Bioelectromagnetics 30:411–421 (2009)

Electromuscular Incapacitation Results From Stimulation of Spinal Reflexes

Florin Despa,[1] Suki Basati,[1] Zhen-Du Zhang,[1] John D'Andrea,[2] J Patrick Reilly,[3] Elena N. Bodnar,[1] and Raphael C. Lee[1]*

[1]*Electrical Trauma Research Program, Department of Surgery, The University of Chicago, Chicago, Illinois*
[2]*Naval Health Research Center, Brooks City-Base, Texas*
[3]*The Johns Hopkins University, Applied Physics Laboratory, Laurel, Maryland*

Electronic stun devices (ESD) often used in law enforcement, military action or self defense can induce total body uncoordinated muscular activity, also known as electromuscular incapacitation (EMI). During EMI the subject is unable to perform purposeful or coordinated movements. The mechanism of EMI induction has not been reported, but has been generally thought to be direct muscle and nerve excitation from the fields generated by ESDs. To determine the neuromuscular mechanisms linking ESD to induction of EMI, we investigated EMI responses using an anesthetized pig model. We found that EMI responses to ESD application can best be simulated by simultaneous stimulation of motor and sensory peripheral nerves. We also found that application of local anesthetics limited the response of ESD to local muscle stimulation and abolished the total body EMI response. Stimulation of the pure sensory peripheral nerves or nerves that are primarily motor nerves induced muscle responses that are consistent with well defined spinal reflexes. These findings suggest that the mechanism of ESD-induced EMI is mediated by excitation of multiple simultaneous spinal reflexes. Although direct motor-neuron stimulation in the region of ESD contact may significantly add to motor reactions from ESD stimulation, multiple spinal reflexes appear to be a major, and probably the dominant mechanism in observed motor response. Bioelectromagnetics 30:411–421, 2009.

Key words: electronic stun devices; spinal reflexes; electromuscular incapacitation

INTRODUCTION

Electronic stun devices have long been used to inhibit undesirable behavior in domestic animals (website in reference). Such devices include electric fences that keep animals from straying, cow trainers that deter undesirable animal movements in milking parlors, and portable electric stun devices, such as ''cattle prods,'' that are used to control non-compliant animals. ESDs have also been used by law enforcement agencies to prevent violent behavior in people but without lethal force [Kornblum and Reddy, 1981; Ho et al., 2006, 2007]. ESDs use transient high-voltage waveforms that are effective for nerve and muscle excitation. The resultant shock causes muscles to contract uncontrollably, appearing as muscle spasms, that often lead to a temporary muscular incapacitation of the subject with general loss of posture. The voltage and peak current is quite high in commercial ESDs. The most commonly used devices are TASERS, produced by TASER International (Scottsdale, AZ). These products produce 50 kV open circuit voltage, and 3–15 amperes of peak current [Reilly et al., 2009]. However, because the duration of the ESD stimulus is very brief (30–80 μs in TASERs), the conducted charge in each pulse is very small—about 100 μC [Reilly, 2007]. As a consequence, the stimulus is capable of causing widespread neural excitation, while maintaining a very low probability of producing undesired effects [Kornblum and Reddy, 1981; Ho et al., 2006, 2007].

While pain and loss of postural control effects from the use of the ESD are readily observable, the neuromuscular mechanisms linking exposure to the

Grant sponsors: Department of Defense Joint Non-Lethal Weapons Program for research in Universities; Department of the Navy.

*Correspondence to: Raphael C. Lee, Department of Surgery, The University of Chicago 5841 S. Maryland Avenue, Chicago, IL 60637. E-mail: r-lee@uchicago.edu

Received for review 4 August 2008; Final revision received 3 February 2009

DOI 10.1002/bem.20489
Published online 7 April 2009 in Wiley InterScience (www.interscience.wiley.com).

ESD and neuromuscular response have been undetermined. A conducted charge of 100 μC is about 100 times that needed to produce pain in human laboratory subjects, and well above thresholds of motor reactions [Reilly, 1998]. According to many sources [Berenson, 2004; American Civil Liberties Union, 2006; Amnesty International, 2006; Anglen, 2006], a shock of half a second duration from an ESD will usually cause intense pain and muscle contractions. Two to three seconds will typically prevent intentional muscle control during the passage of current, cause the subject to become dazed, and drop to the ground. Knowledge of the bio-mechanism of the muscle incapacitation by electrical stimuli is key to optimizing the effectiveness and safety of ESDs.

Although law-enforcement experience with ESDs points to a very low incidence of serious injury [Kornblum and Reddy, 1991; Seattle Police Department, 2003; Battershill et al., 2004] that probability cannot be zero. Indeed, animals exposed to unaltered TASER stimuli have incurred (albeit rarely) induction of ventricular fibrillation (VF) [Dennis et al., 2007] and cardiac capture with loss of blood pressure [Nanthakkumar et al., 2006; Dennis et al., 2007]. Furthermore, orthopedic injuries have been reported in police trainees exposed to TASER stimuli [Battershill et al., 2005].

The safety of ESDs has been questioned by some organizations [Berenson, 2004; Amnesty International, 2006; Anglen, 2006; American Civil Liberties Union, 2006]. Amnesty International has called for suspending use and an ''independent inquiry. . . by acknowledged medical,'' scientific, legal and law enforcement experts who are independent of commercial and political interests in promoting such equipment [Amnesty International, 2004].

Typically, the uncoordinated muscular activity induced by ESD is a generalized whole-body neuromuscular effect that prevents voluntary actions and results in loss of postural control. This can be assessed by measuring the amplitude of the electromyographic response (compound muscle action potential, CMAP) in muscles of the extremities of the body. Here, we use standard testing procedures in a pig model to prove that this effect can be obtained by direct nerve stimulation with sub-millisecond electrical pulses. Our working hypothesis is that the uncoordinated muscular activity represents the manifestation of multiple spinal reflexes. The present study reveals the bio-mechanism of the temporary muscular incapacitation induced by ESD.

