Neuromuscular Effects of Stun Device Discharges

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Submitted for publication January 8, 2007

Background. Stun guns or electromuscular incapacitation devices (EMIs) generate between 25,000 and 250,000 V and can be discharged continuously for as long as 5 to 10 min. In the United States, over 200,000 individuals have been exposed to discharges from the most common type of device used. EMI devices are being used increasingly despite a lack of objective laboratory data describing the physiological effects and safety of these devices. An increasing amount of morbidity, and even death, is associated with EMI device use. To examine this type of electrical injury, we hypothesized that EMI discharges will induce acute or delayed cardiac arrhythmia and neuromuscular injury in an animal model.

Methods. Using an IACUC approved protocol, from May 2005 through June 2006 in a teaching hospital research setting, 30 Yucatan mini-pigs (24 experimentals and 6 sham controls) were deeply anesthetized with ketamine and xylazine without paralytics. Experimentals were exposed to discharges from an EID (MK63; Aegis Industries, Bellevue, ID) over the femoral nerve on the anterior left hind limb for an 80 s exposure delivered as two 40 s discharges. EKGs, EMGs, troponin I, CK-MB, potassium, and myoglobin levels were obtained pre-discharge and post-discharge at 5, 15, 30, and 60 min, 24, 48, and 72 h (n = 6 animals)and 5, 15, and 30 d post-discharge (n = 6 animals at)each time point). Skin, skeletal muscle, and peripheral nerve biopsies were studied bilaterally. Data were compared using one-way analysis of variance and paired *t*-tests. *P*-values <0.05 were considered significant.

Results. No cardiac arrhythmias or sudden deaths

¹ To whom correspondence and reprint requests should be addressed at Department of Trauma, Rm. 1300, Stroger Hospital of Cook County, 1900 West Polk St., Chicago, IL 60612. E-mail: rwalter@cookcountytrauma.org. were seen in any animals at any time point. No evidence of skeletal muscle damage was detected. No significant changes were seen in troponin I, myoglobin, CK-MB, potassium, or creatinine levels. There were no significant changes in compound muscle action potentials (CMAP). No evidence of conduction block, conduction slowing, or axonal loss were detected on EMG. M-wave latency (M_{lat}, ms), amplitude (M_{amp}, mV), area (M_{area}, mV-ms), and duration (M_{dur}, ms) were not significantly affected by MK63 discharge compared with contralateral or sham controls. F-wave latency (F_{lat} , ms), a sensitive indicator of retrograde nerve conduction and function, was not significantly affected by MK63 discharge compared with contralateral or sham controls. No significant histological changes were seen at any time point in skeletal muscle or peripheral nerve biopsies although mild skin inflammation was evident.

Conclusions. There was no evidence of acute arrhythmia from MK63 discharges. No clinically significant changes were seen in any of the physiological parameters measured here at any time point. Neuromuscular function was not significantly altered by the MK63 discharge. In this animal model, even lengthy MK63 discharges did not induce muscle or nerve injury as seen using EMG, blood chemistry, or histology. © 2007 Elsevier Inc. All rights reserved.

Key Words: electromuscular incapacitation; stun device; EID; swine; metabolic acidosis; respiratory acidosis; dysrhythmia; ketamine; xylazine; EKG; EMG; electromyography; combined muscle action potential; troponin I; lactate; MK63; stun baton; heart; blood pressure; CK-MB; pH; potassium; creatinine.

INTRODUCTION

Electrical discharges may produce a wide spectrum of injury [1]. The manifestations of electrical



injury depend on the waveform characteristics of the current applied (AC, DC, mixed, strength, duration, frequency) and its anatomical location and path through the tissues of the body [2-4]. Effects may include skin burns, neuromuscular incapacitation, skeletal muscle death, cardiac arrhythmia, osteocyte and osteoblast death, and blood vessel endothelium dysfunction [3-8]. Electromuscular incapacitation (EMI) devices use high voltage (20 to 250 kV), low frequency (10 to 100 Hz), time-varying amperage (up to 18 A) DC current to produce pain and strong muscle contractions resulting in the incapacitation of volitional movement. The utility of these effects in personal defense and law enforcement has led to the proliferation of EMI devices for use as an alternative to lethal force. EMI devices have been shown to be very effective when used to incapacitate combative individuals while reducing risk to officers, suspects and bystanders [9]. All EMI devices generate time varying DC current with waveforms that are similar but distinctive to each specific device. However, the immediate effects and safety profile of these discharges on living organisms are poorly understood [10 - 12].

