum was diluted 1:1 with normal saline (0.9% NaCl). The diluted serum was then assayed using a CG4+ cartridge to get a numerical lactate value. This value was then doubled and entered into the data set. This dilution method was validated by first diluting iSTAT standards in a similar fashion and analyzing them using a CG4+ cartridge. The values obtained for the diluted iSTAT standards were one-half of the values expected for undiluted standards. The manufacturer has validated the use of serum for lactate determinations instead of whole blood.

Serum Myoglobin and CK-MB Determination

Plasma or serum myoglobin, TnI, and CK-MB have been shown to be useful in evaluating cardiac muscle damage because of myocardial infarction.[23–27] The time course for the appearance of each of these markers is known. Levels of cardiac TnI, the most specific marker for myocardial damage, peak at 12 to 24 hours, and may remain elevated for several days. Serum myoglobin becomes elevated within 2 to 4 hours of myocardial injury. CK-MB is found in cardiac and skeletal muscle but is present in much higher quantities in cardiac muscle. CK-MB levels become elevated within 3 to 4 hours of cardiac injury and remain elevated for 60 to 70 hours. Myoglobin and CK-MB can become elevated from noncardiac related injuries such as chronic muscle disease, skeletal muscle trauma, and renal failure.[23,28,29] As a result, it is common to evaluate all three of these markers to determine the extent of cardiac and skeletal muscle injury.

Serum samples stored at −85°C were thawed once and tested for myoglobin (20 μL/well) and CK-MB (25 μL/well) using solid phase microplate sandwich enzyme-linked immunosorbent assays (Diagnostic Automation, Calabasas, CA). All samples and standards for these assays were performed in duplicate and averaged. Standard curves using four to seven reference standards of different concentrations were generated for each run. Myoglobin and CK-MB concentrations for the experimental serum samples were interpolated from these standard curves using best-fit regression formulas generated by Excel (Microsoft, Redmond, WA).

Data Reduction and Statistical Analysis

All data points represent means ± SEM for each parameter. Parametric statistics including two-way analysis of variance (ANOVA) or paired *t* tests were used to compare quantitative data and groups. Trends were evaluated using linear regression. The experimental groups were compared against their own baseline and against the control group for each parameter (Prism v.3.03, GraphPad Software, San Diego, CA). Vital signs and blood chemistry values obtained for the animals that died within minutes of TASER discharge were not included in the statistical analyses.

RESULTS

Vital Signs Were Severely Altered by TASER Discharge

No spontaneous respiratory effort was observed during TASER discharge. An acute onset of tachycardia was noted after TASER discharge. Heart rate increased from a baseline of 103 bpm ± 9 bpm (mean ± SEM). The heart rate was greatest at the 5 minutes postdischarge time point (157 bpm ± 5 bpm; $p = 0.0085$ vs. baseline). Heart rate then gradually decreased during the remainder of the 60-minute monitoring period, but was not observed to return to the baseline until the 24-hour time point. At the 24-hour time point and subsequent time points, heart rates in experimental animals were similar to those of controls. The acute onset of tachycardia was not seen in control animals. Control animals showed a decrease in heart rate from baseline (91 bpm ± 2 bpm) during the initial 60-minute monitoring period with a nadir at 60 minutes (72 bpm ± 2 bpm; $p < 0.05$ vs. baseline). The observed effect on heart rate in the experimental group was significantly different from that of the control group when compared for the initial 60-minute monitoring time period by two-way ANOVA ($p < 0.0001$).

BP (Fig. 1) showed a decrease after TASER discharge in the first 60 minutes. BP reached a nadir at 15 minutes postdischarge (systolic BP = 79 mm Hg ± 8 mm Hg) in the experimental group. This decrease in systolic BP was significant ($p = 0.02$) compared with the baseline value (133 mm Hg ± 8 mm Hg) in the experimental group. The systolic BP gradually increased during the 60-minute monitoring period and returned to baseline values at 24 hours. BP did not show any significant changes in the control group. The difference observed between controls and experimental BP was significant (two-way ANOVA; $p < 0.001$).

