Repeated Thoracic Discharges From a Stun Device

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Background: Little objective laboratory data are available describing the physiologic effects of stun guns or electromuscular incapacitation (EMI) devices, but increasing morbidity and even deaths are associated with their use. We hypothesized that exposure to EMI discharges in a model animal system would induce clinically significant acidosis and cardiac arrhythmia.

Methods: Ten Yucatan mini-pigs, six experimental and four sham controls, were anesthetized with ketamine, xylazine, and glycopyrrolate. Experimental pigs were exposed to two 40-second discharges from an EMI device over the left thorax. Electrocardiograms, troponin I, blood gases, and lactate levels were obtained pre-exposure, at 5, 15, 30, 60 minutes, and at 24, 48, and 72 hours postdischarge.

Results: No acute or delayed cardiac arrhythmias were seen. Heart rate was not affected significantly (p > 0.05). A subclinical increase in troponin I was seen at 24 hours postdischarge (0.040 ± 0.030 ng/mL, p > 0.05). Central venous blood pH (7.432 ± 0.014) and pCO2 (36.1 ± 0.9 mm Hg) were not changed significantly (p > 0.05) during the 60-minute postdischarge period. A moderate significant increase in lactate occurred in the 5-minute postdischarge group (4.9 ± 0.3 mmol/L, p = 0.0179). All blood chemistry and vital signs were normal at 24, 48, and 72 hours postdischarge.

Conclusions: Although significant changes in some parameters were seen, these changes were small and of little clinical significance. Lengthy EMI exposures did not cause extreme acidosis or cardiac arrhythmias. These findings may differ from those seen with other EMI devices because of the unique MK63 waveform characteristics or to specific characteristics of the model systems.

Key Words: Electromuscular incapacitation, stun gun, swine, cardiac effects, metabolic acidosis, respiratory acidosis, arrhythmia, ventricular fibrillation, ketamine, xylazine, electrocardiogram, troponin I, lactate, MK63, stun baton, heart, blood pressure, creatine kinase-MB isoform, pH, potassium, creatinine.

daily and that thousands are exposed annually, creating a growing public health concern.\(^\text{14}\) Despite the increasing usage of EMI devices, there is no consensus in the medical literature regarding the safety and type of injuries produced by EMI. Many of the initial studies on stun devices were performed using the much less powerful first- or second-generation devices.\(^\text{15}-\text{18}\) The current peer-reviewed literature on fourth-generation EMI devices is slowly emerging, but many of the results are conflicting. Some studies show no evidence of acute arrhythmia\(^\text{10},\text{19}\) in swine and no acidosis or hyperkalemia in healthy human volunteers.\(^\text{18},\text{20}\) Others indicate the potential for the development of significant acidosis\(^\text{21}\) or arrhythmia.\(^\text{22},\text{23}\) Such conflicting results have made it difficult to arrive at a consensus regarding the need for treatment or monitoring of exposed individuals.

As a result of the increasing usage and deployment of EMI devices, a growing number of individuals are presenting with injuries related to their exposure to these devices and a growing number of morbidities and mortalities are being observed. We hypothesize that the time-varying DC current utilized by certain EMI devices may produce significant myocardial injury, acute arrhythmia, acidosis, electrolyte or biochemical abnormalities. We have developed a model system to study the effects of EMI devices in anesthetized miniature swine.

**MATERIALS AND METHODS**

**Animals and Groups**

Three- to 4-month-old Yucatan mini-pigs (Sinclair Research, Columbia, MO) weighing between 16 and 33 kg were used. The experimental group (80-second thoracic discharge) and the negative (or sham control) group, were comprised of six and four animals, respectively. Previous studies in this field have not included sham controls and used only intranimal baseline data as control data. As a result, the data from those studies have been somewhat difficult to interpret. Preliminary experiments showed that, in view of the small magnitude and high reproducibility of the physiologic changes that occur, it would not be necessary to use more animals simply to improve the chances of finding statistical significance when such differences would not possess clinical significance. The Institutional Animal Care and Use Committee was used as the power source.