Spinal reflexes are graded behaviors that can result from stimulation of either cutaneous sensory fibers or sensory afferents in muscle and tendon. The strength of the stimulus and consequent degree of neural recruitment, as well as its repetition pattern, determine the amplitude of the response. A cumulative effect in the spinal neural circuitry determines the onset of a complex reflex response that produces a whole-body muscle activity [Rosenthal et al., 1967; Phillips and Porter, 1977]. In addition, previous studies [Alon et al., 1983; Kantor et al., 1994; Reilly, 1998] have demonstrated the existence of neurosensory and neuromotor strength-duration relationships. According to these studies, the biological response thresholds from a pulsed current stimulus converge to a minimum current as the duration is made very long relative to an effective membrane time constant, and to a minimum charge as the duration is made very short [Reilly, 1998].

Based on these observations [Alon et al., 1983; Kantor et al., 1994; Reilly, 1998], we designed specific electrical pulse sequences that enable various levels of whole-body muscle activity. Electrophysiological response is produced by muscle cells when these cells contract and is measured by an electromyography (CMAP). We examine the relationship between the CMAP and nerve composition (sensory/motor fibers), the dependence on the waveform of the electrical stimuli, and the effect of interrupting the transmission of the sensory stimuli from receptors to the spinal cord by a local injection of lidocaine.

MATERIALS AND METHODS

The experiments described in the following were designed to investigate the bio-mechanism of EMI induced by ESD devices. We postulated that the EMI effect is a manifestation of multiple simultaneous spinal reflexes induced by stimulating multiple afferent nerves with non-physiological applied electric pulses applied to a small anatomical region. To test this concept, we conducted the following experiments to measure the compound action potential responses in the extremities of adult anesthetized pigs in response to a standard ESD device (TASER® X26, TASER International) and in response to selective stimulation of well defined peripheral nerves with a power source that can mimic the ESD signal. Second, we determined if the EMI response could be reduced or abolished by interfering with the spinal reflex path.

In Vivo Model

We used anesthetized 60 kg Yorkshire pigs (animal use protocol approved by the University of Chicago Institutional Animal Care and Use Committee) to investigate the biomechanisms responsible for the electrical muscle incapacitation response. This experimental model was selected for the following reasons. In this body size range, the ratio of heart size to body

weight is equivalent to that in humans [Detweiler, 1966]. The coronary artery anatomy resembles that of humans [Pluth, 1983]. Other cardiovascular similarities to humans include: (1) there are few pre-existing collateral coronary artery anastomoses [Kong et al., 1969]; (2) a relatively large coronary artery provides a dominant supply of blood to the posterior heart surface [Christensen and Competi, 1959]; and (3) retrograde conduction appears to occur through an atrioventricular nodule pathway [Bowman and Hughes, 1984a]. Normal pig intracardiac electrophysiological parameters resemble those of man more closely than any non-primate animal [Bowman and Hughes, 1984b]. The pig is susceptible to ventricular fibrillation, whether spontaneous or induced [Verdouw et al., 1983]. This makes it a better model than smaller mammals, which are less vulnerable [Hearse and Sutherland, 2000].

In addition to cardiovascular similarities, the cerebral cortex, spinal cord and peripheral nerves and the muscles, including the myofibrils, and sarcomeres are anatomically very similar to humans. Pig skeletal muscle cells are more similar in physical size and electrical space constant to humans than smaller lab animals. Thus, the sensitivity to electrical signals and the vulnerability of pig skeletal muscle cells to electroporation will be a more accurate representation of the human response.

Anesthesia

Each pig was received in the animal care facility at least 48 h ahead of time, to permit health assessment prior to entering the protocol. Each experiment was initiated with administration of a pre-anesthetic (Atropine 0.04 mg/kg body weight IM) 10–15 min prior to induction of general endotracial anesthesia. A surgical plane of gas anesthesia induction with 2.2 mg/kg each of tiletamine, zolazopam and xylazine (standard Telazol, mixed with 2.5 ml each of sterile water and 100 mg/kg xylazine, dosed at 1 ml/25 Kg IM). Endotracheal intubation, was followed by 1% isoflurane to effect in 100% oxygen mechanical ventilation. Dextrose (5%)-Lactated Ringer solution was infused via an IV catheter (3/4–1 in 20–22 ga. Intramedic polyethylene tubing) placed in the marginal ear vein.

Depth of anesthesia was verified by loss of palpebral reflex (touching the eye to ensure the animal does not blink). A twitch monitor was used to monitor the depth of anesthesia and ensure that electromotor response level remained constant during experiments and was consistent from one animal to the next. Isoflurane administration was titrated between 1% and 1.4% of inhaled gas. A Datex-Ohmeda monitor was used to record heart rate, respiration rate, EKG, body temperature, pulse oximetry, end-tidal CO2, and blood pressure (non-invasive cuff). The corneas were protected with a layer of ophthalmic petrolatum or other suitable ointment. The animal was placed and secured in dorsal recumbence. During each experiment, vital signs were noted continuously in the manner stated above in 15 min intervals before the application of electrical signals and 30 min intervals during the surface myography. The body temperature was maintained at 37 °C using a Bear-Hugger blanket (Arizant, Eden Prairie, MN) and warming pad as temperature support.

All twitch response measurements started 1 h after the induction of anesthesia to allow the effect from the pre-anesthetic to disappear. The experimental data were collected in conditions of constant anesthesia depth, which was measured by twitch monitors and electromyography (CMAP) and maintained by varying the isoflurane level.

