Sudden Cardiac Arrest and Death Following Application of Shocks From a TASER Electronic Control Device

Douglas P. Zipes, MD

Background—The safety of electronic control devices (ECDs) has been questioned. The goal of this study was to analyze in detail cases of loss of consciousness associated with ECD deployment.

Methods and Results—Eight cases of TASER X26 ECD–induced loss of consciousness were studied. In each instance, when available, police, medical, and emergency response records, ECD dataport interrogation, automated external defibrillator information, ECG strips, depositions, and autopsy results were analyzed. First recorded rhythms were ventricular tachycardia/fibrillation in 6 cases and asystole (after ~30 minutes of nonresponsiveness) in 1 case. An external defibrillator reported a shockable rhythm in 1 case, but no recording was made. This report offers evidence detailing the mechanism by which an ECD can produce transthoracic stimulation resulting in cardiac electrical capture and ventricular arrhythmias leading to cardiac arrest.

Conclusions—ECD stimulation can cause cardiac electrical capture and provoke cardiac arrest resulting from ventricular tachycardia/ventricular fibrillation. After prolonged ventricular tachycardia/ventricular fibrillation without resuscitation, asystole develops. (Circulation. 2012;125:2417-2422.)

Key Words: conducted energy weapon injuries ■ death, sudden ■ heart arrest ■ ventricular fibrillation
barbs in the anterior chest near or over the heart and developed loss of consciousness during or immediately after the ECD shock (Table). First recorded rhythms were VT/VF in 6 and asystole (after >30 minutes of nonresponsiveness) in 1 (Figure). An external defibrillator reported a shockable rhythm in 1, but no recording was made. Except for case 1, all died.

**Discussion**

**New Observations**

This report adds detailed observations on 8 new cases of sudden cardiac arrest/death following ECD shocks to those already in the literature.

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**Table. Summary of the 8 Cases**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, y</th>
<th>Height, Weight, lb</th>
<th>ECD Shock(s), s</th>
<th>Response to ECD Shock</th>
<th>Time to Initial ECG After ECD Shock, min</th>
<th>Initial Recorded Rhythm</th>
<th>Drug Screen</th>
<th>Cardiac Findings at Autopsy</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>48</td>
<td>6 ft 0 in, 155</td>
<td>5, 8, 5</td>
<td>LOC toward end of last ECD cycle</td>
<td>Several</td>
<td>VT/VF</td>
<td>BAC 0.35 g/100 mL; THC present</td>
<td>Survived with memory impairment; normal echocardiogram</td>
<td>Five AED shocks, intravenous epinephrine, and lidocaine eventually restored a perfusing rhythm</td>
</tr>
<tr>
<td>2</td>
<td>17</td>
<td>5 ft 7 in, 170</td>
<td>37, 5</td>
<td>LOC toward end of 37-s cycle</td>
<td>&gt;4.5</td>
<td>VF</td>
<td>Negative</td>
<td>410 g; focal atherosclerosis; plaintiff pathologist: normal; defense pathologist: HCM</td>
<td>3 defibrillating shocks and an additional 3 shocks from a second AED at least 9 min after the collapse failed to resuscitate</td>
</tr>
<tr>
<td>3</td>
<td>17</td>
<td>5 ft 8 in, 115</td>
<td>5</td>
<td>ILLOC</td>
<td>&gt;5</td>
<td>VF</td>
<td>BAC 0.25 g/100 mL; THC present</td>
<td>270 g; normal heart</td>
<td>Asystole developed after the AED shock and then PEA; subsequently, VF recurred and a second AED shock was delivered, followed by asystole/PEA; could not be resuscitated</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>5 ft 10 in, 176</td>
<td>11</td>
<td>ILLOC</td>
<td>−10</td>
<td>Fine VF vs asystole</td>
<td>Gabapentin 31 μg/mL</td>
<td>470 g; 10%–20% narrowing of the LAD; normal histology</td>
<td>Gabapentin taken for seizure disorder</td>
</tr>
<tr>
<td>5</td>
<td>33</td>
<td>6 ft 2 in, 220</td>
<td>13 shocks totaling 62 s in &lt;3 min</td>
<td>LOC toward the end of multiple shocks</td>
<td>−13</td>
<td>Fine VF vs asystole</td>
<td>Gabapentin 31 μg/mL</td>
<td>470 g; 10%–20% narrowing of the LAD; normal histology</td>
<td>Gabapentin taken for seizure disorder</td>
</tr>
<tr>
<td>6</td>
<td>24</td>
<td>5 ft 6 in, 144</td>
<td>49, 5</td>
<td>LOC toward end of 49-s shock</td>
<td>−10</td>
<td>VT/VF</td>
<td>Negative</td>
<td>366.7 g; normal gross and microscopic findings</td>
<td>Said to be breathing initially; could not be resuscitated</td>
</tr>
<tr>
<td>7</td>
<td>16</td>
<td>5 ft 3 in, 130</td>
<td>5</td>
<td>ILLOC</td>
<td>−10</td>
<td>VT/VF</td>
<td>THC</td>
<td>380 g; medical examiner diagnosis: right ventricular cardiomyopathy, disputed by plaintiff’s expert</td>
<td>Six AED shocks for VT/VF resulted in asystole/PEA; could not be resuscitated.</td>
</tr>
<tr>
<td>8</td>
<td>23</td>
<td>5 ft 9 in, 173</td>
<td>21, 7, 3</td>
<td>LOC toward end of 21-s shock</td>
<td>−30</td>
<td>Asystole</td>
<td>BAC 0.111 g/100 mL</td>
<td>400 g; mild interstitial fibrosis of compact atrioventricular node; atrial fibrillation, atrioveolar, and vacuolization of penetrating and branching bundle</td>
<td>Said to be breathing with pulse initially; could not be resuscitated; cardiac pathologist could not determine whether changes contributed to death</td>
</tr>
</tbody>
</table>