One Experimental Animal Died of Acute VF

One animal in the experimental group (29 kg) died from VF after TASER discharge. Cardiac rhythm could not be discerned by EKG during the discharge because of the electrical interference and muscle contractions created by the TASER. Cardiac rhythm was evaluated by echocardiography during the discharge and found to be consistent with ventricular tachycardia. When the discharge ceased, sustained ventricular tachycardia was noted on echocardiography and confirmed by EKG (Fig. 2). During the course of the next few minutes, the ventricular tachycardia then degenerated into fatal VF. As previously indicated, all surviving experimental animals showed brief atrioventricular (AV) dyssynchrony followed by sinus tachycardia

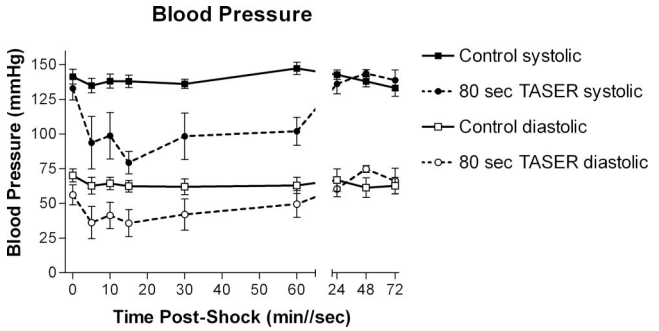

Fig. 1. *Blood pressure versus time for the 72-hour time course after TASER discharge. Both systolic and diastolic pressures (mean ± SEM) are plotted for the experimental and control groups. Systolic blood pressure reached a nadir at* t = *15 minutes (79 mm Hg ± 8 mm Hg). This value was significantly different from experimental baseline (*p = *0.02) and from control values at* t = *15 minutes (*p < *0.001). The differences observed between control and experimental blood pressures at all time points were significant (two-way ANOVA;* p < *0.001).*

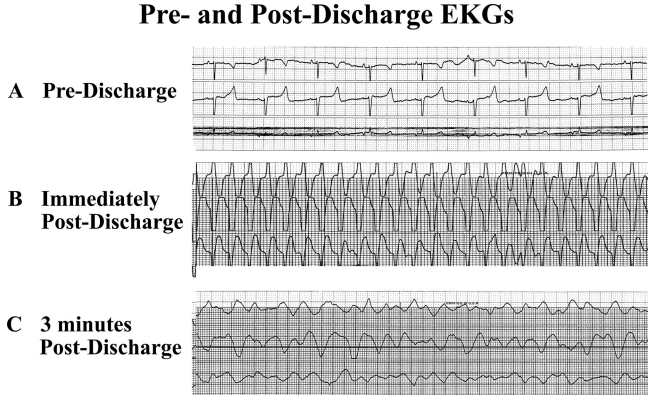

Fig. 2. *EKGs from one animal taken before (*A*) and after TASER discharge showing sustained ventricular tachycardia immediately after the discharge (*B*) followed by VF approximately 3 minutes later (*C*).*

after the discharge. Despite persistent sinus tachycardia, no EKG evidence of acute dysrhythmia was seen in the surviving animals.

Echocardiography (echo) showed capture of the ventricular rhythm during TASER discharge but motion artifacts prevented quantitative analysis of cardiac output and ejection fraction. One animal, as described above, went into VF after the discharge as confirmed by EKG and echo. The remaining three animals all showed capture of ventricular rhythm with rapid ventricular contractions seen on echo consistent with ventricular tachycardia (approximate rate of 300 bpm). This capture of cardiac rhythm occurred immediately after the start and continued for the duration of the TASER discharge as seen by echo. Sinus rhythm was regained after a brief period of AV dyssynchrony in each of these three animals and sinus tachycardia began within 1 minute after termination of the discharge.