More than 100 fatalities have been reported to be associated with EMI use in the United States for the 2001–2004 period [9]. This growing list of fatalities has drawn a great deal of public attention and raised questions about the safety of EMI devices and their potential complications, especially their association with fatal ventricular arrhythmia [10-13]. More than 100 types of EMI devices are marketed to law enforcement agencies around the world to subdue combative subjects and are used increasingly by private citizens for personal protection. Despite the increasing usage of EMI devices there is no consensus in the medical literature regarding the safety or type of injuries produced by EMI. Many of the initial studies on stun devices used the much less powerful first or second generation devices [14–17]. 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 [10, 18] in swine and no acidosis or hyperkalemia in healthy human volunteers [17, 19], whereas others indicate the potential for the development of significant acidosis [20] or arrhythmia [21, 22].

As a result of the increasing use 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 timevarying DC current used by certain EMI devices may produce significant neuromuscular and/or myocardial injury which can result in neuromuscular dysfunction, acute dysrhythmia and even death. We have developed a model system to study the effects of EMI devices in anesthetized miniature swine and report the neuromuscular effects of exposure to discharges from this device here.

MATERIALS AND METHODS

Animals and Groups

Three to 4-mo-old Yucatan mini-pigs (Sinclair Research, Columbia, MO) weighing between 13 and 33 kg were used. Animals in the experimental groups received 80 s discharges over the anterior thigh (n = 24). Animals in this group were divided into two groups, a short-term subgroup (up to 72 h post-discharge) and a long-term subgroup (5, 15, and 30 d post-discharge, n = 6 in each group). Immediate effects from electroporation were expected during the 60 min period post-discharge. Acute effects from electroporation and joule heating should be seen during the 5 d postdischarge period. In humans exposed to high voltage or lightning discharges, neuromuscular symptoms continue to manifest over weeks to months after exposure [4]. If longer term effects occur with this device, they might be seen within the subsequent 30 d. Since no previous short- or long-term follow-up studies had been performed on the neuromuscular effects of stun devices, we determined that a 30 d follow-up would provide adequate opportunity for the development of delayed injury. The negative or sham control group was divided similarly and was comprised of six animals. This project was reviewed and approved by the IACUC for the Hektoen Institute for Medical Research, LLC.

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) 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 21G or 23G cannula placed into an ear vein at a rate of 3 mL/h/kg (16.8/2.4 mg/kg). Animals were intubated using cuffed endotracheal tubes (5.0 to 6.5 mm; Rusch; Kernen, Germany) after anesthetizing the larynx with 0.25 to 1.0 cc of sprayed 20% benzocaine (Hurricaine; Beutlich Pharmaceuticals, Waukegan, IL). Breathing was controlled (15 breaths per min; tidal volume = 10cc/kg; min volume = 150 cc/kg). Animals were maintained in dorsal recumbency for all electrical discharges and monitoring procedures. At the conclusion of each monitoring session from which animals were to recover, intravenous yohimbine (0.05 to 0.15 mg/kg Yobine; BenVenue Laboratories, 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. This effect is sensitive to the presence of lipids or highly lipid-soluble agents such as isoflurane [7], 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 [23, 24] and our data confirm this (see below).

