Original Contribution

Echocardiographic evaluation of TASER X26 probe deployment into the chests of human volunteers

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Abstract Several animal studies have shown that the TASER X26 (TASER International, Scottsdale, Ariz) conducted electrical weapon can electrically capture the myocardium when discharged on the thorax. These results have not been reproduced in human echocardiographic studies. A primary limitation of those human studies is that the TASER device was connected by taping the wires into conductive gel on the skin surface of the thorax. This study overcomes those limitations. In this study, a training instructor discharged a TASER X26 into the chests of 10 subjects from a distance of 7 ft so that a 5-second discharge could be administered through the probes as in field exposures. Limited echocardiography was performed before, during, and after discharge. In agreement with 2 prior studies by these authors, the TASER X26 did not electrically capture the human myocardium when used with probe deployment. These data are contrary to animal studies in which capture occurred.

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1. Introduction

The TASER X26 (TASER International, Scottsdale, Ariz) is a conducted electrical weapon used by law enforcement to control violently resisting suspects. The device uses a small electrical current (net current over one second of 1.7 mA) to depolarize afferent sensory neurons and efferent motor neurons causing pain and involuntary muscle contraction. The current is delivered in pulses at 19 pulses per second causing subtetanic contractions. There has been controversy in the lay press because these devices have been temporally associated with some in-custody deaths. Critics have cited animal studies as evidence that these devices can electrically capture the myocardium and induce ventricular arrhythmias.

Several animal studies have shown that the TASER X26 can electrically capture the myocardium when discharged on the thorax. In Nanthakumar et al [1], capture occurred in 78% of thoracic discharges. There was one episode of ventricular fibrillation in this study, but only after infusion of epinephrine. In Walter et al [2], capture occurred in 100% of thoracic discharges. There was one episode of ventricular fibrillation in this study. These results have not been reproduced in human studies. In Ho et al [3], 37 subjects had a 15-second TASER X26 exposure to the chest after a maximal exercise regimen. Mean heart rates were 86, 153, 140, and 115 pre-exercise, post-exercise, during-TASER X26 exposure, and post-TASER exposure, respectively, as determined by echocardiography. In half of the subjects, sinus rhythm was apparent by echocardiography. In a second study, 34 subjects had a 10-second exposure in the cardiac axis. Mean heart rates were 106, 123, and 94...
2. Methods

This was a prospective, nonblinded study of human subjects. The institutional review board at Hennepin County Medical Center (Minneapolis, Minn) approved the study. Subjects provided informed consent and completed a medical screening questionnaire that was reviewed by a study physician. There were no specific exclusion criteria. Subjects were given a TASER X26 as compensation for their participation.

The subjects were a convenience sample of law enforcement officers receiving an exposure as part of a training course. Enrollment in the study was not a course requirement. Subjects were provided face, neck, and groin protection. Clothing was removed from the thorax (except for a sports bra in the 1 female). A training instructor discharged a TASER X26 into the chest of the subjects from a distance of 7 ft. The instructor cantered the device to achieve a probe placement of upper right chest to lower left chest (except in 3 subjects in whom a normal shot orientation was performed). This was to attempt to place the probe in the cardiac electrical axis (training at TASER International wanted to see how difficult such placement would be in field conditions). The device was modified to only deliver one single pulse that discharged the cartridge but did not deliver a shock to the subject. The subject was then laid supine on a training mat. The cartridge was disconnected from the original device and connected to a standard TASER X26. A programmable logic controller (Allen-Bradley MicroLogix 1500, Maple Systems, Inc, Everett, Wash) was used to control the timing of the device. An emergency physician expert in ultrasonography used a Sonosite (Bothell, Wash) M-Turbo portable ultrasound device with a P21x 5-1 MHz 21-mm broadband phased array probe to obtain a parasternal long axis view through the anterior leaflet of the mitral valve in the continuous M-mode to determine heart rate and rhythm before, during, and after the discharge. E and A waves in this mode were used to determine sinus rhythm. The E wave corresponds to the mitral valve opening with atrial contraction. The device was discharged for 5 seconds. After the 5-second discharge, the probes were removed and the wounds were dressed.

Data were entered into a Microsoft Excel spreadsheet (Microsoft, Redmond, Wash) and then exported to STATA 10.0 (Stata Corp, College Station, Tex). Descriptive statistics were used.

