What is ventricular fibrillation (VF)?

VF is a dysrhythmia (defective cardiac rhythm) of rapid, fibrillary movements of the ventricular muscle. VF does not provide adequate cardiac output to sustain life. Unconsciousness occurs within 5 to 15 seconds (s) and death follows quickly unless a perfusing rhythm is re-established.

What are the common causes of VF?

Sufficiently strong electrical shock delivered to the heart, legal drugs (psychotropic, anti-arrhythmic), illegal drugs (especially stimulants), myocardial infarction, certain genetic heart abnormalities, and electrolyte and acid/base imbalances.

How does VF relate to a TASER® Electronic Control Device (ECD) exposure?

Because a TASER ECD discharges electricity, there has been speculation that the delivered electrical charge from an ECD could cause VF in humans.

What is an evidence-based conclusion with regard to a TASER ECD and VF?

The preponderance of the data, including all of the human studies, suggests that VF is not caused by ECDs in real-world usage. There is no evidence of important electrocardiogram (ECG) changes, or capture (pacing response of the heart to electrical stimulation), and finite element modeling (FEM) does not suggest a current density in real-world use able to induce fibrillation in humans. Also, epidemiological studies do not find that real-world human ECD use causes VF.

Historical Scientific Overview

There is no reliable published peer-reviewed medical, scientific, engineering, or electrical human study that has found to a reasonable degree of certainty that a TASER ECD, or the amount of net electrical charge delivered as by an ECD, can foreseeably cause VF in a field or training deployment.

The evidence:

**Human Prospective Studies**

(Ho 2008³) *Echocardiographic [ECG] Evaluation of Human Transcutaneous TASER® [ECD] Application Along the Cardiac Axis, Human subjects with sternum to cardiac*
<table>
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<tr>
<th>Human Retrospective Studies</th>
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<tr>
<td>apex ECD exposure, sinus rhythm demonstrated in most, and heart rhythm not greater than 156 beats per minute (BPM) in all. A 10-second ECD exposure in an ideal cardiac axis application did not demonstrate concerning tachyarrhythmias using human models. The swine model may have limitations when evaluating ECD technology.</td>
<td>(Sloan 2008⁴) Serum troponin I measurement of subjects exposed to the TASER X-26. 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 ECD did not develop an abnormal serum troponin I level 6 h after shock, suggesting that there was no myocardial necrosis or infarction.</td>
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<td>(Ho 2007⁵) 25 subjects exposed to 15 s TASER X26 discharge. ECG before and immediately after. No observed changes as read by a blinded cardiologist.</td>
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<td>(Vilke 2007⁶) 32 volunteers. 5 s or shorter ECD discharge. No arrhythmias or interval, or morphology changes.</td>
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<td>(Levine 2007⁷) 105 subjects. Mean shock duration 3.0 s. Mean heart rate increased 15 BPM. No ectopy (extra beats) or dysrhythmia (except one who had ectopy pre and post).</td>
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<td></td>
<td>(Ho 2007⁸) Normal sinus rhythm demonstrated in half. Others had elevated heart rate but never greater than 156 BPM.</td>
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<tr>
<td></td>
<td>(Ho 2008⁹) 5 s ECD exposure with laboratory and ECG data collected serially over 24 hours. No ECG changes.</td>
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<td>(Barnes 2006¹⁰) Variable exposure between 1 and 5 s. No cardiac dysrhythmias. Increases in heart rate and blood pressure.</td>
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<tr>
<td></td>
<td>(Levine 2005¹¹) Variable exposure between 1 and 5 s. No cardiac dysrhythmias or conduction or morphologic changes on ECG. Average increase in heart rate by 20 BPM.</td>
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<tr>
<td>(Swerdlow 2008¹²) Presenting Rhythm in Sudden Custodial Deaths After Use of TASER®</td>
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Electronic Control Device.
(Bozeman 2007\textsuperscript{13}) 99.7% of 962 subjects had no injuries or mild injuries only. No deaths attributed to ECDs.

(Ho 2006\textsuperscript{14}) Study of 162 custodial deaths found none immediately following ECD application, thus eliminating VF as possible factor.