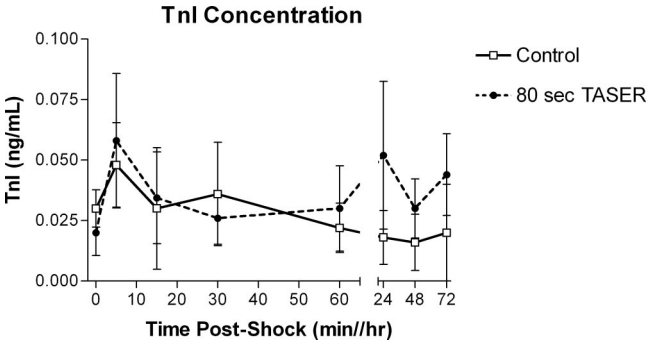

Fig. 3. *Troponin I (TnI) values during the 72-hour time course after TASER discharge. TnI values showed an initial increase from baseline values at 5 minutes postdischarge in both experimental and control animals. No significant differences were seen when TnI values for experimental and control animals were compared using two-way ANOVA (*p > *0.05).*

TnI (Fig. 3) showed an initial increase from baseline in the experimental animals at the 5-minute time point. A similar increase was also noted in control animals. TnI levels peaked at 24 hours postdischarge (0.052 ng/mL ± 0.03 ng/mL), this was not a significant increase from baseline values (0.02 ± 0.01, $p > 0.05$). No significant differences were seen when TnI values for experimental and control animals were compared using two-way ANOVA ($p > 0.05$). No significant changes were seen in CK-MB at anytime compared with that of controls.

Severe Metabolic and Respiratory Acidosis was Seen After TASER Discharge

Central venous blood pH (Fig. 4) showed a large decrease from baseline (7.45 ± 0.02) after the TASER discharge at the 5-minute time point (6.81 ± 0.07; $p = 0.0004$).

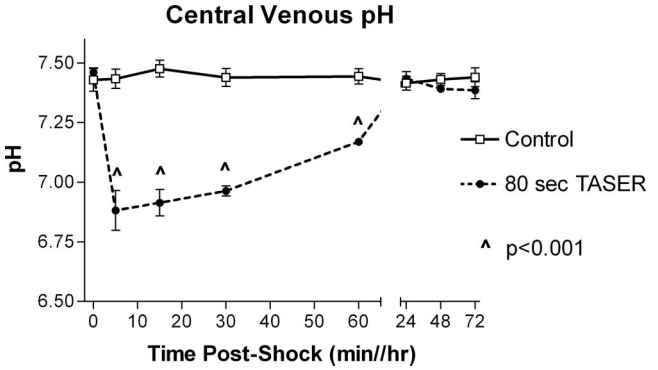

Fig. 4. *Central venous pH over time for control and experimental groups. Control animals had a baseline similar to experimentals but showed no significant changes from this baseline during the 72-hour monitoring period. A large decrease in central venous pH was noted after TASER discharge in the experimental group. Central venous blood pH was decreased throughout the 60-minute postdischarge monitoring session (*p̂ < *0.001, paired* t *tests) but returned to baseline values subsequently.*

In the experimental group, central venous blood pH decreased throughout the 60-minute postdischarge monitoring session but returned to baseline values subsequently. Control animals had a similar baseline and showed no significant changes during the 72-hour monitoring period. The observed difference was significant when compared using two-way ANOVA during the initial 60 minutes ($p < 0.001$).

Extreme hypercapnia was noted after TASER discharge (Fig. 5). A dramatic increase in P_{CO_2} was seen at 5 minutes (108.3 mm Hg ± 14.6 mm Hg) postdischarge. This change was in stark contrast to baseline values (45.3 mm Hg ± 2.6 mm Hg; $p < 0.0048$) for this group. The P_{CO_2} gradually decreased during the 60-minute monitoring period, and returned to baseline at subsequent time points. Control animals had normal P_{CO_2} values throughout the entire monitoring period. The difference observed between controls and experimental animals was significant (2-way ANOVA; $p < 0.001$).