ECD indicates electronic control device; LOC, loss of consciousness during/aftew initial shock; VT, ventricular tachycardia; VF, ventricular fibrillation; BAC, blood alcohol concentration; THC, tetrahydrocannabinol, positive screen for marijuana; AED, automated external defibrillator; HCM, hypertrophic cardiomyopathy; PEA, pulseless electrical activity; ILOC, immediate loss of consciousness during/aftew initial shock; and LAD, left anterior descending coronary artery. Heart weight is given in grams. Gabapentin is Neurontin.

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**Published Reports**

The first published report of sudden cardiac arrest after ECD discharge was a letter to the editor about a 14-year-old boy who immediately lost consciousness after a 17-second ECD chest shock. He initially had a pulse and was breathing but 2 minutes after collapse had VF documented in an ECG recorded by paramedics; he was ultimately resuscitated. The accuracy of statements made in this publication was contested, but sworn testimony by a paramedic who witnessed the entire event stated that VF was recorded 2 minutes after the ECD shock.

The second observation was of a 17-year-old man who received ECD applications of 25 and 5 seconds in the anterior
chest. He immediately dropped to the ground and was observed to become cyanotic and apneic. The initial rhythm was asystole recorded >10 minutes after the ECD application. He was eventually resuscitated with hypothermia6 but had memory impairment.

The third publication reviewed the presenting rhythm in 56 sudden deaths temporally proximate to discharge of a TASER ECD, finding VF in 4, but concluding that only 1 could be related to ECD discharge. That individual was a 25-year-old man who received ECD shocks in the anterior chest for 16, 5, and 5 seconds. He immediately lost consciousness, and prompt application of an automated external defibrillator showed VF. After 2 shocks, he could not be resuscitated. The report stated that “…the time course and the electrode location are consistent with electrically induced VF.”7

Animal and Clinical Studies

The concept of cardiac capture by transthoracic electrical impulses in humans was pioneered by Zoll,8 replicated by many others subsequently,9 and is now a standard part of resuscitative equipment. The threshold for transthoracic cardiac electrical capture is 100 microcoulombs,10 which is the output of the TASER model X26.1

Studies in pigs,11–15 sheep,16 and humans17 established that transthoracic shocks from the TASER model X26 or a new prototype ECD18 caused cardiac electrical capture. In addition, porcine research showed that such electrical capture could provoke VF at normal12–15 or higher-than-normal11 TASER model X26 outputs. Vectors encompassing the heart and probes closer to the heart facilitated electrical capture.11,12,15 In 1 study, standard 9-mm probes produced VF in pigs at dart-to-heart distances of 4 to 8 mm.19 In another porcine study testing barbs at 5 locations, the authors found that 5-second shocks from the TASER X26 produced cardiac electrical capture without VF at normal outputs but that VF induction at increased output was initiated when the capture ratio was ≥2:1. Bracketing the heart with darts in a sternal notch to cardiac apex position along the cardiac axis resulted in the lowest safety margin for VF induction. The authors stated that cardiac disease could reduce the VF threshold and provide a substrate for arrhythmia induction and that rapid ventricular capture was the likely mechanism of VF induction.20

In addition to the importance of probe location, longer-duration shocks at normal TASER X26 outputs appear more likely to induce VF.13,14 Although it is possible that body size might influence cardiac capture and development of VF, clearly big people can still develop VF from an ECD shock (see the Table).