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<th>FEM Studies</th>
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<tr>
<td>(Panescu 2008\textsuperscript{15}) Theoretical Possibility of Ventricular Fibrillation During Use of TASER Neuromuscular Incapacitation Devices. The results indicated that TASER devices, while not risk free, have a very low cardiac risk profile when used for suspect temporary incapacitation.</td>
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<tr>
<td>(Holden 2007\textsuperscript{16}) Using a numerical phantom constructed from medical images of the human body in which the material properties of the tissues are represented, computational electromagnetic modeling has been used to predict the currents arising at the heart following injection of M26 and X26 waveforms at the anterior surface of the chest (with one TASER “barb” directly overlying the ventricles). The modeling indicated that the peak absolute current densities at the ventricles were 0.66 and 0.11 milliamperes (mA) per square millimeter (mm\textsuperscript{2}) for the M26 and X26 waveforms, respectively. When applied during the vulnerable period to the ventricular epicardial surface of guinea pig isolated hearts, the M26 and X26 waveforms induced ectopic beats, but only at current densities greater than 60-fold those predicted by the modeling. When applied to the ventricles in trains designed to mimic the discharge patterns of the TASER devices, neither waveform induced VF at peak currents &gt;70-fold (for the M26 waveform) and &gt;240-fold (for the X26) higher than the modeled current densities. This study provides evidence for a lack of arrhythmogenic action of the M26 and X26 TASER devices.</td>
</tr>
<tr>
<td>(Panescu 2007\textsuperscript{17}) Even with an unrealistically thin layer of fat, worst-case skeletal muscle maximum values for TASER ECD electrical fields (E) and current densities (J) that reach into deeper layers of tissue are insufficient to trigger VF.</td>
</tr>
<tr>
<td>(Panescu 2006\textsuperscript{18}) Both J and E values are higher than thresholds required for</td>
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neuromuscular activation but significantly lower than levels needed for permanent cellular
electroporation or tissue damage.

(Stratbucker 200619) The models analyzed described strength-duration thresholds for
myocyte excitation and VF induction. FEM was used to compute current density in the heart
for worst-case TASER electrode placement. The model predicts a maximum TASER current
density of 0.27 mA/cm\(^2\) in the heart. It is concluded that the numerically simulated TASER
ECD current density in the heart is about half the threshold for myocytes excitation and more
than 500 times lower than the threshold required for inducing VF.

(Panescu 200620) The goal of this paper was to analyze the distribution of currents in
muscle layers and understand the electro-muscular incapacitation safety and efficacy of
TASER ECDs. The analyses described skeletal muscle and motor nerve activation, cell
electroporation and current and electric field distributions through skin, fat and muscle
layers, under worst-case assumptions for TASER electrode penetration and separation. For
the muscle layer, the analysis predicted worst-case current-density and field-strength values
of 94 mA/cm\(^2\) and 47 volts (V) per centimeter (cm). Both values are higher than thresholds
required for neuromuscular activation but significantly lower than levels needed for
permanent cellular electroporation or tissue damage.

<table>
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<tr>
<th>Modeling Based on Human Transcutaneous Pacing Thresholds</th>
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<tr>
<td>(Ideker 2007\textsuperscript{21}) Cardiac pacing threshold for adults is 2.33 times the size of the TASER pulses andVF threshold is 12.6 times the minimum pacing threshold, so VF threshold is 29 times the size of the TASER pulses. Electroporation and heat necrosis unlikely.</td>
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<th>Indirectly Related Human Studies</th>
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<tr>
<td>(Bouton 2007\textsuperscript{22}) The purpose of this study was to examine the effects of a single TASER ECD exposure on markers of physiological stress. Cardiorespiratory and blood parameters were followed before and for 60 minutes after a 5-second TASER ECD exposure on 21 men and women law enforcement officer volunteers.</td>
</tr>
<tr>
<td>(Klein 1988\textsuperscript{23}) Ventricular pacing threshold using transcutaneous pacing was determined in 11 of 16 patients. The mean threshold was 61 mA (range 45 to 80 mA).</td>
</tr>
</tbody>
</table>
### Human Case Reports

(Haegeli 2006\textsuperscript{24}) A subject with an implantable defibrillator.