Selection of Peripheral Nerves

Both mixed nerves containing sensory and motor nerves and pure sensory peripheral nerves were stimulated to compare the responses [Pasquini et al., 2003]. In the context of this discussion, we refer to a mixed nerve as a peripheral nerve containing both cutaneous sensory and motor nerve axons. Pure motor nerves would contain both efferent and position afferent axons.

The *Femoral nerve* (*FN*) is located just below the inguinal ligament in the proximal thigh and is adjacent to the Femoral Artery. It is a mixed nerve providing both sensory and motor axons to the hindlimb. This nerve is involved in flexions, abductions, lateral rotations of the femur at hip and extends the hindlimb at knee joint. In pigs, the saphenous nerve joins the FN which separates from the fermoral nerve just below the inguinal ligament. Below this separation the FN primarily innervates the hind limb muscles. For purposes of investigating the effects of ESD stimulation of a motor nerve, we stimulated the FN distal to the point of separation from the saphenous nerve at which point it contains mostly fibers to innervate hindlimb muscles.

The *Saphenous nerve* (*SN*) is a cutaneous sensory nerve containing pain, pressure and temperature sensitive afferent nerve fibers. It courses through the medial thigh, hindlimb and foot. SN innervates the skin over medial side and front of knee and patellar ligament, on medial and dorsal side hindfoot and ankle. The SN lies just beneath the skin on the medial aspect of the distal thigh above the knee. It is at this location that the nerve was dissected out, exposed, and stimulated.

The *Ulnar nerve* (*UN*) is a mixed motor and sensory nerve in the distal forelimb before it reaches the

hoof. The UN controls the muscles in the distal forelimb below the knee and muscles in the forefoot. The ulnar nerve lies just beneath the skin on the medial side of the knee in the forelimb. It is at this point that we selected to expose and stimulate the nerves.

Intercostal nerves (*INs*) are positioned at the inferodorsal surface of the ribs and provide efferents and afferent innervation for intercostal muscles and sensory innervation of soft tissues in the region of the nerves' path. As the nerve extends toward the center of the chest over the sternum, it becomes pure sensory and provides sensation to the skin over the sternum. The terminal dermal branches of these nerves were stimulated in the IN stimulation experiments at a location over the manubrium and laterally beneath the rib.

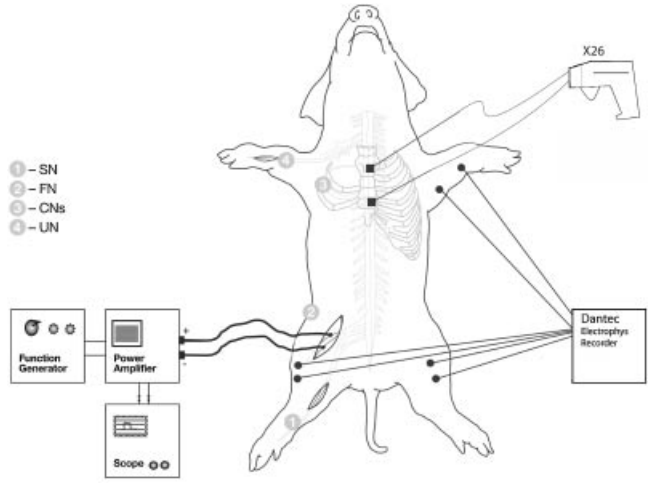

Fig. 1. Experimental setup: the incision sites, placement of the electrical stimulation electrodes, CMAP recording electrodes and twitch monitor for measuring the anesthesia depth.

Stimulation Electronics

Electrical pulses were generated by a function generator (DS345, Stanford Research Systems, Sunnyvale, CA) driving a bipolar power operational amplifier (Kepco BOP 200-1M, Flushing, NY). Stainless steel bipolar surgical forceps electrodes were used to contact the gel. In this way, the electrical pulse was confined to the space between the bipolar electrodes. The amplifier case was grounded to the pig using a standard surgical grounding pad. Possible common mode current passing through the ground pad was monitored to ensure that it was too small to trigger reflexes.

To apply ESD pulses to a specific peripheral nerve, it was surgically isolated. To minimize artifact caused by direct muscular stimulation by the ESD pulse we electrically isolated the nerves from skeletal muscle by inserting a sterile latex sheet between the nerve and surrounding tissue to electrically isolate the nerve. This eliminated direct muscular activation and secondary reflexes. A 1 cm thick layer of 4 M KCL conducting electrode gel was placed in the latex barrier around the nerve at the point selected for electrical stimulation. The bipolar electrodes were inserted into the surface of the conducting gel and maintained at a 4–5 mm distance from the nerve. This obviated harmful effects of electrochemical byproducts of electrode reactions. The electrodes were equidistant from the nerve, and oriented such that the electric field was applied parallel to the nerve.

In the first set of experiments, the CMAP response to a standard TASER X26 ESD as used by law enforcement and military officers was measured. The electrode contacts from the TASER were placed in the skin over the sternum in the cranial-caudal orientation illustrated in Figure 1. CMAP responses were recorded. To determine if the TASER mode of action was mediated by spinal reflexes, lidocaine was infiltrated subcutaneously beneath the TASER electrodes and the intercostal nerves. Sensory innervations to the area were blocked with lidocaine injected beneath the ribs in the mid-axillary line.

In the second set of experiments, peripheral nerves from the right hindlimb (i.e., femoral, saphenous) and right forelimb (i.e., ulnar) were isolated and stimulated as described above. A sequence of electric field pulses was applied directly to the conducting gel around the nerves. Motor response was monitored with an EMG recorder (Dantec® Counterpoint, Dantec, Skovlunde, Denmark) set up to measure compound muscle action potentials in three extremities (hindlimbs and left forelimb) using adhesive gel surface electrodes. The right forelimb remained available for monitoring anesthesia depth by twitch monitoring.