Animals were sedated with intramuscular 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. During EMI discharge and for all subsequent monitoring, animals were anesthetized with ketamine and xylazine (Ketaset; Fort Dodge Animal Health, Fort Dodge, 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. During EMI discharge and for all subsequent monitoring, animals were anesthetized with ketamine and xylazine (5.6/0.8 mg/mL) in sterile saline instilled intravenously using an infusion pump (Flogard 6200; Travenol, Deerfield, IL) through a 21-G or 23-G cannula placed into an ear vein at a rate of 3 mL/hr/kg (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 to 1.0 mL of sprayed 20% benzocaine (Hurricane; Beutlich Pharm., Waukegan, IL). Breathing was controlled (15 breaths per minute; tidal volume = 10 mL/kg; min volume = 150 mL/kg). Animals were maintained in dorsal recumbence for all electrical discharges and monitoring procedures. At the conclusion of each monitoring session from which animals were to recover, 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. The primary local electrical injury anticipated with these waveforms was membrane electroporation, particularly of nerve and muscle.\(^\text{7}\) This effect is sensitive to the presence of lipids or highly lipid-soluble agents such as isoflurane, halothane, or barbiturates. These anesthetics may act to artifactually reverse electroporation effects generated in this experimental system. Ketamine and xylazine have some lipid character, but less than isoflurane or halothane, so they are preferred anesthetics for this study. The ketamine/xylazine combination used here has been shown to be an effective general anesthetic in swine\(^\text{24},\text{25}\) and our data confirm this (see below).

**Test Device**

Anecdotal reports from the MK63 manufacturer (Aegis Industries, Bellevue, ID) indicated that discharges from the device caused severe pain and complete voluntary muscle incapacitation. The MK63 stun baton and specifically the component containing the electronic circuitry for generating EMI discharges (e-pod) was studied. The e-pod was incorporated into a custom-made laboratory apparatus fashioned from 1.25- and 2-inch diameter polyvinyl chloride (PVC) pipe and weights (Fig. 1). This apparatus was held vertically by clamping the 2-inch PVC pipe to a heavy stand and was assembled such that the 1.25-inch diameter PVC pipe contained the e-pod and this pipe could slide freely up and down within the larger pipe while maintaining uniform downward force resulting from a final total mass of 1.5 kg for the e-pod/pipe assembly. A 12.0 VDC, 800 mA power supply was used as the power source.

**Experimental Setup and EMI Discharge**

While in dorsal recumbence, all four limbs of the animal were restrained to the table. The e-pod was placed over the head of the animal with the shock electrode parallel to the cardiac axis from the base of the heart to its apex. Based on our preliminary experiments using a broad range of discharge times, we concluded that 80-second discharges would be feasible and that any ill effects that might occur would most likely be seen with such a prolonged discharge. Discharges performed for longer times resulted in effects very similar to those seen with 80 seconds, so longer discharges offered no advantage here. The e-pod
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 (during 10 seconds) were administered between the 40-second discharges.

Cardiac rhythm was evaluated and monitored continuously during anesthesia using a five-lead electrocardiogram (EKG) and monitor (Datex Instruments, Helsinki, Finland) at each experimental time point; 10- to 15-second tracings were printed and retained. EKGs were also recorded during the discharge. There were eight time points at which central venous blood was drawn from the pre caval venous complex, and vital signs (tissue oxygen saturation, heart rate, and blood pressure) and additional EKGs were recorded. The sampling time points were pre-discharge (time 0); 5, 10, 15, 30, and 60 minutes; and 24, 48, and 72 hours postdischarge. Animals were euthanized according to American Veterinary Medical Association standards after the 72-hour time point by switching the anesthesia to 5% inhaled isoflurane and subsequently injecting 3 mol/L KCl into the heart.

Immediately after drawing, 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 a variety of parameters including pH, pCO2, bicarbonate, lactate, potassium, TnI, and creatinine. Blood samples were stored on ice for a maximum of 2 hours, centrifuged (3000× 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 (ELISAs).

**Serum Myoglobin and CK-MB Determination**

Plasma or serum myoglobin, TnI, and CK-MB have been shown to be useful in evaluating possible cardiac muscle damage usually as a result of myocardial infarction. 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. As a result, all three of these markers were studied 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 ELISAs (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

Each of the animals described above were studied for all EKGs and blood chemistry. To simplify comparisons, wherever possible the values for each of these parameters were graphed during the entire time course studied including immediately pretreatment (0 time); 5, 10, 15, 30, and 60 minutes; and 24, 48, and 72 hours postdischarge. All data points represent means ± SEM. Normal values were drawn from published data for mini-pigs, full-sized swine, or humans in that order of preference based on data availability and reliability.24,25,34–39

Parametric statistics including analysis of variance (ANOVA), paired or unpaired t tests, followed by Tukey’s or Student-Neuman-Keuls posttests were employed to compare quantitative data and groups. Trends were evaluated using linear or nonlinear regression. The experimental groups were compared against their baseline for each parameter to assess whether changes from baseline were significant. In addition, the experimental and control groups were compared with each other (Prism and InStat, GraphPad Software, San Diego, CA).