Test Device

The MK63 stun baton (Aegis Industries, Bellevue, ID) and specifically the component containing the electronic circuitry for



FIG. 1. E-Pod and MK63 lab simulator. The distance between the outer electrodes on the MK63 was 2.1 in. and the distance between the inner electrodes was 0.35 in. The total mass of experimental device was 1.5 kg.

generating EMI discharges (e-pod) was studied. The MK63 was studied as a representative device, which causes electromuscular incapacitation using the usual principles of high voltage and time-varying current to produce EMI. Whether different stun devices with different waveforms have unique or unusual physiological effects is not known. Our goal here was to study the neuromuscular effects of EMI, and then later, study the effects of different waveforms from different devices. The e-pod was incorporated into a custom-made laboratory apparatus fashioned from 1-1/4 in. and 2 in. diameter PVC pipe and weights (see Fig. 1). This apparatus was held vertically by clamping the 2 in. PVC pipe to a heavy stand and was assembled such that the 1-1/4 in. 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 V DC, 800 mA power supply was used as the power source.

Experimental Set-Up and EMI Discharge

EMI devices exert their main effects through the musculoskeletal system potentially causing nerve or muscle damage. These neuromuscular effects have not been studied previously. Torso discharges would make EMG and related studies very difficult whereas limb discharges provide a much more useful anatomical location for studying neuromuscular effects. At present, the relationship between the discharge vector and the proximity of the discharge to the heart is not understood. So stun devices discharged at some distance from the heart, i.e., on extremities, may have cardiac consequences. In addition, some subjects exposed to electrical discharges report subsequent lasting neuromuscular deficiencies. As a result, we examined cardiac function in all animals studied here but focused on neuromuscular function.

While in dorsal recumbency, all four limbs of the animal were restrained with moderate extension to the table. The e-pod was placed over the left anterior thigh inferior to the inguinal ligament and discharges were administered with the electrodes oriented parallel to the femur and thus to the muscle fibers of the underlying quadratus muscle group. The e-pod was discharged continuously for two 40 s intervals separated by a 10 s rest for the experimental group. The ventilator was shut off during the discharges, but spontaneous breaths were permitted. Two ventilated breaths (during 10 s) were administered between the 40 s discharges.

Cardiac rhythm was evaluated and monitored continuously during anesthesia using a 5-lead EKG and monitor (Datex Instruments, Helsinki, Finland) at each experimental time point, 10 to 15 s tracings were printed, and retained. EKGs were also recorded during the discharge. There were 11 time points at which central venous blood was drawn from the pre-caval venous complex, 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, 60 min, 24, 48, 72 h, and 5, 15, and 30 d post-discharge. Animals were humanely euthanized according to AVMA standards after the last monitoring time point by switching the anesthesia to 5% inhaled isoflurane and subsequently injecting 3M 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 h, centrifuged (3000 × g for 15 min at 4°C), plasma and serum aliquoted into 400 μ l 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 due to myocardial infarction [25–31]. 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 h, and may remain elevated for several days. Serum myoglobin becomes elevated within 2 to 4 h 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 h of cardiac injury and remain elevated for 60 to 70 h. Myoglobin and CK-MB can become elevated from non-cardiac related injuries such as chronic muscle disease, skeletal muscle trauma, and renal failure [25, 28, 32]. 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 μ L/well) and CK-MB (25 μ L/well) using solid phase microplate sandwich ELISA assays (Diagnostic Automation, Calabasas, CA). All samples and standards for these assays were performed in duplicate and averaged. Standard curves using 4 to 7 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).

Electromyography

Compound muscle action potential (CMAP) recordings were obtained using EMG (Dantec Instruments, Skovlunde, Denmark) with pediatric Ag/AgCl surface electrodes (Neotrode, Conmed Corp., Utica, NY). Electrodes were placed over the middle of the muscle belly (recording electrode) and on the quadratus tendon (reference electrode) at the knee (8 cm distal to the recording electrode). Stimulatory pulses were delivered cutaneously over the femoral nerve using gold plated electrodes separated by 1 cm. The amplitude of the CMAP was maximized by adjusting the position of the trigger electrodes and the amperage of the stimulating current. The positions of the trigger electrodes, reference, and sensing electrodes were marked using indelible marker so that electrodes could be placed in the same exact positions at each subsequent monitoring session. A grounding electrode was placed nearby and the stimulation current used was 20 mA, which exceeded the amperage needed to achieve a maximal CMAP by approximately 50% [33]. Pumice alcohol pads (Electrode Prep Pads, Professional Disposables, Orangeburg, NY) were used to mildly abrade the skin surface and reduce the impedance and electrode gel (Redux Crème, Parker Labs, Fair Field, NJ) was placed under each electrode. Four or five sequential stimulations and recordings were performed at each time point and the measured values averaged to yield the values reported here. Values were obtained for both the right and left hind legs in each animal. Right leg EMGs served as internal controls for animals in the experimental group.