3. Results

Eleven subjects were enrolled. No subjects were excluded based on the review of their medical screening questionnaires. The first subject was excluded from the analysis because the device malfunctioned and delivered only a one-second discharge. Ten subjects completed the testing. The median age was 30.0 years, with a range of 32 to 48 years. Ninety percent of the subjects were male. Their demographic data are presented in Table 1. A commercial skin resistance analyzer (Omron Fat Loss Monitor HBF-306, Omron Healthcare, Inc, Bannockburn, Ill) was used to determine body fat percentage. Body mass index (BMI) was based on stated heights and weights. The median BMI was 27.5 (interquartile range, 25.9-29.0; range, 23.8-35.2). There was no syncope during the exposures. There were no significant adverse outcomes reported. A few subjects had minor bleeding at the probe sites that was controlled with mild pressure and band-aids. The probe placements are shown in Fig. 1. In 3 subjects, the device was deployed normally (ie, vertical hand-grip position). In the remaining subjects, the instructor cantered the device to attempt to achieve the cardiac electrical axis orientation. In all subjects except one, the rhythm was determined to be sinus rhythm. In one subject, the ultrasonographer was unable to get the necessary view owing to motion of the subject before the 5-second discharge was complete. The median heart rate before the discharge was 87.5, with a range of 72 to 118 (mean, 91.0; 95% CI, 81.1-100.9). During the discharge, the median heart

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (y)</th>
<th>Sex</th>
<th>BMI</th>
<th>Body fat</th>
<th>PMH</th>
<th>Medications</th>
</tr>
</thead>
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</table>

PMH indicates past medical history; ND, not determined.
rate was 78, with a range of 55 to 143 (mean, 95.8; 95% CI, 70.2-121.4). After the discharge, the median heart rate was 82.5, with a range of 55 to 130 (mean, 85.7; 95% CI, 71.7-99.7). The results are presented in Table 2. A sample tracing is shown in Fig. 2.

4. Discussion

Conducted electrical weapons, such as the TASER X26, are used by law enforcement to control violently resisting subjects. The devices discharge electrical charge into the subject that leads to depolarization of afferent sensory neurons, causing pain, and efferent motor neurons, causing involuntary subtetanic muscle contraction and therefore incapacitation. Excitable tissues in the body can be modelled as resistors and capacitors in series. Because of this, changes in transmembrane electrical potential occur over time. If the threshold to initiate an action potential is not reached before the discontinuation of the stimulus, then the transmembrane potential will decay to its starting point. This physiology establishes a strength-duration relationship. A stimulus must be of sufficient strength and duration to initiate an action potential. Shorter duration stimuli require higher strengths to initiate an action potential. Different tissues have different strength-duration relationships. Sensory and motor neurons can be depolarized by small intensity, short duration stimuli, whereas cardiac cells require higher intensity, longer duration stimuli [5]. This is the principle behind the low-amperage, short pulse duration of the TASER devices. Longer pulse durations would increase muscle torque, but would decrease cardiac safety [6]. Using electro-stimulation theory, Ideker and Dosdall [5] determined that the TASER device current was 2.63 SDs less than the current needed to stimulate an ectopic beat, and 29 times less than the current needed to cause ventricular fibrillation. This calculation is in good agreement with the experimental findings of other authors [7].

There has been controversy in the lay press and medical literature about the use of conducted electrical weapons and temporally associated in-custody deaths. Amnesty International claims that more than 300 deaths have been caused by these devices [8]. Critics have cited papers such as those by Walter [2] and Nanthakumar [1], both swine studies, as evidence that these devices can capture the human myocardium and therefore induce lethal ventricular arrhythmias, contrary to theoretical modeling and the animal studies of other authors. However, human studies have not replicated these findings [3,4]. Several studies in the medical literature have shown no evidence of arrhythmias by electrocardiogram when performed immediately after the TASER device exposure [9-11]. These studies have been limited in that electrical interference from the TASER device during discharge precludes capturing a surface electrocardiogram during the exposure. In addition to prospective human studies, there have been retrospective studies. In a study by Ho et al [12], use of a TASER device occurred in only 30% of the in-custody deaths and was not associated with instant collapse in any of the deaths. In a study by McManus et al [13], there were no deaths, dysrythmias, or cardiac complaints. In a study by Bozeman et al [14], 99.5% of subjects had no injuries or mild injuries. There were no deaths and no cardiac events. Lastly, in a study by Swerdlow et al [15], 95% of the presenting arrhythmias in the in-

<table>
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<th>Table 2</th>
<th>Echocardiographic results during exposure</th>
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<td>------------</td>
</tr>
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Fig. 1  Probe shot distribution.
custody deaths reviewed were pulse-less electrical activity or asystole. These are generally not considered electrically induced arrhythmias.