Bicarbonate levels (Fig. 6) were found to be acutely and severely decreased from baseline values (30.8 mmol/L ± 0.9 mmol/L) at 5 minutes postdischarge (15.7 mmol/L ± 1.0 mmol/L; $p = 0.0046$). This decrease contrasted with the control group, which showed no significant changes in bicarbonate levels during the entire experimental time course. Bicarbonate levels remained decreased throughout the initial 60-minute postdischarge monitoring period and returned to baseline subsequently. When compared with controls over time, the observed changes in bicarbonate were significant (2-way ANOVA; $p < 0.001$).

Lactate values (Fig. 7) increased more than 13-fold after TASER discharge. Lactate levels increased from the experimental baseline of 1.6 mmol/L ± 0.3 mmol/L to 22.1 mmol/L ± 1.5 mmol/L ($p < 0.0001$) at 5 minutes postdischarge. Lactate levels remained elevated throughout the initial 60-minute monitoring period and returned to baseline values at 24 hours postdischarge.

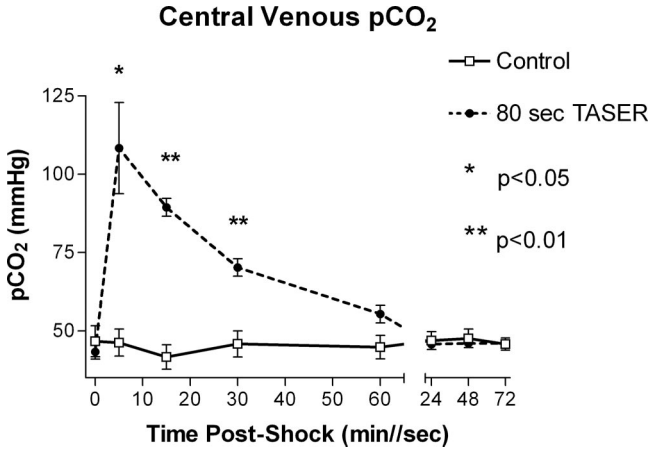

Fig. 5. *Central venous P_{CO_2} during the 72-hour time course after TASER discharge. A massive increase in P_{CO_2} was seen postdischarge (*p < 0.05, **p < 0.01, paired t tests). The P_{CO_2} gradually decreased during the 60-minute monitoring period and returned to baseline subsequently. Control animals had normal P_{CO_2} values throughout the entire monitoring period.*

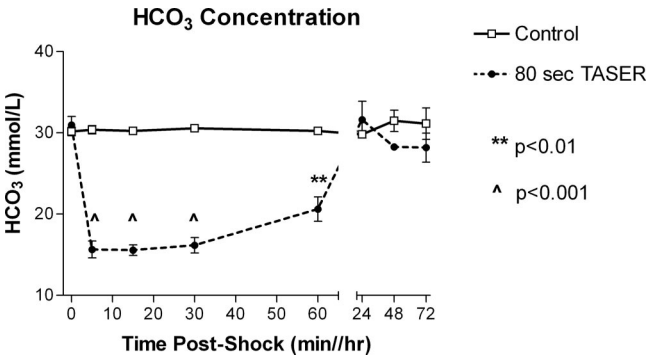

Fig. 6. *Bicarbonate concentration during the 72-hour time course after TASER discharge. The control group showed no significant changes in bicarbonate levels during the experimental time course. However, in the experimental group, bicarbonate levels were greatly decreased from baseline values (30.8 mmol/L ± 0.9 mmol/L) at 5 minutes postdischarge (15.7 mmol/L ± 1.0 mmol/L; p = 0.0046, t test). These levels remained decreased throughout the initial 60-minute postdischarge monitoring period and returned to baseline subsequently. When compared with controls over time, the observed changes were significant (two-way ANOVA; p < 0.001). Time points at which experimental and control values differed significantly are shown (**p < 0.01, ^p < 0.001, paired t tests).*