Tissue Histology

Immediately after euthanasia, tissue biopsies were obtained from the skin at the discharge site, skeletal muscle underlying this site, and from femoral and lateral femoral cutaneous nerves in the sub-inguinal region. Biopsies were also obtained from these tissues in the contralateral unshocked leg. Tissue samples were immersed in 10% buffered formalin and refrigerated until they were processed for paraffin embedding, sectioning, and staining with hematoxylin and eosin. Sections were evaluated for possible pathological changes including membrane disruption, cell swelling, interstitial edema, intracellular vacuolization, loss of banding in skeletal muscle, inflammatory cell infiltration.

Data Reduction and Statistical Analysis

Each of the animals described above was studied for all EKGs and blood chemistry. All data points represent means \pm SEM. Reference or normal values for each parameter were drawn from published data for mini-pigs, full-sized swine, or humans in that order of preference based on data availability and reliability [23, 24, 34–38]. Parametric statistics including one-way or two-way analysis of variance (ANOVA) followed by Tukey's post-tests, paired or unpaired *t*-tests were used to compare parametric data. The experimental groups were compared against their own baseline for each parameter to assess whether changes from baseline were significant. In addition the experimental and control groups were (GraphPad Software, San Diego, CA).

RESULTS

Vital Signs Were Not Affected by EMI Discharge

Heart rate (Fig. 2) was not significantly affected by MK63 discharges in the 60 min post-discharge period



FIG. 2. Heart rate before EMI discharge and during the 30 d time course after EMI discharge. Heart rate was not significantly affected by MK63 discharges in the 60 min post-discharge period, in the subsequent 24 to 72 h, or 5 to 30 d time points.

CK-MB Concentration



FIG. 3. CK-MB concentration during the 30 d time course after EMI discharge. CK-MB concentration (mean \pm SEM) did not rise significantly at any time in either group. The control group showed some variability with time, but the differences between the control and experimental groups were not significant (one-way ANOVA).

or in the subsequent time points (one-way ANOVA P > 0.05). No acute changes in pulse oximetry were observed at any time. Neither bradycardia nor tachycardia was seen in response to EMI discharges. Blood pressure showed minor fluctuations during the 60 min post-discharge time period but no significant change was observed (one-way ANOVA P > 0.05). Blood pressure did not become elevated or show any other changes that might suggest 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 left thigh showed no acute changes in cardiac rhythm at any time (not shown). EKGs show continued regular ventricular contractions throughout the EMI discharge. No arrhythmias were observed in any animal at any time point. Mean CK-MB (Fig. 3) levels were not significantly affected in the experimental group compared with experimental baseline values. CK-MB levels were not elevated in any of the individual experimental animals. When compared over time, CK-MB levels were not significantly different from controls (one-way ANOVA, P > 0.05). Mean TnI values (Fig. 4) increased at the 24 h time point in dedicated controls $(0.023 \pm 0.019 \text{ ng/mL})$ and experimental $(0.023 \pm 0.015 \text{ ng/mL})$ discharge groups. An increase was also seen at the 5 d time point (0.03 \pm 0.018 ng/mL), which was not observed in control animals. These observed increases in experimentals, however, were not statistically significant when compared to controls over the monitoring time course (one-way ANOVA, P = 0.814).