(Kim 2005\textsuperscript{25}) 14-year-old (180 lb) agitated, violent psychiatric patient noted to have VF arrest after the TASER ECD exposure. However, the cardiac arrest was 14 minutes after the ECD application and the VF 23 minutes after ECD application. [See, Response to Letter in the New England Journal of Medicine, Open Letter to Law Enforcement Community, dated September 23, 2005.]

### Animal Studies

(Hughes 2008\textsuperscript{26}) In porcine model study of continuous TASER X26 exposures of from 3 to 30 minutes there was no indication of electrical causes of death such as ventricular fibrillation.

(Lakkireddy 2008\textsuperscript{27}) Cardiac effects of electrical stun guns: does position of barbs contact make a difference? Standard TASER discharges did not cause VF at any of the positions. Induction of VF at higher output multiples appear to be sensitive to electrode distance from the heart, giving highest ventricular fibrillation safety margin when the electrodes are placed on the dorsum. Rapid ventricular capture appears to be a likely mechanism of VF induction by higher output TASER discharges.

(Lakkireddy 2008\textsuperscript{28}) Can Electrical-Conductive Weapons (TASER®) alter the functional integrity of pacemakers and defibrillators and cause rapid myocardial capture?

(Walter 2008\textsuperscript{29}) TASER X26 discharges immediately and invariably produced myocardial capture. This usually reverted spontaneously to sinus rhythm post discharge, but fatal VF was seen in one small swine.

(Lakkireddy 2007\textsuperscript{30}) No VF with standard discharge at any of the 5 positions tested on the chest. VF threshold was from 5–50 times the standard discharge depending on location on thorax.

(Valentino 2007\textsuperscript{31}) Ventricular capture occurred in all animals at 300 BPM during the discharge by ultrasound. Three animals with ventricular arrhythmias after discharge.

(Esquivel 2007\textsuperscript{32}) Stinger™ S-400 applied 21 times over 31 minutes. Transient respiratory
acidosis (7.34), metabolic vasodilation, and increase in lactate (3.99) with recovery in 4 hours. Effect less than exhaustive exercise. As with Jauchem et al., visually decreased ventilation during exposures. Few PVCs in one animal. No elevation of troponin.

(McDaniel 2006\textsuperscript{33}) Canine model. No episodes of VF with standard ECD discharge, discharge with epinephrine infusion, discharge with isoproterenol infusion, and two simultaneous discharges.

(Lakkireddy 2006\textsuperscript{34}) Discharges did cause occasional myocardial capture, but never at a fast enough rate (necessary to induce VF in this model) even in the worst case positioning. Cocaine increased the safety margin 1.5–2 times.

(Nanthakumar 2006\textsuperscript{35}) ECD discharges did cause myocardial capture in 78% of thoracic discharges, and in 0% of abdominal discharges. The mean ventricular rate was 324 ± 66 BPM. One episode of VF and one episode of non-sustained VT with epinephrine infusion only.

(Nanthakumar 2007\textsuperscript{36}) “We did not state that NIDs cause [VF] in humans, and we agree that we cannot conclude from our study that NID discharges cause arrhythmias in typical use.”

(Webster 2006\textsuperscript{37}) Removed fat and muscle and created tunnel filled with conductive gel. Determined the average dart-to-heart distance to induce VF was 17 mm. Noted skin to heart distances in humans 10–58 mm. Calculated probability of VF = 0.000187. [See also, deposition transcript of Professor John Webster dated October 5, 2006; and updated unpublished no-gel paper.]

(McDaniel 2005\textsuperscript{38}) Safety index for VF ranged from 15 x – 42 x as the weight increased from 30 to 117 kg.

(Tisdale 1996\textsuperscript{39}) Cocaine does not increase ventricular vulnerability to fibrillation in anesthetized dogs with normal intact hearts. Its electrophysiologic effects are similar to those of class I antiarrhythmic agents in this model.
(Stratbucker 1996\textsuperscript{40}) AIR TASER Model 34000 Safety Study.