RESULTS

Vital Signs Were Moderately Altered by EMI Discharge

Heart rate (Fig. 2) in experimental animals showed a significant (p < 0.05) decrease from baseline (117 ± 3 bpm at t = 0) at 5 minutes postdischarge (104 ± 4 bpm). It returned to baseline levels at subsequent time points. No acute changes in pulse oximetry were observed at any time. Tachycardia was not seen in response to EMI discharge.

Fig. 2. Heart rate before EMI discharge and during the 72-hour time course after EMI discharge. Heart rate (mean ± SEM) showed an initial decrease 5 minutes after discharge but was maintained between 100 and 120 bpm at all time points in the experimental group. None of these variations were statistically significant nor were there any significant differences between the control and experimental groups (one-way ANOVA).

Blood pressure (Fig. 3) showed minor fluctuations during the 60-minute postdischarge time period (systolic = 126 ± 3 mm Hg, diastolic = 53 ± 2 mm Hg). The systolic blood pressure decreased during the initial 60-minute monitoring period reaching a nadir at 30 minutes (115 ± 8 mm Hg). This decrease in systolic blood pressure was not significant (p > 0.05) compared with the baseline value (132 ± 10 mm Hg) in the experimental group. Blood pressure did not become elevated or show any other changes suggesting pain perception.

No Evidence of Acute Arrhythmia or Myocardial Injury was Found

Rhythm strips taken before, during, and after discharge of the MK63 over the thorax showed no acute changes in cardiac rhythm at any time (Fig. 4). EKGs show continued regular ventricular contractions throughout the EMI discharge. No arrhythmias were observed in any animal at any time point. Mean CK-MB levels (Fig. 5) were not significantly affected in the experimental group as compared with controls or with experimental baseline values. CK-MB levels were not elevated in any of the individual experimental animals. Mean TnI values (Fig. 6) increased at the 24-hour time point in negative controls (0.023 ± 0.019 ng/mL) and experimental animals (0.040 ± 0.031 ng/mL). The observed increase in experimental animals, however, was not statistically significant when compared with that of controls (p = 0.695) or with baseline levels for the experimental group (0.000 ± 0.000 ng/mL). A TnI value of 0.040 ng/mL represents the upper limit of normal.40 The largest value of TnI observed in an individual experimental animal was 0.190 ng/mL at 24 hours postdischarge. TnI in this animal returned to baseline at 48 hours.
No Clinically Significant Acidosis was Seen With EMI Discharge

Central venous blood pH (Fig. 7) showed an initial decrease after EMI discharge at the 5-minute time point. This change (7.45 ± 0.03 to 7.39 ± 0.02) was statistically significant ($p < 0.05$), although the observed values were still within normal ranges. Central venous blood pH then returned to baseline during the 60-minute monitoring session. Control animals had a significantly higher pH during the initial 60-minute time period. A slight acidosis was noted only in one animal (pH 7.32) at the 5-minute time interval. All other animals maintained blood pH at or above normal levels for the duration of the experiment.

Blood pCO$_2$ was not significantly changed by EMI discharge. A small increase in pCO$_2$ was seen at 5 minutes (39.2 ± 2.8 mm Hg) postdischarge. This change was not significantly different ($p > 0.05$) from the baseline value of 36.6 ± 2.7 mm Hg. The pCO$_2$ returned to baseline during the 60-minute monitoring period. One animal had pCO$_2$ levels exceeding normal (pCO$_2$ = 49.6 mm Hg) at the 5-minute postdischarge time. The levels for this animal then returned to normal subsequently during the 60-minute time course.

Bicarbonate levels decreased at 5 minutes postdischarge (24.3 ± 0.6 mmol/L) from baseline values (25.2 ± 1.1 mmol/L), but this was not a significant change from baseline ($p > 0.05$). This decrease contrasted to a small increase in the control group. There was no significant difference when comparing controls with experimental animals during the initial 60-minute period (one-way ANOVA, $p > 0.05$). All bicarbonate levels were within the normal reference range. One animal in the experimental group had a bicarbonate level of 20.6 mmol/L before EMI exposure. The bicarbonate level in this animal rose to normal levels after exposure.