In the third set of experiments, we used similar electrodes and electrical pulses to stimulate a 1 cm partial thickness skin wound over the sternum at the level of 6th rib insertion. The wound oriented in a cranio-caudad fashion and the electric field pulses were applied parallel to the wound. The CMAP activity was recorded, again, in all the pig's extremities.

The effect of various amplitudes, pulse durations, waveforms and frequencies of the electrical stimulus on the EMI response to determine the dose-response functionality was measured. Based on the different stimuli-response data, an optimum signal waveform was selected which was used to demonstrate that the spinal reflex can be shaped to produce EMI. To verify that the stimulated nerve is responsible for the motor reflexes, the nerve was blocked with 1% lidocaine. The lidocaine was injected adjacent to, but not directly into the nerve, at a point proximal to where the nerve was

stimulated. In addition, the lidocaine was also infiltrated around the point of electrical stimulation. Thus, at some point, ~10 cc of 2% lidocaine hydrochloride (4 mg/kg) was injected intramuscularly beneath the stimulating electrode, to determine if it blocked the generalized responses.

Recording Instrumentation

A four channel 5 Gigahertz LeCroy digital oscilloscope (Chestnut Ridge, NY) with 10× or 100× probes (10 MΩ input impedance) was used to record ESD stimulus signals. A Faraday coil was used to monitor current output from stimulus or potential ground loops. To reduce CMAP stimulation artifact, the animal was well grounded using an electrocautery grounding pad. The skin was abraded to remove stratum corneum and 4 M KCl conducting gel was applied to increase the conductivity.

Transcutaneous compound muscle action potentials were measured with a Dantec CounterPoint II clinical electrophysiology system (Dantec, Denmark). Care was taken to standardize the position of the electrodes and the position of the animals. The CMAP recording electrodes were positioned to measure both extension and flexion muscle activity. The Dantec has high-impedance front-end FET amplifiers connected to a 12 bit A/D digitizer. The data analysis including noise filters are software preprogrammed. The Dantec was operated in EMG mode. The adhesive 4 M KCL conducting gel surface electrodes for CMAP recording were placed on skin extensor surfaces of both hindlimb (over biceps femoris muscle) and left forelimb (over the triceps muscle). Before applying the conducting gel electrode to the skin, the skin was abraded to remove the stratum corneum.

In preliminary experiments, the ability to generate consistent surface CMAP recordings was confirmed. In pilot studies, the CMAP signal was verified by selectively eliminating it by administering the neuromuscular blocking drug pancuronium (0.11 mg/kg IV) to prevent muscle excitation coupling. The CMAP occurred approximately 2 to 3 ms following peripheral nerve stimulation. Experiments were repeated in five different animals.

Data Analysis

Each experiment was repeated three to five times. The CMAP data was analyzed by measuring the steady state amplitude of the CMAP response to a specific input stimulus. The steady state CMAP amplitude at 20 Hz stimulation was the graphical average of 10 peaks of the CMAP recording. This graphical average was defined as the CMAP response. Assuming complete independence of each animal, the mean and standard error of the mean were calculated for each stimulation parameter set and plotted in the data shown in the figures.

RESULTS

All animals were well pre-sedated and anesthetized at the beginning of the study protocol. Anesthesia depth was assured by the absence of papillary reflexes or responses to toe pinch and was maintained at the same level through all experiments. There were no burns or other visible signs of thermal injury at the site of electrode contact. In addition, from the anesthesia records we observed that the baseline heart rate and blood pressure for the EMI-stimulated animals were not altered in the exposed animals, nor did they differ from controls. The fluctuations in heart rate and blood pressure over the duration of the experiments are shown in Figure 2. The average decrease (~20%) in time of these parameters is the usual sign of a prolonged anesthesia. The initial expedited decrease within the first hour reflects the influence of the pre-anesthetic, prior to induction of anesthesia. The results suggest a very minimal cardiovascular effect of the peripheral nerve stimulation.

ESD Response

Application of the ESD (i.e., TASER® X26) 5 s stimulus did not result in significant changes either in blood pressure (Fig. 2a) or heart rate. Ventilation was controlled by a mechanical ventilator at a constant rate. The hindlimb CMAP response to ESD stimulation is illustrated in Figure 3. The plot on the left is the CMAP activation patterns from the left (above) and right (below) thigh during ESD stimulation. The frequency of the CMAP spikes was consistent with the 17 Hz firing rate of the ESD. Given that the ESD electrodes were placed in the midline of the sternum, we have no explanation for the asymmetry in the hindlimb CMAP response magnitude. The animals appeared to have symmetrical muscle mass.

The ESD response was abolished by the combination of infiltrating 1% lidocaine beneath the electrical contacts and bilateral intercostal nerve blockade with lidocaine. The hindlimb CMAP responses elicted by the ESD 20 min following the lidocaine block is shown in the plot labeled after lidocaine in Figure 3. No hindlimb response was observed. However, the lidocaine did not interfere with direct intercostal stimulation or stimulation of the thoracoabdominal muscles, indicating that the lidocaine did not interfere with electrical pulse