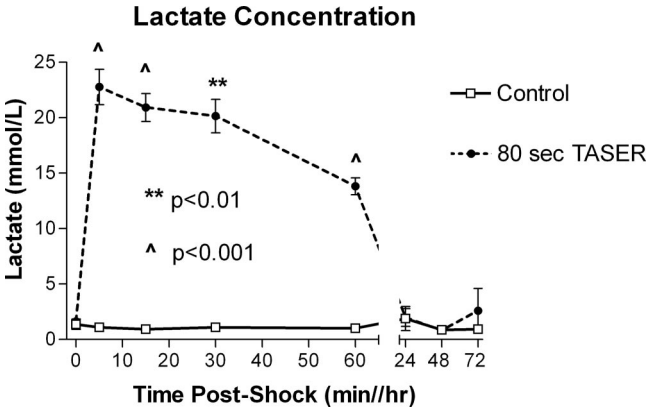

Fig. 7. *Lactate concentration during the 72-hour time course after TASER discharge. Control animals showed no significant changes in lactate levels, but these values increased more than 13-fold after TASER discharge in the experimental group. Lactate levels remained elevated throughout the initial 60-minute monitoring period and returned to baseline values at 24 hours postdischarge. Time points at which experimental and control values differed significantly are shown (**p < 0.01, ^p < 0.001, paired t tests).*

Control animals did not show any significant changes in lactate levels. Lactate levels in the control and experimental groups were significantly different when compared for the initial 60-minute monitoring time period (2-way ANOVA; $p < 0.001$).

Central venous oxygen saturation (Fig. 8) for the control and experimental groups was not significantly different at time 0 ($p = 0.70$). However, it decreased significantly after TASER discharge from the experimental baseline of 78.8% ± 4.6% at time 0 to 50.5% ± 5.8% at 15 minutes postdischarge ($p = 0.02$).

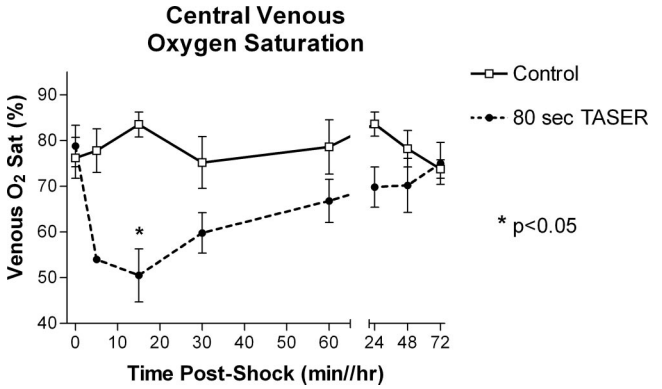

Fig. 8. *Venous oxygen saturation during the 72-hour time course after TASER discharge. Central venous oxygen saturation decreased significantly after TASER discharge and remained low throughout the initial 60-minute monitoring period. For the entire 60-minute postdischarge period, venous saturation for the experimental group was significantly decreased compared with that for controls (two-way ANOVA;* $p < 0.001$*). Time points at which experimental and control values differed significantly are marked (*$p < 0.05$*). Values returned to predischarge levels at 24 hours and remained at these levels subsequently.*

At 5 minutes postdischarge, venous oxygen saturation for only one sample (54%) was available in the experimental group. Other samples tested at this time returned "not measurable" results, likely because of the severe acid-base disturbance. As a consequence, this time point was excluded from the comparisons. Venous oxygen saturation remained low throughout the initial 60-minute monitoring period. Values for the experimental group then returned to predischarge levels at 24 hours and remained at these levels subsequently. For the entire 60-minute postdischarge period, venous saturation for the experimental group was significantly decreased compared with that for controls (2-way ANOVA; $p < 0.001$).