In this study, to overcome the problem of electrical interference, echocardiography was used to determine rhythm and heart rate during the exposure. Two previous studies by Ho et al [3,4] used a similar methodology and found no evidence of myocardial capture. These studies were limited, as previously discussed, by the taping of conducting wire into conducting gel on the skin surface. Although the extent to which this affects the results is not clear as the conductive gel does significantly reduce the skin resistance, it is a valid criticism. In this study, probes were shot into the skin and the charge was applied through these probes as would be the case in the field. In our subjects, there was no evidence of myocardial capture.

Experience and improving ultrasound technology have enabled us to obtain better images that are more readily
interpretable. In addition, by programming a device to discharge one single pulse, thereby discharging the probes but not any charge to the subject, we have overcome the primary limitation of the previous studies, which was taping the conducting wires into conducting gel on the skin. In this study, the trainers also attempted to place probes in the cardiac axis (right upper chest to left lower chest). It is important to note that this was difficult to achieve even by experts in a laboratory setting and police officers in the field are never taught to canter the device. It is not likely therefore that a suspect would have this probe placement in field conditions. In a law enforcement–reported field-use database maintained by TASER International, probe placements in 4642 top probe hits and 3948 bottom probe hits are distributed as shown in Fig. 3 (misses account for the difference) [16]. The cardiac axis would require a top probe to be in position B1 or C1 and the bottom probe to be in position D2. Based on these field reports, only 4.22% of the top probe hits occurred in position B1 or C1, and only 5.98% of the bottom probe hits occurred in position D2.

There are plausible explanations for the discordance between human and animal studies. Swine, the animal chosen by previous investigators, have a chest shape that is very different from humans. Rather than being dorsi-ventrally flattened, as in humans, the chest in swine is laterally compressed [17]. This could establish very different charge distributions. It is estimated that only 4% to 10% of the surface applied energy in the lowest stimulation threshold location reaches the heart. Most of the current is believed to flow around the chest in the intercostal muscles [5]. Because the swine chest is laterally compressed, forming an extreme triangular shape, this might cause more current to preferentially reach the heart than in the human. There are other significant anatomic and electrical differences between human and swine hearts, including that swine hearts are more susceptible to ventricular fibrillation and that the Purkinje network in swine transverses from the endocardium to near the epicardium, whereas in humans it remains confined to the endocardium [17,18]. In addition, the swine used by some of these investigators tend to be small, approximating the size of children to small adults. Most of the in-custody deaths are in high BMI individuals. In Stratton et al [19], 56% of the subjects were obese. The mean weight was 91 kg and the mean BMI was 30 kg/m² [19]. In a study by Strote et al [20], the mean BMI was 30.8. In a study by Ross and Chan [21], the mean weight was 100 kg. In our current study, the mean BMI was almost 28. These factors may explain why human findings differ so greatly from animal findings.

The in-custody death phenomenon is not a new problem and predates the conducted electrical weapon technology. Wetli and Fishbain [22] published their case series in 1985, greater than a decade before the advent of the modern TASER device. These deaths today occur with and without the use of TASER devices. Studies by Ross and Chan [21], Stratton et al [19], and Ho [12] showed TASER device use in

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**Fig. 2**  A, Sample pre-exposure tracing. B, Sample tracing during exposure. C, Sample post-exposure tracing.
21% to 30% of their in-custody death series. Other factors, such as acute and chronic drug abuse, chronic mental illness, an excited delirium, and a prolonged physical struggle with manual restraint, are associated with these deaths. Although the exact etiology of many of these deaths is indeterminate, it is important to be wary of the difference between temporality and causality in these deaths. The authors have conducted numerous studies on these devices and have not determined a link between the devices and the in-custody death phenomenon. We recommend other investigators attempt to replicate our findings.

The primary limitation of this study is the number of subjects. In addition, only a 5-second discharge was used. The latter is less likely to be a true limitation. The TASER device has 53 milliseconds between pulses. According to Ideker and Dosdall [5], the cardiac cell transmembrane electrical potential should return to within 0.0001% of its initial value between pulses. Therefore, theoretically, there should be no additive effect [5]. In addition, human studies have not shown evidence of cardiac injury by troponin even in long duration exposures (up to 15 seconds) [23,24].

5. Conclusion

In agreement with 2 prior studies by these authors, the TASER X26 did not capture the myocardium when used with probe deployment, even in the cardiac electrical axis. These data are contrary to animal studies in which capture occurred. We recommend other investigators replicate our findings.

Acknowledgments

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References