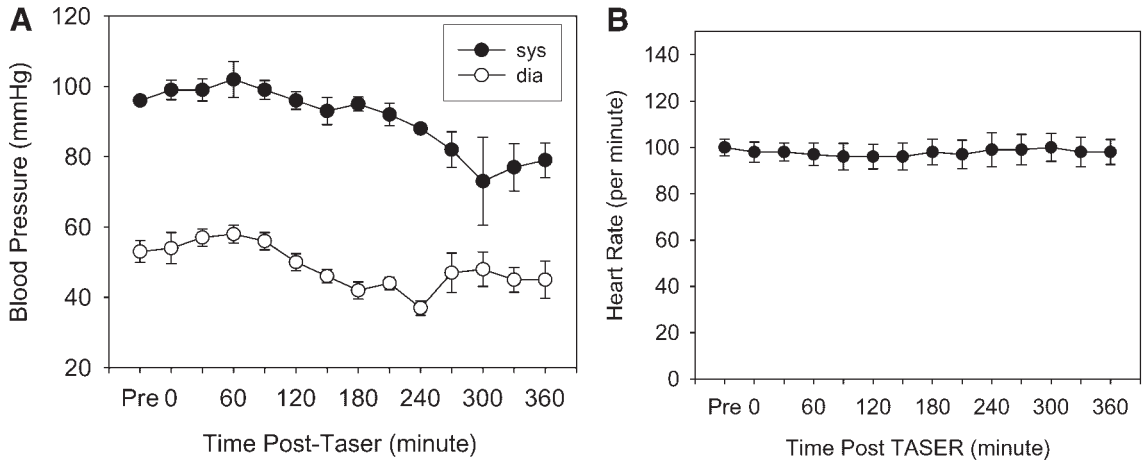

Fig. 2. The systolic and diastolic blood pressure (**A**) and heart rate (**B**) before and after TASER® X26 application. The first data point (pre) represents the measurement before Taser application. The second data point and after were recorded at 0–360 min after taser application. TASER electrodes were located on the right thigh. The pig was tased one round (5 s). Data represent mean and standard error of results from four different animals.

transmission and did not spread widely into thorocoabdominal muscles.

CMAP Responses to Isolated Nerve Stimulation

CMAP values recorded in the right hindlimb (RHL) and left hindlimb (LHL) during a direct stimulation of the right SN with monophasic 10–120 V electric pulses of 20 Hz, 100 μs duration are shown in Figure 4. The intensity of the muscular response increases with the increase of the stimulation amplitude and reaches a plateau at the excitation amplitude of about 100 V. The strength of the stimulus determines the amplitude of the response, as expected. Noteworthy is the difference between the values of the CMAP recorded in the RHL and LHL. Stimulation of the SN would be expected to produce a withdrawal spinal

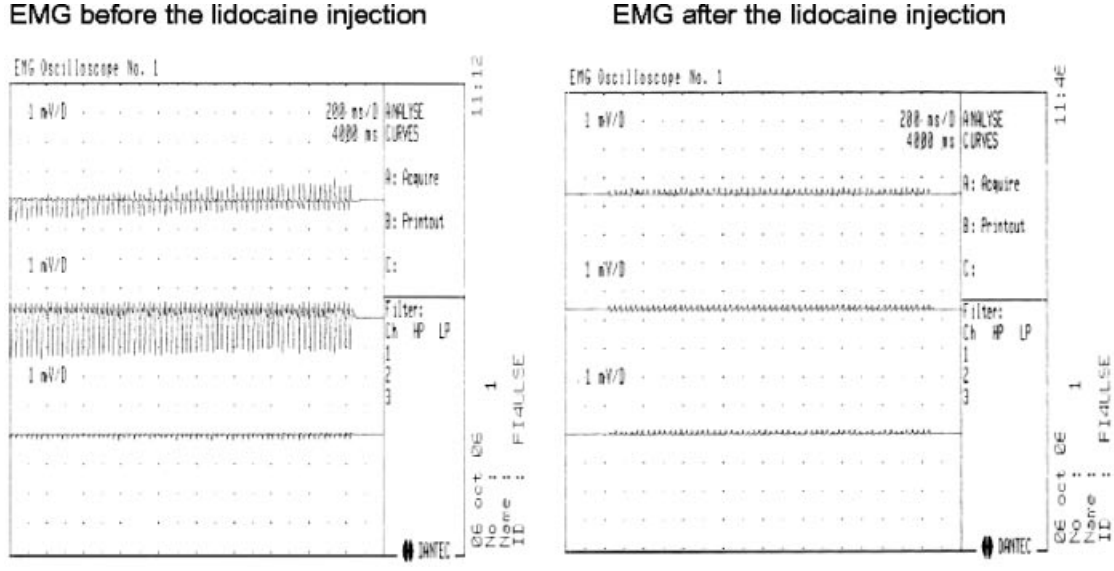

Fig. 3. The effect of lidocaine on TASER® X26-induced EMG recorded on right (top tracing), left (middle tracing) hind limbs and left front limb (bottom tracing). The electrodes were placed in the intercostical space at right side. Lidocaine was injected into the subcutaneous tissue around the electrodes.

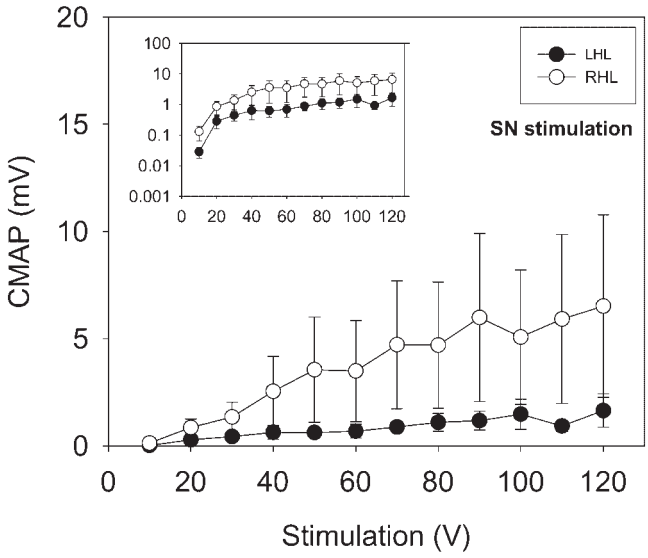

Fig. 4. CMAPs recorded in the right hind limb (RHL) and left hind limb (LHL) during a direct stimulation of the sphenouse nerve (SN) in the right hind limb (RHL) with monophasic electric pulses of 20 Hz, 100 μs width and 10–120 V amplitude range. Each data point represents mean and SEM of four separate measurements from four different animals. The inserted plot is the same data set plotted in log scale for CMAP in *Y* axis.