Thoracotomy

Before TASER discharge, with the heart exposed via left anterolateral thoracotomy (see supplemental video clip, ArticlePlus, www.jtrauma.com), normal sinus rhythm was directly visualized and confirmed by EKG. When the TASER discharge started, sinus rhythm was immediately (within 1 second) disrupted. In the 31-kg animal, the first 40-second discharge resulted in immediate capture of the myocardium producing rapid ventricular contractions consistent with ventricular tachycardia. During this discharge, atrial standstill was also seen. When the first discharge ceased, approximately 15 seconds of dyssynchronous atrial and ventricular contractions were noted, after which sinus rhythm resumed. During the period between discharges, the current emitting dart was relocated to the superior position. The second discharge resulted again in immediate disruption of sinus rhythm and ventricular tachycardia. Atrial standstill was again noted during this discharge. However, after 16 seconds, the ventricular tachycardia was replaced by fatal VF.

In the 46-kg animal, both 40-second discharges were administered with the TASER darts in the usual positions described in Methods. The first discharge resulted in immediate capture of ventricular rhythm resulting in ventricular tachycardia. Normal atrial contractions were noted during the discharge, but these contractions were not synchronized with ventricular contractions. When the discharge ceased, sinus rhythm resumed immediately. Similar cardiac effects to those seen in the first discharge were observed during the second 40-second discharge. At the end of the second discharge, sinus rhythm resumed immediately and sinus tachycardia was noted. This animal survived for 20 minutes without apparent ill effects at which time it was euthanized. For both animals, the cardiac activity directly visualized by thoracotomy was consistent with that seen by echocardiography.

TASER Discharge had Moderate Affects on Potassium, Sodium, and Creatinine Levels

Potassium values increased slightly in all animals from baseline to 5 minutes postdischarge. This increase was seen in both controls and experimental animals. The observed increase in potassium concentration at 5 minutes (4.2 mmol/L $\pm$ 0.1 mmol/L) in experimental animals was significant when compared with baseline (3.7 mmol/L $\pm$ 0.1 mmol/L). Potassium levels in the control and experimental groups were significantly different ($p = 0.0328$), but the differences were not clinically significant. At no time point did potassium values fall outside the normal range in any of the animals.

Creatinine values did not change significantly after TASER discharge nor did they exceed normal levels in any of the experimental animals (range, 1.1–1.7 mg/dL). Sodium levels showed an acute increase at 5 minutes after TASER discharge. The sodium concentration increased from a baseline value of 141.0 mmol/L $\pm$ 1.2 mmol/L to 148.2 mmol/L $\pm$ 1.6 mmol/L ($p = 0.0052$) then gradually returned to baseline at the 60-minute time point. Control animals showed a small decrease from baseline (138.3 mmol/L $\pm$ 4.8 mmol/L) at 5 minutes postdischarge (135.7 mmol/L $\pm$ 3.8 mmol/L; $p > 0.05$) and a return to baseline at 60 minutes postdischarge. The observed difference between control and experimental animals in sodium concentration was significant when compared for the 60-minute postdischarge time period (2-way ANOVA; $p < 0.0001$).

TASER Discharge Moderately Affected Serum Myoglobin

Mean serum myoglobin levels in the experimental group at 30 minutes postdischarge (25.9 ng/mL $\pm$ 3.2 ng/mL, $p = 0.0082$) were elevated when compared with baseline (12.7 ng/mL $\pm$ 1.9 ng/mL). However, during all time periods, myoglobin levels in the control and experimental groups were not significantly different (2-way ANOVA; $p > 0.05$). All other values were within normal limits and variations were not of clinical significance.

DISCUSSION

Case reports, autopsies, and retrospective analyses have suggested that EID discharge may be associated with fatal dysrhythmias in humans, although the occurrence of this complication is rare.[3–6,30,31] The dart placement chosen for the present study, with the current path traversing the left thorax, may represent a worst-case type of configuration for cardiac consequences from an EID such as the TASER X26. Our results show that during TASER discharges with this transcardiac vector there is a highly reproducible capture of cardiac rhythm producing ventricular tachycardia. Postdischarge effects included AV dyssynchrony and sometimes fatal VF.