Determining whether an ECD shock caused cardiac electrical capture can be difficult because the shock produces electrical interference in the ECG recording. In a clinical study of a new TASER ECD prototype, cardiac electrical capture monitored echocardiographically was shown to occur at 240 bpm in 1 volunteer.18 In addition, a case report about a man with an implanted pacemaker demonstrated cardiac electrical capture at rates exceeding 200 bpm during each of two 5-second TASER model X26 applications that was found when the pacemaker was interrogated.17

Multiple clinical studies have not shown ECD-induced VF in healthy volunteers. But, because of ethical considerations, even those few studies testing actual barbs to the anterior chest and single 15-second exposures21 may not be able to replicate the clinical scenario of a frightened/fleeing/fighting individual.

Several epidemiological studies have not shown a link between ECD shocks and sudden cardiac death.22,23 However, a recent review24 determined that single shocks in healthy people “…could have deleterious effects when used in the field, in particular if persons receive multiple exposures…or present with medical comorbidities.”

Proposed Mechanism of ECD-Induced Sudden Cardiac Arrest

Electrical stimulation can induce VF by causing ventricular capture during the vulnerable period of the T wave of the previous beat or ventricular capture at rates too fast for ventricular activation to remain organized. Rapid pacing also can cause a precipitous blood pressure fall, leading to ischemia. VF by rapid pacing was often the outcome of runaway pacemakers many years ago.25

It is clear from the information cited above that an ECD shock to the chest can produce cardiac electrical capture at rapid rates in animals and humans.11–18 Furthermore, it is clear that VF has been documented as early as 2 minutes after an ECD shock to humans.3,5 What is lacking is the actual ECG recording of VF induction during an ECD shock in humans, a practical impossibility unless it fortuitously occurred in an individual with a recording device already in place. Even then, electrical interference may obscure the recording. However, ECD-induced VT and VF have been clearly and repeatedly shown in pigs.11–15,19 In 1 example, intravenous epinephrine in an anesthetized pig, infused at a concentration that increased the spontaneous sinus rate 50% to replicate the clinical “fight or flight” situation, improved the TASER model X26 electrical capture ratio from 3:1 to 2:1 and resulted in VF induction.12

Thus, from the pig studies, a likely clinical scenario is that ECD induced cardiac electrical capture at rates of 200 to 240 bpm (a 6:1–4:1 ratio), as already shown in humans.17,18 The
increased rate plus sympathetic effects can shorten ventricular refractoriness to permit further ECD-induced rate acceleration that eventually causes VF from the rapid rate or R on T. Because a sharp blood pressure reduction results from the rapid rate, repeated shocks or those exceeding the recommended 5-second ECD discharge can add an ischemic component and would be more likely to provoke VF. Furthermore, on the basis of clinical electrophysiological studies performed over many years, the presence of underlying heart disease or arrhythmogenic drugs would be expected to facilitate VF induction by electrical stimulation. Finally, an individual falling forward and then lying prone may be at even greater risk for cardiac capture since the heart can move closer to the chest wall and hence, be closer to the bars and site of stimulation. The fall might even push the bars into the skin to a greater depth.

Certainly not every in-custody death occurring after ECD deployment is due to the effects of the ECD shock. Restraint asphyxia and the concept of “excited delirium” are among other explanations.26 Excited delirium may be a form of takotsubo cardiomyopathy.27 Considering the fact that extreme sympathetic stimulation likely accompanies most restraint attempts, particularly those with ECD discharge, and the relatively few reported sudden deaths, it seems more logical to conclude that the ECD rather than sympathetic stimulation was responsible for the sudden death.

Alternative explanations such as excited delirium would be more relevant when there was a significant time delay between ECD deployment and loss of consciousness/responsiveness or death.28 However, when loss of consciousness/responsiveness occurs during/immediately after an ECD chest shot, as it did in each of the cases above, and the subsequent rhythm is VT/VF or asystole (if a long time has elapsed without resuscitation) with no other cause apparent, it becomes difficult to exonerate the effects of the shock. It is also possible that combinations exist. For example, prolonged QT interval in takotsubo cardiomyopathy or metabolic changes from prolonged or repeated shocks might predispose to pacing-induced VT/VF.