**Fig. 4.** Representative EKGs from predischarge through EMI discharge (A) and through 72 hours postdischarge (B). EKGs show continued regular ventricular contractions throughout the EMI discharge. No evidence of acute arrhythmia was detected during, immediately postdischarge, or at later time points postdischarge. No arrhythmias were observed in any animal at any time point. Note that there is little noise in the bottom tracing (lead aVR) in (A) and that sinus rhythm is maintained throughout the discharge.
Lactate values showed a small increase at the 5-minute time point (4.89 ± 0.7 mmol/L). This increase was not statistically significant ($p > 0.05$) from baseline values (3.7 ± 1.3 mmol/L). The experimental group showed significantly higher levels of lactate when compared with controls during the initial 60-minute monitoring period (one-way ANOVA, $p < 0.05$). Lactate values were below baseline values at 24 hours and were similar to controls. Two of the experimental animals had elevated baseline lactate levels (8.13 and 7.43 mmol/L). These were peak values seen for these animals and they decreased to normal baseline levels during the initial 60-minute postdischarge period. The other four experimental animals had normal starting lactate levels and showed slight increases after EMI exposure, which then returned to baseline.

**EMI Discharge Did Not Affect Electrolyte Levels**

Potassium and creatinine levels were not affected by EMI discharge. Potassium values (Fig. 8) increased slightly after EMI exposure in all animals during the initial 60 minutes (peak value = 3.6 ± 0.1 mmol/L at $t = 60$ minutes). These increases were not statistically significant ($p > 0.05$) compared with baseline values (3.5 ± 0.1 mmol/L). All values were within normal limits for both of these parameters at all time points in all animals. Creatinine values (Fig. 9) did not change significantly after EMI discharge. At no time did creatinine values exceed normal levels in any animal (range 0.6–1.2 mg/dL).

**EMI Discharge Did Not Significantly Affect Serum Myoglobin**

Mean myoglobin levels in the experimental group were increased when compared with the control group at all time points except 72 hours, but these differences were not significantly different (one-way ANOVA, $p > 0.05$). Mean myo-
Cardiac Effects

Case reports, autopsies, and retrospective analysis have found EMI discharge to be associated with fatal ventricular fibrillation in humans, although the frequency of this complication is extremely low.10–13,41,42 In the present study, no changes in cardiac rhythm were seen even after lengthy EMI exposures. At no time in the 72-hour monitoring period did any animal die. The experimental animals maintained a mean heart rate that decreased slightly from but returned rapidly to baseline values. Other studies in anesthetized swine have reported that TASER X26 discharges resulted in acute onset of tachycardia.21 This effect was also reported in studies of reported that TASER X26 discharges resulted in acute onset of tachycardia in this experimental model suggests a deep plane of anesthesia that suppresses pain or may indicate that the MK63 device evoked less pain in this model than did the TASER X26 in anesthetized swine or conscious humans. In any case, the MK63 device did not appear to directly interrupt or capture cardiac rhythm.

Alternatively, sudden deaths associated with TASER discharges in humans may result from direct or indirect damage to the myocardium, which then leads to delayed arrhythmia.10–12 Two cardiac markers, CK-MB and TnI, were assayed in the present study to assess myocardial injury. There were no elevations in CK-MB after 80-second MK63 discharges and TnI showed small but insignificant rises. TnI is released from cardiac myocytes26–32 when their cell membranes are damaged. Free TnI then diffuses into the interstitial space and eventually into the blood.43 The high sensitivity and specificity of commercially available assays have made TnI the gold standard for detecting myocardial injury. Release of TnI from both human and swine cardiac myocytes peaks 18 to 24 hours after the injury and then gradually decreases to normal during the course of the next several days.13,44

For the i-STAT TnI assay, the cutoff value for the upper limit of normal is 0.030 μg/mL, which represents the 97.5th percentile of healthy individuals without heart disease (iSTAT manufacturer manual, Abbott Laboratories, Abbott Park, IL). Based on subsequent evaluation of a large number of patients, Apple et al.49 have determined the value for the 99th percentile of healthy individuals to be 0.040 ng/mL. This value is based on complication rates from patients presenting with symptoms of acute coronary syndrome. The TnI value considered diagnostic of myocardial infarction is higher than the upper limit of normal and is determined by each individual clinical laboratory.45 There are no published guidelines that establish levels of clinically significant TnI levels in swine.