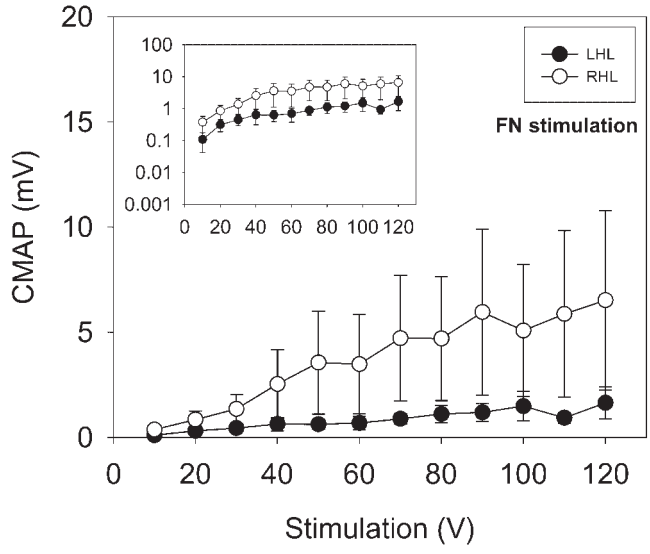

Fig. 5. CMAPs recorded in right hind limb (RHL) and left hind limb (LHL) during a direct stimulation of the femoral nerve (FN) (in RHL) with the electrical pulse sequence described above. Each data point represents mean and SEM of four separate measurements from four different animals. The inserted plot is the same data set plotted in log scales for CAMP in *Y* axis.

reflex, meaning that hindlimb flexion would be expected on the ipslateral side and extension on the contralateral side. In a four legged animal, not much response would be needed in the forelimb. This means that the precise location of the stimulus determines in a fixed way the particular muscle that contracts to produce the reflex response.

A similar behavior is evident in the data of Figure 5 where we display CMAP values recorded in RHL and LHL during a direct stimulation of the right FN distal to the point of departure for the SN. Note that the CMAP recorded in the LHL during the FN stimulation is considerably smaller than those corresponding to stimulation of the SN described above. This again is consistent with a different pattern of spinal reflex. A stretch reflex might be anticipated which would not require much change in position in the ipsilateral leg. Also, from a functional physiological perspective, one would not expect much response in the contralateral hindlimb or the forelimbs.

However, the results of simultaneous direct stimulation of SN and FN from the RHL with a 100 μs electrical pulse are shown in Figure 6. We can see that this stimulation produces spinal cord reflexes that excite muscle activity in both hindlimb and forelimb extremities, although the magnitude of the CMAP LFL response was much less than RHL or LHL responses. The CMAP amplitudes recorded with simultaneous SN and FN stimulation were much greater than with either SN or FN stimulation alone. In addition, the magnitude of the CMAP produced by simultaneous FN/SN stimulation was consistent with that produced by ESD stimulation. A much lower

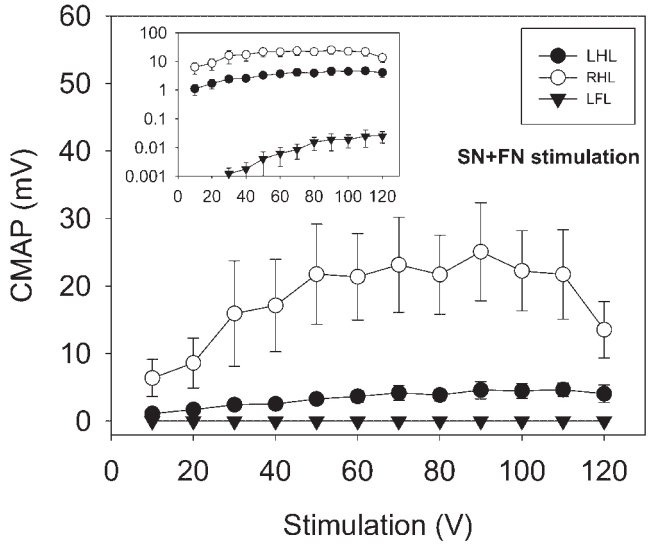

Fig. 6. CMAPs recorded in right hind limb (RHL), left hind limb (LHL) and left front limb (LFL) during simultaneous, direct stimulation of the sophenous nerve (SN) and femoral nerve (FN) from the right hind limb (RHL) with microsecond electrical pulse. Each data point represents mean and SEM of four separate measurements from four different animals. The inserted plot is the same data set plotted in log scale for CMAP in *Y* axis.

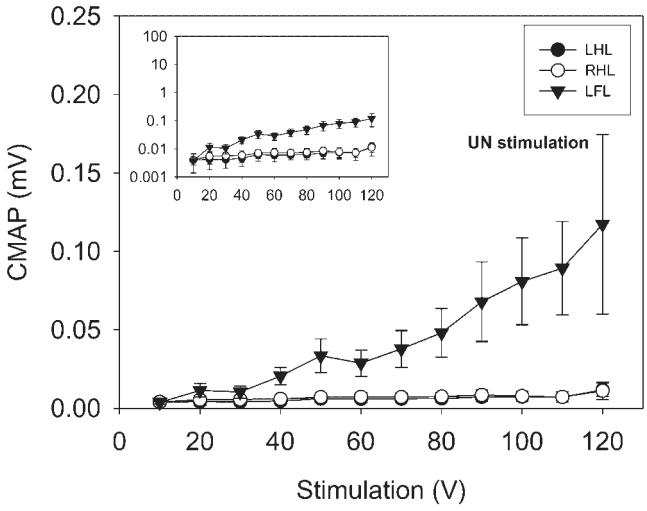

Fig. 7. CMAPs values recorded in right himd limb (RHL), left hind limb (LHL) and left front limb (LFL) during direct stimulation of the ulnar nerve (UN) in the right front limb (RFL) with microsecond electrical pulse. Each data point represents mean and SEM of four separate measurements from four different animals. The inserted plot is the same data set plotted in log scale for CMAP in *Y* axis.