Several victims were alleged to have structural heart disease (cases 2, 4, 7, and 8) and/or had elevated blood alcohol concentrations (cases 1, 3, 4, and 8). Although sudden death caused by underlying heart disease or alcohol is possible, one would have to postulate that the heart disease or alcohol coincidentally induced sudden loss of consciousness precisely at the time of ECD application. Far more likely is that stimulation from the ECD in the presence of structural heart disease and/or alcohol intoxication induced VT/VF. Clinical electrophysiology studies over many years have established that the presence of structural heart disease facilitates electrical induction of VT/VF, as does alcohol.29

Institutional Reviews

A contemporary review by the National Institute of Justice concluded that the case reported as a letter to the editor was ECD-induced VF and stated that an ECD “may induce rapid ventricular pacing or VT in an individual who appears to be in satisfactory condition…leading to VF after a short delay,” and therefore “use involving the area of the chest in front of the heart area is not totally risk free.”30 The Report of the Braidwood Commission of Inquiry,31 after an ECD-related death recorded on video in the Vancouver, BC, airport, stated, “There is evidence that the electrical current from a conducted energy weapon is capable of triggering ventricular capture…and that the risk of ventricular fibrillation increases as the tips of the probes get closer to the wall of the heart…[I]f a person dies suddenly and from no obvious cause after being subjected to a conducted energy weapon, death is almost certainly due to an arrhythmia.”

Incidence

Sudden death occurs infrequently after ECD deployment, considering the number of ECD applications and the apparently few reported sudden deaths. However, the actual incidence of death when the darts are impaled in the chest is unknown because accurate numerators and denominators are uncertain owing to potential underreporting of total number of sudden deaths (numerator) and the actual number of chest shocks that might cause cardiac electrical capture (denominator). ECD applications without 1 or both probes in the anterior chest would not be expected to influence cardiac rhythm and likely make up a large number of the total applications (denominator) cited. Until a detailed database of ECD deployments and outcomes is created, the exact incidence will remain unknown.

Clinical Implications

It is important to stress that the purpose of this article is not to condemn ECD use by trained professionals. Law enforcement experts must make those decisions, not physicians. Intuitively, one would expect a less lethal weapon to reduce in-custody–related sudden deaths and to be preferable to firearms. Such may not always be the case, however. One study32 noted that the rate of in-custody sudden deaths increased 6-fold and the rate of firearm deaths increased 2-fold in the first full year after ECD deployment compared with the average rate in the 5 years before deployment.

The main purpose of this article is to make ECD users aware that cardiac arrest caused by VF can result from an ECD shock. Users should be judicious in how and when to use the ECD weapon, avoid chest shocks if possible, as TASER International recommended in September 2009, monitor the person after an ECD shock, and suspect this adverse response in any victim who loses consciousness. Users should be prepared to resuscitate, including deployment of an automated external defibrillator if indicated.

Limitations

The incidence of ECD-induced sudden cardiac arrest/death cannot be determined without accurate data compiled in a national registry of ECD deployments and outcomes. Such a registry should also chart precise dart locations and should be administered and reviewed by an independent oversight group.

The major limitation of this study is not having an ECG recording during ECD application, a practical impossibility in the field situation, as noted earlier. The hard facts of each encounter are 2 events separated in time: ECD deployment and the recorded ECG. The explanation of the cause of the
events in between, ie, the loss of consciousness/responsiveness and sudden death, is based on the animal and clinical data detailed above. However, in cases 4, 6, and 8, reports stated that a pulse and/or respirations were recorded initially, which would seem to be incompatible with ECD-induced VF. Yet, the victims were totally unconscious/unresponsive directly after the ECD discharge without any other explanation (no head trauma, seizure activity, etc). Although it may be possible that the sudden loss of consciousness was not due to the effects of the ECD, it is my opinion that finding a pulse (often radial) could have been spurious during the tumultuous event and that agonal breathing could be mistaken for normal respirations. It is also possible that a pulse and respiration were present initially if the first rhythm was VT before VF, as has been found in the ECD animal studies.12 In fact, in 1 porcine study,13 ECD discharge induced a stable monomorphic VT that remained for \( \approx 3 \) minutes before degeneration to VF. An example of recording an initial pulse and respiration immediately after ECD-induced loss of consciousness was given in the published New England Journal of Medicine letter3 in which a paramedic, present during the entire ECD deployment, felt an initial pulse of 100 (counted for 15 seconds) and respirations of 16,5 with VF documented by ECG 2 minutes later. Normal breathing has been documented in pigs and sheep for a minute after the onset of ventricular fibrillation and in humans for at least the first 12–15 seconds.3,33 From these observations, it is clear that continuous monitoring of vital signs and ECG, if available, in an individual unconscious following ECD deployment, should be mandatory.

