Serum Troponin I Measurement of Subjects Exposed to the Taser X-26®

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Abstract

The Taser® is a high-voltage, low-amperage conducted energy device used by many law enforcement agencies as a less lethal force weapon. The objective of this study was to evaluate for a rise in serum troponin I level after deployment of the Taser® on law enforcement training volunteers. A prospective, observational cohort study was performed evaluating serum troponin I levels in human subjects 6 h after an exposure to the Taser X-26®. Outcome measures included abnormal elevation in serum troponin I level (> 0.2 ng/mL). There were 66 subjects evaluated. The mean shock duration was 4.36 s (range 1.2–5 s). None of the subjects had a positive troponin I level 6 h after exposure. It was concluded that human volunteers exposed to a single shock from the Taser® did not develop an abnormal serum troponin I level 6 h after shock, suggesting that there was no myocardial necrosis or infarction.

Keywords: Taser; cardiac; troponin I; CED (conductive energy/electrical device); myocardial necrosis; less lethal weapon

Article Outline

Introduction

The Taser® is a weapon that delivers high-voltage, low-amperage electricity in a pulsed waveform and is representative of the group of less lethal weapons known as conducted energy devices (CEDs). Although generally regarded as safe, there is little research on the effects of these devices in the medical literature despite deaths reported in proximity to CED use. These deaths have drawn wide media and lay public attention, raising questions regarding the overall safety of CEDs as less lethal devices (**[1]**, **[2]** and **[3]**).

Although deaths have been associated with the device's use (so-called “proximity deaths”), no direct causal link has yet been identified. Recent studies have aimed to identify potential physiologic consequences of a CED application (primarily in animals, but also in human trials) (**[4]**, **[5]**, **[6]**, **[7]** and **[8]**). Because these devices deliver a high-voltage electrical discharge to the body, some have suggested that CEDs may cause cardiac injury that could lead to sudden death (**9**). Animal studies have drawn equivocal conclusions as to the cardiac effect of these devices (**[5]** and **[6]**). Our group has previously reported on the effect of CEDs in human subjects, finding no significant cardiac dysrhythmias immediately after a CED application (**8**). For the current study, we hypothesized that a Taser X-26® discharge would not result in myocardial injury as measured by a rise in the cardiac enzyme troponin I 6 h post-activation in a population of law enforcement training volunteers.

Materials and Methods

Study Design

This is a prospective cohort study performed with San Diego Police and San Diego County Sheriff law enforcement officers undergoing training in the use of the Taser X-26® between December 2005 and June 2006. As a component of training, officers were offered the opportunity to experience the effects of the device. This strictly voluntary exposure was delivered by either firing the Taser® dart at the subject from a distance of 10 feet or attaching the subject to the device with two alligator clips. The maximum duration of shock delivered was 5 s, although one agency allowed that subjects could abort the deployment before the typical 5-s standard firing duration. Duration is the only variable that can be adjusted. Each shock delivers 50,000 volts, 21 milliamps. This is constant and cannot be altered.

We enrolled only subjects who had previously volunteered to experience the Taser® exposure as a part of their training. Those trainees who did volunteer were then approached and asked to participate in our study. Informed consent was obtained from each subject. All subjects were attached to a three-lead monitor both for safety and to determine the duration of the shock. Subject inclusion criteria for study consisted of law enforcement personnel between the ages of 18 and 60 years who were willing to participate. There was only one shock delivered.

Exclusion criteria for our study, but not the voluntary exposure, included individuals who were found to have a dysrhythmia before deployment of the Taser®, known or suspected history of cardiac disease, individuals suspected to be under the influence of drugs, or inability to provide informed consent. The police agencies did not exclude anyone who volunteered to get an exposure.

Demographic data were not obtained from the subjects. The subjects as a group were typical police officers, male and female, who were members of both the San Diego Police Department and the San Diego County Sheriff's Office. The typical class consisted of 20–30 officers, of whom anywhere from 1 to 8 volunteered for exposure. Of those who had volunteered for the exposure, 90% agreed to participate in our study. The main reason given for declining our study was aversion to a blood draw.

This study was approved by the UCSD Institutional Review Board.

Data Collection and Processing

Six hours after application of the CED, a single 5-mL sample of venous blood was obtained from all subjects using standard blood draw technique and analyzed for troponin I levels. Samples from the initial 9 subjects were analyzed using a Biosite (San Diego, CA) “Triage Meter” point-of-care assay. Due to an improved cost profile, all subsequent samples were analyzed at the UCSD Medical Center chemistry laboratory using the Advia Centaur Immunoassay System (Siemens Healthcare Diagnostics, Tarrytown, NY).

Primary Data Analysis

Laboratory results were recorded as positive (troponin I ≥ 0.2 ng/mL) or negative (troponin I < 0.2 ng/mL). After the results were obtained, all blood samples were subsequently destroyed.

The primary endpoint was a positive troponin I. The duration of each CED application was also recorded. Data were entered into an Excel spreadsheet (Microsoft Corporation, Redmond, WA) and confidence intervals calculated.

Results

A total of 66 subjects volunteered and underwent a Taser X-26® shock delivery. A total of 47 experienced a 5-s discharge. No patients were excluded from participation based on an abnormal baseline rhythm strip and none was excluded based on a history of cardiac disease.

The mean duration of discharge was 4.36 s (median 5 s, range 1.2–5 s). Six-hour troponin I results were negative (troponin I ≤ 0.2 ng/mL) in all subjects (95% confidence interval 0–5.4%).