At its peak, the mean TnI value in the present study reached the human upper limit of normal (0.040 ng/mL) at the 24-hour monitoring point in the experimental group. At this time point, two animals (out of six) showed elevated TnI levels (0.190 ng/mL and 0.050 ng/mL). For both animals, TnI returned to the baseline level of 0.000 ng/mL at the subsequent (48 and 72 hours) monitoring time points. TnI also increased in one control animal at the 24-hour time point (0.080 ng/mL) but values similarly returned to baseline at the next monitoring time.

The time-related pattern and magnitude of TnI increases seen here were very different from that seen in humans or swine with myocardial injury.44,45 In models of myocardial ischemia, TnI values peak at 18 to 24 hours and remain elevated for days to weeks thereafter. Because TnI values here decreased to zero within 48 hours, it is unlikely that these elevated TnI values signify myocardial injury such as that seen in human heart attack or swine models of myocardial ischemia. The induction and prolonged anesthesia sessions (2–3 hours) employed on the first day of the experiment may have caused some degree of cardiac stress that contributed to the TnI elevation seen here. Anesthesia, especially at induction, is a known cardiac stressor that results in an increased risk of adverse cardiac events.46

Blood Gas Data

In rare circumstances, humans subjected to EMI discharges have died within 24 hours of discharge exposure. It has been postulated that such “in-custody deaths” may result from cardiac instability as a result of EMI-induced lactic acidosis.42,47 No evidence of severe acidosis was seen in the
There were minor physiologic fluctuations in acid-base status, but none of these changes were statistically or clinically significant. Similarly, the changes in pCO₂, lactate, and bicarbonate showed only small, insignificant (p < 0.05) changes that correlated with the observed changes in blood pH.

The results of the present study are largely at variance with those of Jauchem et al., which examined the effects of discharges from a TASER X26 in swine. In that study, severe acidosis (pH < 7.0) was seen immediately after EMI exposure and this was accompanied by dramatic hypercapnia (pCO₂ > 100 mm Hg) and lactate elevation (>15 mmol/L). There are, however, a number of methodologic differences between the present study and that of Jauchem et al., which may have contributed to the discrepancies between the studies. Third, different anesthetic agents and methods of physiologic support were used. Jauchem et al. did not mechanically ventilate the intubated animals during the study, whereas the swine in the present study were ventilated while anesthetized, except during the 80-seconds EMI discharge. As a result, it is unclear what role respiratory depression from the propofol/telazol anesthesia and butenorphine analgesia or the lack of respiratory support in the Jauchem et al. study may have played in the reported acid-base abnormalities.

The animals used in the present study did not show any acute or delayed changes in electrolytes. Potassium levels can be used to assess the extent of skeletal muscle injury and also the potential for electrolyte-associated cardiac arrhythmia. In the present study, no significant changes in electrolytes were seen. Jauchem et al. reported significant acute increases in potassium levels. Again, differences in experimental design or devices studied may have contributed to the discrepancies in electrolyte levels seen in these studies.

In summary, no evidence of acute arrhythmia, myocardial damage, severe acidosis, or electrolyte/biochemical abnormalities were seen in the present study. In this swine model, prolonged discharges from the MK63 device produced no significant or harmful physiologic changes. Because previous animal studies of the TASER X26 showed some dramatic physiologic changes, the present findings may be a result of the unique waveform and pulse power generated by the MK63 device, differences in the electrode spacing for the MK63 as compared with the TASER X26, or differences between the model systems. Further studies are needed to distinguish among these possibilities, to evaluate the short- and long-term physiologic effects of these devices, and to elucidate the mechanism by which these devices trigger EMI. This knowledge will be instrumental in developing future guidelines and treatment protocols for the growing number of individuals exposed to EMI.

**Study Limitations**

For ethical reasons, anesthesia (ketamine + xylazine) was used in this swine model. Anesthesia precludes pain perception, which is one of the two principal effects of EMI discharges in conscious humans. Pain perception would undoubtedly alter some of the responses seen here. The number of animals used here was also limited, but this was counter-balanced by the high animal-to-animal reproducibility of the results. Finally, in the field, EMI devices are used to subdue combative individuals in a state of greatly increased sympathetic activity. In many cases, these persons are under the influence of alcohol or other drugs. Under those conditions, the effects of EMI discharge may vary from those seen here.

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**References**