EMI Discharge Did Not Affect Electrolyte Levels

Potassium and creatinine levels were not affected by EMI discharge. All values were within normal limits



FIG. 4. Troponin-I (TnI) values during the 30 d time course after EMI discharge. At the 24 h time point, the TnI value for the control group rose to 0.023 ± 0.019 ng/mL and for the experimental group to 0.023 ± 0.018 ng/mL (mean \pm SEM). TnI returned to baseline values for the control group subsequently, but became elevated again in the experimental group on day 5 (0.030 ± 0.017 ng/mL). This elevation was largely due to a high value in one animal (0.110 ng/mL). None-theless, the mean TnI values at day 5 were not significantly different for the experimental and control groups (one-way ANOVA, P > 0.05).

for these parameters at all time points in all animals. Potassium values (Fig. 5) did not exceed normal limits in any animal at any time point (range = 2.9 to 4.4 mmol/L). Creatinine values (Fig. 6) did not change significantly following EMI discharge. At no time did creatinine values exceed normal levels in any animal (range = 0.6 to 1.3 mg/dL).

EMI Discharge did not Significantly Affect Serum Myoglobin

Mean myoglobin levels (Fig. 7) in the experimental group were increased when compared to the control group at most time points, but these increases were not significantly different (one-way ANOVA, P > 0.05). Mean myoglobin levels did not exceed the upper limit of normal (54 ng/mL). The highest single myoglobin level seen was 131 ng/mL at 48 h after EMI exposure.



FIG. 5. Potassium ion concentrations during the 30 d time course. Potassium levels did not change significantly after EMI discharge. There were no significant differences between the experimental and control groups indicating a lack of significant muscle injury.



FIG. 6. Creatinine concentrations seen during the 30 d time course. Creatinine levels did not change significantly after EMI discharge. There were no statistically significant differences between the experimental and control groups indicating a lack of muscle injury.

All elevated myoglobin values returned to normal levels by the next monitoring interval.

EMI Discharge had Limited Effects on EMG Responses

M-wave CMAP latency reflects nerve conduction rates from the site of stimulation to the muscle belly where the majority of motor end plates are located. Longer latencies indicate slower rates of nerve conduction [33]. M-wave latency (Fig. 8) was not affected in the shocked limb or the contralateral control limb. While an initial increase in values in the experimental group was observed there was no statistical difference (one-way ANOVA, P > 0.05) between internal controls (right leg) and the experimental side (left leg). Additionally, no difference was noted between the dedicated control animals and the experimental limbs (one-way ANOVA, P > 0.05).

M-wave CMAP amplitude reflects the vectorial sum of fields produced by individual action potential con-



Myoglobin Concentration

FIG. 7. Myoglobin concentrations seen during the 30 d time course. Mean myoglobin levels in both groups fluctuated somewhat over the time course, but did not change significantly relative to the baseline for each group.



FIG. 8. M-wave CMAP latency for femoral nerve seen during the 30 d time course. M-wave latency was not significantly affected in the shocked limb or the contralateral control limb.

ducting muscle cells. Only cells with intact membranes that can actively produce ATP are capable of generating action potentials. Therefore, changes in CMAP amplitude can be used to quantify the extent of tissue injury associated with damaged cell membranes. CMAP recordings have previously been used to estimate electroporation injury accumulation in the anesthetized rat hind limb [39]. M-wave amplitude (Fig. 9) showed no significant changes during the 72 h postdischarge period but rose gradually from mean values of 15.3 \pm 1.3 and 16.4 \pm 1.5 mV to 22.1 and 20.8 mV during the long-term follow-up for the control and experimental limbs, respectively. No significant changes were noted in M-wave CMAP amplitude when the values for EMI exposed animals were compared to nonshocked controls (one-way ANOVA, P > 0.05).

M-wave CMAP area represents the total amount of the compound muscle action potential elicited over the



FIG. 9. M-wave CMAP amplitude for femoral nerve seen during the 30 d time course. M-wave amplitude showed no significant changes during the 72 h post-discharge period but rose gradually from mean values of 15.3 ± 1.3 and 16.4 ± 1.5 mV to 22.1 and 20.8 mV during the long-term follow-up for the control and experimental limbs, respectively.