(Voorhees 1984\textsuperscript{41}) In a canine model, “[w]hile the current required to produce either fibrillation or pacing decreased as pulse duration increased, the safety factor (i.e., the ration of fibrillation current to pacing current) remained nearly constant averaging 12.6 for all pulse durations examined. That is, on average, a stimulus of any given duration between 1 and 50 ms required at least 12 times the current required for pacing to produce [VF]. We conclude that in normal canine hearts, the risk of inducing VF during precordial pacing is small.”

Other Relevant Studies or Materials

(Kroll 2008\textsuperscript{42}) Science and Medicine of TASER\textsuperscript{®} Electronic Control Devices.


(Kroll 2008\textsuperscript{43}) Spectrum article, Crafting the Perfect Shock.

(Branch 2007\textsuperscript{44}) Supplement to HOSDB Evaluations of TASER Devices A collection of medical evidence and other source material.

(Canadian Police Research Centre 2005\textsuperscript{45}) Review of conducted energy devices.


(Toxicology Excellence for Risk Assessment [TERA] 2005\textsuperscript{47}) Human Effectiveness and Risk Characterization of the Electromuscular Incapacitation Device – A Limited Analysis of the TASER Part II – Appendices.

(Cooper 2005\textsuperscript{48}) UK government’s assessment of the medical risks of M26 and X26 TASERs.

(Wilkinson 2005\textsuperscript{49}) PSDB Further Evaluation of TASER Devices.
Ventricular Fibrillation – Detail

(Battershill 2004) TASER Technology Review & Interim Recommendations.
(United Kingdom Defence Scientific Advisory Council 2004) DSAC Sub-Committee on the Medical Implications of Less-lethal Weapons.
(Kester 2003) Patterns of Injury, Recognition, and Treatment for Less Lethal Law Enforcement.

(Bleetman 2003) The ADVANCED TASER: A Medical Review.
(Karch 1999) Drug abusers who die during arrest or in custody.
(Roy 1989) Tests on a shocking device—the stun gun.
Ventricular Fibrillation – Detail

(Underwriters’ Laboratories 195562) Electric Shock as it Pertains to the Electric Fence.

1 This document, and the entirety of its contents, is for discussion and demonstration purposes only. All numbers, references, and values in this document are nominal. Actual measurements on particular products, references, and/or analogies may vary as a result of many factors including, but not limited to, factors outside TASER International, Inc.’s (TASER’s) control. Please refer to TASER published product specifications, manuals and product literature for additional information including specified limits, test conditions, and allowed tolerances. For more information please see current TASER Web site (www.taser.com). TASER reserves the right to change or modify this document without notice. TASER is a registered trademark of TASER International, Inc.

2 Stedman’s Medical Dictionary, 26th Edition.


12 Heart Rhythm 2008, 29th Annual Scientific Sessions, May 14-17, 2008, San Francisco, CA USA. Charles Swerdlow, MD, FHRS, Mark W. Kroll, PhD, FHRS, Howard Williams, Mazda Biria, MD, Dhanunjaya Lakkireddy, MD and Patrick J. Tchou, MD. Cedars-Sinai Medical Center, Los Angeles, CA, University of Minnesota, Minneapolis, MN, San Marcos Police Department, San Marcos, TX, University of Kansas Medical Center, Kansas City, KS, Cleveland Clinic, Cleveland, OH.


28 Heart Rhythm 2008, 29th Annual Scientific Sessions, May 14-17, 2008, San Francisco, CA USA. Dhanunjaya R. Lakkireddy, MD, Mazda Biria, MD, Esam Baryun, MD, Loren Berenbom, MD, Rhea Pimentel, MD, Martin P. Emert, MD, Kevin Kreighbaum, RN, Mark W. Kroll, PhD and Atul Verma, MD.  Mid America Cardiology @ University of Kansas Hospital, Kansas City, KS, University of Minnesota, Minneapolis, MN, Southlake Regional Health Center, Toronto, ON, Canada.


Ventricular Fibrillation – Detail