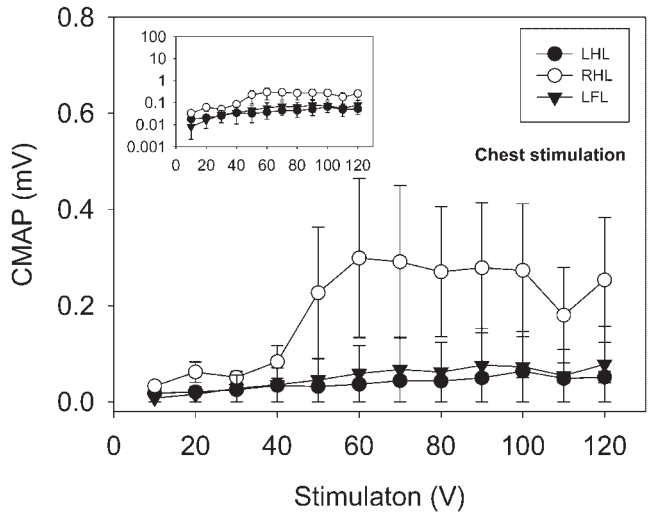

Fig. 8. CMAPs values recorded in right hind limb (RHL), left hind limb (LHL) and left front limb (LFL) during direct stimulation of intercostals nerves (Ins) with microsecond electrical pulse. Each data point represents mean and SEM of four separate measurements from four different animals. The inserted plot is the same data set plotted in log scale for CMAP in *Y* axis.

stimulation voltage of the electrical pulse (~70 V) required to attain the CMAP saturation activity described above. CMAP activity in all extremities was also detected during stimulations of the ulnar nerve (Fig. 7) and intercostal nerves (Fig. 8). However, the CMAP recorded for these stimulations is about one order of magnitude lower.

The CMAP signals in the three extremities monitored during a simultaneous, direct stimulation of SN and FN with electrical pulses widths of 100 μ, 200 μ, and 400 μ are shown in Figure 9A–C. All other parameters of the electrical stimuli were kept constant. We can see that the saturation of the CMAP response depends on the pulse width. The stimulation amplitude required to set a maximum CMAP response decreases to about 60 V for a pulse width of 200 μ and to about 40 V for a pulse width of 400 μ. Similar results were obtained for stimulations of saphenous, femoral, ulnar, and intercostal nerves (not shown).

Each excited neuron will produce a single action potential (AP) on each stimulus pulse as a result of direct stimulation of the affected neuron (i.e., the rate of APs should match the pulse repetition rate of the stimulus, at least up to the 60 Hz repetition rate tested here). After processing these APs through the spinal cord, the result is a train of efferent APs emanating from the spinal cord that nearly matches the input rate.

Muscle fatigue after prolonged stimulation was noticed. In most of the situations presented in Figures 3–8, one can notice a small decrease in the CMAP signal associated with high amplitudes of the stimulus. This effect is consistent across the range of pulse widths used. The effect is possibly due to fatigue ensuing from the long exposure time of the animal.

DISCUSSION

We note that stimulation at one site (e.g., 1 or 2 in Fig. 1) results in motor response distant from the stimulation site (at LHL and LFL recording sites). One of two alternate hypotheses could explain this observation: (1) Stimulation of muscle at the distant site is due to the stimulus energy reaching the remote site passively through the conducting medium of the intervening tissue; (2) the remote stimulation occurs actively through motor neurons that are activated via a spinal reflex. Lidocaine blocks both afferent and efferent nerve response at the stimulation site, but not elsewhere. The fact that the CMAP response largely disappears with the application of lidocaine suggests that muscle activity at remote sites must occur through mechanism (2).

The ESD technology is useful because it leads to electromuscular incapacitation, which implies that the target cannot perform purposeful acts. Because portable battery operated ESDs are low output power devices, the field strengths generated in the body by these devices is only large enough to excite action potentials in nerve and muscle tissue in the anatomical region close the electrical contacts. However, ESDs are known

to produce EMI in regions well outside the distribution of the fields generated by the ESD contact. The results of this study indicate that this near-total body EMI response to ESD contact is caused by ESD stimulation of spinal reflexes.

The fact that peripheral CMAP responses to ESD stimulation in the torso could be abolished by blockage of nerves innervating the region is compelling evidence that peripheral nerve excitation is an essential mechanism of generalized EMI responses. Lidocaine injection will not alter the anatomical electrical field distribution. Therefore, the hindlimb CMAP response to TASER® X26 stimulation on the chest is not due to direct electrical field stimulation of hindlimb muscles. Rather, the hindlimb CMAP excitation was mediated by spinal reflexes.

To further test the spinal reflex hypothesis, we directly stimulated peripheral nerves that were electrically isolated from surrounding muscles by the insertion of electrically insulating barriers. Capacitative coupling of fields across this barrier was negligible because of the conduction gel placed around the barrier and the use of bipolar electrodes separated by 1 cm. By comparing the effects of stimulating primarily sensory or primarily motor nerves to the effect of simultaneous simulation of both, it was clear that the effect of the ESD application could be mimicked only by simultaneous stimulation of both motor and sensory nerves.

Also, we observed that the CMAP response to peripheral nerve stimulation was dependent on amplitude, repetition frequency and pulse width of the field applied. It is clear that direct stimulation of both sensory and motor nerves yielded simultaneous flexion and extension muscular activation in all extremities (Figs. 5–8). This response was consistent with that produced by electomuscular incapacitation devices like the TASER® X26 used in this project. The data suggests that ESD responses are dependent on the nerve (sensory/motor) composition and on the waveform of the electrical stimulus.