FIG. 10. M-wave CMAP area for femoral nerve seen during the 30 d time course. M-wave area showed no significant changes during the 72 h post-discharge period but rose gradually from mean values of 40.6 \pm 2.7 and 39.4 \pm 3.1 mV-ms to 54.8 and 53.4 mV-ms during the long-term follow-up for the control and experimental limbs, respectively.

several milliseconds after the CMAP depolarization begins until it ends. M-wave area (Fig. 10) showed no significant changes during the 72 h post-discharge period but rose gradually from mean values of 40.6 ± 2.7 and 39.4 ± 3.1 mV-ms to 54.8 and 53.4 mV-ms during the long-term follow-up for the control and experimental limbs, respectively. For internal and sham controls, M-wave area showed little variation during the entire monitoring time course (one-way ANOVA, P > 0.05).

F-wave latency reflects the amount of time that is required for the EMG stimulus to be conducted antidromically or retrograde along the motor nerves until it reaches the spinal cord, where it elicits firing of anterior horn motor neurons. These action potentials subsequently trigger distal muscle contractions in the same muscle or muscle group innervated by the nerve originally stimulated by the EMG. The muscle re-



FIG. 11. F-wave latency for femoral nerve seen during the 30 d time course. Mean F-wave latency decreased by 7% to 14% in the experimental and contralateral control limbs at the 5 min time point, but this difference was not significant. F-wave latency was similar in both limbs at all time points studied.

sponse will be much weaker than the M-wave response and will be seen at a later time as a result of the longer path that the stimulus must travel. It will also reflect the patency of the axons and synapses in the spinal cord proximal to the MK63 discharge. Mean F-wave latency (Fig. 11) decreased by 7% to 14% in the experimental and contralateral control limbs at the 5 min time point, but this difference was not significant (oneway ANOVA, P > 0.05). Additionally, no significant changes were seen when comparing F-wave latency in EMI exposed animals to sham controls. F-wave latency was similar in both limbs at all time points studied. This suggests that MK63 discharges caused no significant damage to the proximal nerve pathways leading to the spinal cord and then back to the quadratus femoris muscle group.

M-wave CMAP duration represents the length of time that the CMAP from the EMG stimulus continued. This may provide further information about possible tissue injury. There were no changes in M-wave duration seen at any time point here (data not shown).

Minor Tissue Alterations Occurred After EMI Discharge

Normal cell and tissue histology (Fig. 12) was seen at all time points (3, 5, 15, 30, d) studied for skeletal muscle and peripheral nerve. No evidence of muscle or axonal cell membrane disruption or tissue necrosis was seen. In some biopsies taken at 72 h post-discharge, minor highly localized inflammatory changes in skin were seen at the discharge site.

DISCUSSION

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 [10-12,40, 41] In the present study, no changes in cardiac rhythm were seen even after lengthy EMI exposures. At no time in the 72 h monitoring period did any animal expire prior to being intentionally euthanized. Other studies in anesthetized swine have reported that TASER X26 discharges resulted in acute onset of tachycardia [20]. This effect was also reported in studies of healthy human volunteers where the response was ascribed to intense pain associated with the discharge [17, 19]. The absence 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.



FIG. 12. Tissue biopsies obtained at 72 h post-discharge. Tissue from the dedicated control animals is shown on the left and from animals exposed to 80 s MK63 discharges is on the right. Normal tissue structure is seen for the skeletal (rectus femoris) muscle and peripheral (femoral) nerve biopsies. No evidence of muscle or axonal cell membrane disruption or tissue necrosis was seen. In some biopsies, some inflammatory cell infiltrate is seen in the dermis of skin obtained from the discharge site.

Alternatively, sudden deaths associated with TA-SER 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 s MK63 discharges and TnI showed small but insignificant rises. TnI is released from cardiac myocytes [25-31, 42, 43] when their cell membranes are damaged. Free TnI then diffuses into the interstitial space and eventually into the blood [44]. 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 h after the injury and then gradually decreases to normal over the course of the next several days [44, 45].