Conclusions
The animal and clinical data support the conclusion that ECD shocks from a TASER model X26 delivered via probes to the chest can cause cardiac electrical capture. Furthermore, if the capture rate increases sufficiently or if \( R \) on \( T \) occurs, the development of VF, either directly or via a transition through VT, occurs in animals and, in my opinion, in humans as well. How often this happens is unknown. Although it would seem more likely to occur in individuals exposed to potentially arrhythmogenic drugs, in those who have structural heart disease, and after long or repeated ECD shocks, electrophysiological studies in humans clearly show that only 1 or 2 extra stimuli can provoke VT/VF in particularly susceptible individuals.

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Disclosures
Dr Zipes has served (and in the future may serve) as a paid plaintiff expert witness in ECD-related sudden cardiac arrest/death cases. However, that role has provided access to critical and detailed records necessary to determine the potential for ECD-induced sudden cardiac arrest and death. Despite this conflict, the author has attempted to present the salient facts about the cases and to offer scientific evidence, credible argument, and logic to support the conclusions to a reasonable degree of medical certainty.

References
The safety of electronic control devices (ECDs) has been questioned. This article reports 8 cases of TASER X26 ECD–induced loss of consciousness in which first recorded rhythms were ventricular tachycardia/ventricular fibrillation in 6 and asystole (after 30 minutes of nonresponsiveness) in 1. An external defibrillator reported a shockable rhythm in 1 case, but no recording was made. From an analysis of the cases and data from animal and clinical ECD studies, it is the opinion of the author that ECD stimulation can cause cardiac electrical capture and provoke cardiac arrest resulting from ventricular tachycardia/ventricular fibrillation. The cause is probably ventricular capture during the vulnerable period of the previous beat or ventricular capture at rates too fast for ventricular activation to remain organized. After prolonged ventricular tachycardia/ventricular fibrillation without resuscitation, asystole develops. The purpose of this article is not to condemn ECD use by trained professionals but to make ECD users aware that cardiac arrest caused by ventricular fibrillation can result from an ECD shock and to encourage users to be judicious concerning how and when to use the ECD weapon, to avoid chest shocks if possible, to monitor the person after an ECD shock, and to suspect this adverse response in any victim who loses consciousness. Users should be prepared to resuscitate, including deployment of an automated external defibrillator if indicated.
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In the article by Zipes, “Sudden Cardiac Arrest and Death Following Application of Shocks From a TASER Electronic Control Device,” which was published in the May 22, 2012 issue of the journal (*Circulation*, 2012;125:2417–2422), two points are in error.

First, where it is stated that “Individuals were previously clinically healthy males . . .” it was meant that they were clinically healthy from a cardiovascular standpoint without manifest cardiovascular symptoms. As indicated in the table of the original manuscript, autopsy may have shown underlying cardiac disease not clinically manifest. Four were inebriated, as indicated by the drug screen (table), with a history of alcohol abuse; case 3 had an attention deficit disorder, possibly bipolar; case 5 was mentally confused, perhaps due to a post ictal state after a seizure; and case 6 was agitated with a diagnosis of depressive disorder, schizophrenia and medication noncompliance. Second, the first available ECG shown in the figure was a representative ECG strip chosen from those recorded at the time of attempted resuscitation and was not the very first ECG recorded in all cases. Upon comparison of these tracings, there is no material difference in the recorded rhythms. None of these statements change the author’s opinion expressed in the conclusion of the article but are presented for completeness and accuracy.
Correction

In the article by Zipes, “Sudden Cardiac Arrest and Death Following Application of Shocks From a TASER Electronic Control Device,” which appeared in the May 22, 2012 issue of the journal (Circulation. 2012;125:2417-2422), Dr Zipes did not acknowledge the contributions of Atty. John Burton, Dr Kamaraswamy Nanthakumar, Dr John Miller, and Ms Joan Zipes. The current online version of the letter has been corrected.