The CMAP response to direct nerve stimulation was also inhibited by local anesthetic block of peripheral nerves stimulated by the ESD (data not shown). This interrupted the transmission of the sensory stimuli from receptors in muscles, joints and skin to the spinal circuitry responsible for the motor response. Based on the present results, we can say that the biomechanism of electromuscular incapacitation is based

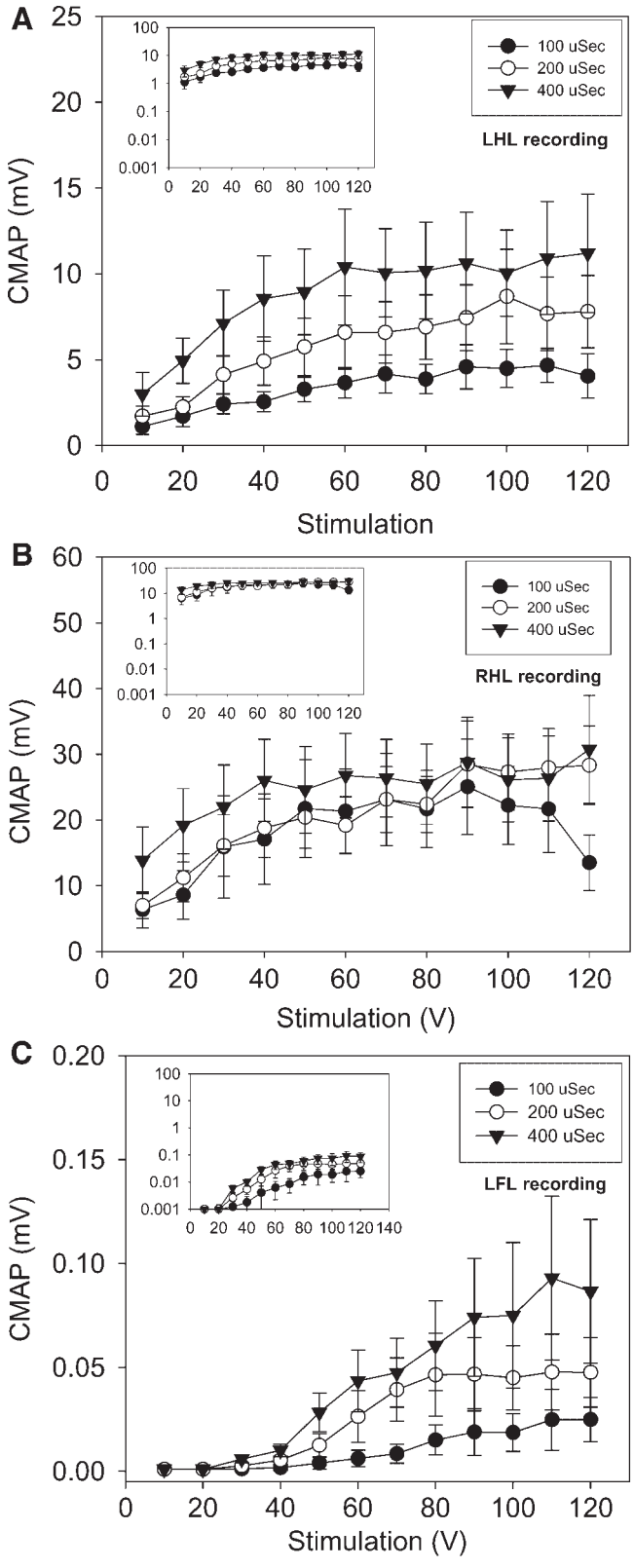

Fig. 9. CMAPs amplitudes recorded in all three extremities (**A**, left hind limb (LHL); **B**, right hind limb (RHL); and **C**, left front limb (LFL)) during a simultaneous, direct stimulation of saphenous nerve (SN) and femoral nerve (FN) with electrical pulses of increased widths (from 100 μ to 200 μ and 400 μ, respectively). Each data point represents mean and SEM of four separate measurements from four different animals. The inserted plot is the same data set plotted in log scale for CMAP in *Y* axis.

on a cumulative effect in the spinal cord processing of simultaneous stimulation of both sensory and motor nerves, and this leads to the onset of a chaotic reflex response. Furthermore, the use of general anesthesia eliminated pain perception in the animals. Therefore, the muscular activity generated by ESD is an involuntary spinal reflex response, and not a response to pain.

Under our experimental condition, it seems clear that the electric fields produced in the body by contact with an ESD are limited to the anatomical region near the electrodes, but the stimulation results in control of the entire target. EMI by stimulation of spinal reflexes can result from one electrode in contact with the body if the tissue field produced by the electrode activates multiple nerve types in the region of the electrode. More studies are required to determine the whole sequence of spastic activities, the initiation and propagation of these spinal reflexes as a function of the position of the stimulus, as well as the duration of the EMI response effect, and conditions for blockage of the response. If these can be clearly understood and harnessed then ESD technology will be more effective and will have much greater value in reducing the collateral morbidity and mortality associated with military conflict.

We postulate that a combination of electrical pulses with optimized physical parameters and durations will elicit an improved muscular incapacitation response. During our studies, we also observed that threshold values of voltage and pulse width are necessary for generating a whole-body muscular activity response. These threshold values depend on the anatomical location of the stimuli. Further investigations should be directed to optimize a sequence of pulses of certain pulse number and duration. The effect of applying such an electrical stimulation in various anatomical parts also needs to be studied.

ACKNOWLEDGMENTS

The authors thank Dr. Craig Wardrip and Dr. Marek Niekrasz and the Animal Resources Center animal care technicians of University of Chicago for critical support of the project.

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