The time-related pattern and magnitude of TnI increases seen here was 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 h and remain elevated for days to weeks thereafter. Since TnI values here decreased to zero within 48 h, 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 to

3 h) used 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 which results in an increased risk of adverse cardiac events [46].

Electrical injuries can cause severe muscle and deep tissue injury by a combination of joule heating and membrane electroporation [1, 4]. Injury to skeletal muscle can result in rises in serum myoglobin, creatinine, and potassium concentrations. It had been previously reported by Ordog et al. [14] that EMI exposure may be associated with rhabdomyolysis, which will also increase these values. Additionally, acute changes in potassium concentration may precipitate fatal dysrhythmia. Jauchem et al. [20], in a swine model of TASER X26 exposure, reported small increases in potassium concentration with no changes in myoglobin with no evidence of acute dysrhythmia. Ho et al. [19] reported no significant changes in potassium or creatinine concentration, with small increases in myoglobin after exposure to the TASER X26 in humans. No significant changes in potassium, myoglobin, or creatinine were observed in this study. Thus it appears from these data that the application of EMI electrical energy does not result in any consistently reproducible biochemical evidence of muscle injury. This conclusion is further supported by the lack of structural changes in our histology specimens.

EMG is a sensitive indicator of neuromuscular dysfunction. No EMG evidence of acute or delayed neuromuscular injury was seen here. This study is the first to look at the effects of EMI electrical energy on neuromuscular function through EMG. The present data show no significant changes in EMG parameters when shocked limbs are compared to contralateral non-EMI exposed limbs. Additionally, no significant differences were seen in EMG parameters between EMI-exposed animals and sham control animals. Thus, it appears that the electrical energy produced by the MK63 does not induce measurable neuromuscular dysfunction. The electrical energy produced by other EMI devices, however, is not uniform and varies from device to device. Other devices, such as the TASER X26, which have a different waveform and energy output, may produce different results.

As a positive comparison, swine exposed to electrical discharges on the thigh from a cardiac defibrillator showed signs of severe neuromuscular injury. Blood levels for myoglobin, creatinine, and potassium were significantly increased (data not reported). These animals also showed gross muscle necrosis and microscopic pathologic changes including severe cell swelling, interstitial edema, and vacuolization were seen in skeletal muscle cells and peripheral nerve biopsies (data not shown). EMG values from these animals showed marked increases in F-wave and M-wave CMAP latency and large (>40%) decreases in M-wave area and amplitude.

The present study has examined the effects of the MK63 stun baton using anterior thigh discharges 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 stun device discharges in conscious humans. Pain perception would undoubtedly alter some of the responses reported here. (3) In the field, stun devices 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 that may alter the thresholds for dysrhythmia and for pain. Under those conditions, the effects of MK63 discharges might deviate considerably from those seen here. We have, as yet, not studied the effects of stimulants in this system. Now that we have characterized the system and established the types of responses that occur, we can proceed to further examine the effects of stimulants. Lakkireddy et al. [18] were the first to report on the effects of cocaine on TASER discharges in swine and Nanthakumar *et al.* [21] have similarly reported on the effects of epinephrine.

In summary, no evidence of acute or delayed arrhythmia, myocardial damage, electrolyte abnormalities, or neuromuscular dysfunction were seen in the present study. In this swine model, prolonged discharges from the MK63 device produced no significant or harmful physiological changes. Since previous animal studies of the TASER X26 showed some dramatic physiological changes [20, 21], the present findings may be due to the unique waveform and pulse power generated by the MK63 device, to differences in the electrode spacing for the MK63 compared with the TASER X26, or differences between the model systems. Further studies are needed to distinguish among these possibilities 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.

We conclude that, within the limitations of this study, discharges from the MK63 administered on the lower extremity do not appear to cause any measurable neuromuscular or cardiac injury in this model. Thus, it appears that EMI can be safely achieved using this device even for lengthy periods without causing significant injury.

ACKNOWLEDGMENTS

The authors thank the staff of the Animal Facility for their assistance and Raphael Lee, M.D., D.Sc. for helpful discussions. They also thank Avocet Polymer Technologies, Plainfield, IL, for their support of these studies.

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