Echocardiography showed that cardiac rhythm was unmistakably affected during every TASER discharge studied. Rapid or immediate onset of atrial standstill, ventricular tachycardia, or VF occurred during these discharges. This study is the first to show the effects of the TASER X26 on the myocardium during thoracic discharges using a combination of echo, thoracotomy, and EKG. Our observations are in general agreement with those of Nanthakumar et al.[16] who showed, using intracardiac EKG monitoring, that an unmodified TASER X26 can capture myocardial rhythm resulting in high rates of ventricular stimulation and potential dysrhythmia.[16]

In two of eight animals exposed to TASER discharge, one with and the other without thoracotomy, the capture of cardiac rhythm and ventricular tachycardia were followed by VF and death. These animals had not been exposed to TASER discharges previously, showed no pre-existing electrolyte abnormalities and displayed no other physiologic abnormalities before the TASER discharges. The experimental conditions used for each of these animals differed somewhat. One animal had undergone a thoracotomy and had been anesthetized with inhaled anesthesia, whereas the other animal did not undergo thoracotomy and had been anesthetized with intravenous ketamine and xylazine. It could be argued that inhaled anesthesia reduces the threshold for VF[16,19] and thoracotomy provides an atypical, more direct current path to the heart; however, neither of these conditions existed in the second case and fatal VF was seen nonetheless. It is possible that VF is a direct result of the current vector in combination with cardiac capture during the vulnerable period of ventricular repolarization (T-wave). Stimulation of the myocardium during this period has long been recognized as a cause of sustained ventricular dysrhythmia and sudden death.[16,32] The frequency of the TASER wave form (19 Hz) makes it highly likely that one or more pulses will occur during T-waves even with brief discharges (1–5 seconds), yet sudden death is very rarely seen subsequent to TASER discharges in humans. The mechanism whereby these discharges capture cardiac function clearly requires further study.

McDaniel et al.[4] showed that the threshold for VF with EID discharges was directly proportional to body mass for animals ranging from 30 to 117 kg. They also reported that the output of their custom-built TASER-like device had to be increased by a factor of 15 to induce VF with a 5-second discharge in 30 kg swine. Our animals varied in mass from 22 to 46 kg and two animals (29 kg and 31 kg) showed fatal VF after two 40-second discharges. If an unmodified TASER X26 has the same safety factor as that reported by McDaniel et al.,[4] then we should never have seen VF.

Webster et al.[15] showed that discharges from a standard TASER X26 can cause VF and that the distance of the current emitting dart from the heart is a determining factor. The darts used here were placed with consistent reference to anatomic landmarks but the specific dart-to-heart distances were not measured. However, the approximate dart-to-heart distances (5–10 cm from the superior dart to the right ventricle and twice this from the inferior dart to the right ventricle) greatly exceeded the average distance of 1.5 cm and the maximum distance of 2.4 cm to the right ventricle where VF was reported by Webster et al.[15] To some extent, this may be related to the thinner body wall and smaller thoracic dimensions in our animals (22–46 kg) when compared with those (54–74 kg) used by Webster et al.[15]

In addition to direct electrical disruption of cardiac rhythm, it has been postulated that deaths associated with EID exposure may result from cardiac instability related to EID-induced lactic acidosis.[31,33] Acidosis at pH <7.20 can lower the VF threshold, cause hyperkalemia, and reduce cardiac output.[34] The profound metabolic and respiratory acidosis observed here was caused by the extreme degree of repetitive, global skeletal muscle contraction, by apnea, or by severe circulatory dysfunction.