Discussion

Both the San Diego Police Department and San Diego Sheriff's Department purchased the Taser® X26, and it was estimated that more than 1000 units were placed into service by the end of 2006. The training procedure for both agencies includes a multi-day course including how to operate the device, safety, and tactics. Officers are also provided an opportunity to actually experience the deployment of the Taser® but are under no pressure or obligation to do so. No firm numbers were recorded, but in our observation, approximately 10% of all officers participating in the CED training over the study period volunteered to experience the Taser® shock.

The Taser® is designed to be deployed up to 7 meters (21 feet) from the subject. By pulling the trigger, a compressed nitrogen cartridge device deploys two, approximately 2-cm-long barb-tipped darts at 160 feet per second that are attached to the gun by thin, 7-meter (21-foot) copper wires through which the electrical shock is delivered. The Taser® delivers energy as a sequence of dampened sine-wave current pulses, each lasting about 11 ms. This energy is reportedly neither pure alternating current (AC) nor pure direct current (DC), but akin to rapid-fire, low-amplitude DC shocks (**10**). The power output of the device is 26 watts, with an average 2.1 mA of current and a maximum of 50,000 volts. A pulse of 5 s duration is automatically delivered through the wires to incapacitate the subject by causing involuntary tonic-clonic muscular contractions. An officer may repeat the delivery of electricity by pulling the device trigger again. When used in demonstrations, an alligator clip adaptor permits electrical discharge without actual deployment of dart. The manufacturer states that thousands of law enforcement volunteers have received shocks without harm, although many of these uses employed only 0.5-s discharges (**11**).

Although the potential for adverse effects of the Taser® are not well understood, the device generally has been regarded as safe ([5], [11] and [12]). However, deaths have been reported in individuals in the field after a CED deployment. The vast majority of deaths reported in "tasered" subjects are associated with illicit drug use, especially phencyclidine in the 1980s ([13]). There are some reported deaths of "tasered" subjects found not to be under the influence of drugs, although these cases generally involve subjects with other co-morbid factors presenting in a state of excited delirium ([13], [14] and [15]).

There are a number of animal studies investigating the physiologic effects of Taser® and CEDs. A 1989 study on earlier "stun-gun" models (with higher energy output) demonstrated the ability to induce deadly cardiac rhythms, including asystole and ventricular fibrillation, in a swine model ([6]). Subsequent studies directly stimulating porcine hearts using the newer Taser® failed to induce cardiac dysrhythmia ([5]). In a swine model using much more aggressive and prolonged CED exposures consisting of 5 s on, 5 s off for 3 min, Jauchem et al. showed that neither troponin T nor troponin I became elevated when measured at 60 min post-exposure ([4]).

Research on human subjects is more limited. Our group recently reported on human subjects who were monitored electrocardiographically before and after a Taser® deployment. In this study of 120 subjects, there were no significant rhythm disturbances other than an increase in sinus tachycardia after a Taser X-26® ([8]).

The next logical step for us was to assess whether there is any cardiac damage as a result of a CED application. We measured serum troponin I levels 6 h after the shock in our human volunteers. Troponin I is a standard marker with excellent sensitivity and specificity at 6 h for myocardial ischemia, infarction, and necrosis ([16] and [17]). In fact, troponin I is routinely used in emergency departments to assess patients with chest pain for myocardial infarction ([16] and [18]). We hypothesized that if the Taser® is directly injuring heart muscle as a result of electrical current, this should be apparent by 6 h, as the release of troponin I is typically measurable as soon as 4 h after cardiac injury. Our study demonstrates that there is no significant injury to cardiac myocytes measured by troponin I as a result of a Taser® shock.

Manufacturer-sponsored studies have reported similar results to our study. Ho et al. measured troponin I levels in 66 human subjects measured immediately after a 5-s Taser X26® application in resting human subjects, then again at 16 and 24 h after Taser® application ([7]). All values were negative except for one subject who had a slight troponin I elevation at 24 h. This patient had a complete cardiac evaluation including cardiac stress test, which was normal. No cause for the elevated troponin was identified. A repeat level 8 h later was negative. Possible explanations given by the authors for the elevated troponin were laboratory error, delayed physiologic clearance, or idiopathic. The subject suffered no complications ([7]).

Limitations

Our subjects were healthy resting volunteers without coexisting use of stimulant drugs or excited delirium. We also excluded subjects with significant cardiac disease. These are potentially the patients who would be the most likely to show a rise in troponin if one were to occur. However, due to ethical limitations and institutional review board policies, this is an area of study that simply cannot be done in humans at present.

Whereas the majority of our subjects (47 of 66; 71%) were exposed to a full 5-s shock, a small number of subjects did abort the shock after a shorter duration. As we found no abnormal elevation in either group (full 5-s shock or those who stopped before the full 5-s discharge), it is unclear what effect this may have had on our results.

As this was an initial study, we delivered only one shock to each volunteer. This may not always be the case in field deployments. In our study in humans, we found no troponin elevation after a single shock, but the effect of multiple sequential shocks remains to be determined.

Finally, we measured only a 6-h troponin I level and did not measure subsequent troponin I levels. However, as discussed previously, we believe the 6-h assessment was appropriate to assess for direct myocardial injury as a result of electrical current.

Conclusions

In human volunteers who received a single Taser X-26® activation, there was no evidence of myocardial injury as measured by serum troponin I at 6 h post-activation. This finding suggests that there is no cardiac injury in healthy subjects who receive a single 5-s CED shock. This study adds to the growing body of literature on human subjects that assesses the physiologic result of a CED application on human subjects.

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