In this regard, our findings concur with those of Jauchem et al.,[14] where TASER X26 discharges in anesthetized swine caused severe acidosis (pH <7.0) accompanied by dramatic hypercapnia ($P_{CO_2} >100$ mm Hg) and elevated lactate (>15 mmol/L). It is known that when swine are exercised to exhaustion, large increases in lactate (>15 mmol/L) and resultant decreases in bicarbonate are seen.[35] These metabolic changes from exhaustive exercise are countered in conscious swine by hyperventilation and resultant decreases in P_{CO_2}.[35] In the Jauchem et al.[14] study, acidosis may have arisen from inadequate spontaneous respiration and a lack of mechanical ventilation. In the present study, all animals were mechanically ventilated except during the two 40-second actual or sham discharge intervals. Immediately postdischarge, the respiratory rate was adjusted upward to meet the minute ventilation demand of each animal. Despite this intervention, clinically significant respiratory and metabolic acidosis persisted after the two 40-second TASER discharges but not after sham discharges.

The combination of severe hypercapnia and acidosis in the presence of hypotension indicates that circulatory function was affected by TASER X26 discharges. The hypercapnia seen in venous samples is similar to that seen with patients in cardiac arrest.[36] When the heart is not contracting

effectively, the tissues will continue to consume oxygen and produce CO_2. The result is a rise in venous CO_2, accompanied by a drop in venous oxygen saturation.[37] The degree of hypercapnia seen in this study is well beyond that which would be expected in the setting of vigorous muscle contraction alone.[35] The blood gas data observed here suggest that circulatory function was severely affected by the TASER discharge and this is confirmed by the observed decrease in BP. Similar cardiac effects were also seen by Nanthakumar et al.,[16] who showed a loss of BP in swine during TASER discharge measured by aortic manometry.

Two cardiac markers, CK-MB and TnI, were assayed here to assess myocardial injury. There were no elevations in CK-MB. TnI showed small, nonsignificant elevations in both the experimental and control groups. The induction and prolonged anesthesia sessions (2–3 hours) employed on the first day of the experiment may have evoked cardiac stress that contributed to these minor elevations in TnI.[38] Anesthesia, especially at induction, is a known cardiac stressor, which results in an increased risk of adverse cardiac events.

The present study has examined the effects of the TASER X26 using thoracic discharges with a transcardiac vector in anesthetized healthy swine. It does, however, have some limitations. (1) The number of animals used was relatively small but was counter-balanced by the high inter-animal reproducibility of the results. (2) For ethical reasons, ketamine/xylazine anesthesia was used in this swine model. Anesthesia precludes pain perception, which is one of the two principal effects of TASER discharges in conscious humans. Pain perception would undoubtedly alter some of the responses reported here. (3) Only one vector of discharge (transcardiac) was utilized. Alternate discharge vectors may result in greater or lesser myocardial capture.[16] (4) In the field, TASERs are used to subdue combative individuals who are usually in a state of greatly increased sympathetic activity and, in many cases, are under the influence of alcohol or other drugs, which may alter the thresholds for dysrhythmia and for pain. Under those conditions, the effects of TASER discharge might deviate considerably from those seen here.[11] (5) Only two 40-second discharges were used here. These lengthy discharges may have contributed to the incidence of VF, but Webster et al. used 5-second discharges and still observed VF.

The results of this study are in accordance with other published animal studies[14,16] that have used standard, law enforcement-grade TASER X26 devices to study effects in swine. However, they are at variance with those obtained using custom-built TASER-like devices.[4,11] In this swine model, lengthy thoracic discharges from a TASER X26 produced a reversible cardiorespiratory dysfunction which, when coupled with intense muscle contractions, resulted in severe acidosis, tachycardia, hypotension, and sometimes fatal VF. The cardiac capture and VF reported here may be facilitated by the vector of the current, the proximity of the emitting probe to the heart, or the temporal relationship of the discharge pulses to the vulnerable phase of the heart. This model of thoracic TASER discharge indicates that risk of cardiac dysrhythmia exists when the heart is interposed between the darts.

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Editor's Note: Due to a transcription error, the discussion for this paper by Frederic J. Cole, Jr., MD and others will not appear.