33rd Annual International Conference of the IEEE EMBS
Boston, Massachusetts USA, August 30 - September 3, 2011

Ventricular Fibrillation Risk Estimation for Conducted Electrical Weapons: Critical Convolutions

Mark W. Kroll, PhD, Senior Member IEEE; Dhanunjaya Lakkireddy, MD; Peter S. Rahko, MD; Dorin Panescu, PhD, Fellow IEEE

Abstract—The TASER® Conducted Electrical Weapon (CEW) is used by law enforcement agencies about 900 times per day worldwide and has been shown to reduce suspect and officer injuries by about 65%. However, since a CEW delivers rapid electrical pulses through injected probes, the risk of inducing ventricular fibrillation (VF) has been considered. Animal studies have shown that the tip of the probe must come within a few millimeters of the surface of the heart for the CEW to induce VF in a typical animal application. Early calculations of the CEW VF risk in humans used sophisticated 3-D chest models to determine the size of the probe landing areas that had cardiac tissue within a given distance of the inner surface of the ribs. This produced a distribution of area (cm^2) vs. mm of depth. Echocardiography was then used to determine the shortest distance from the skin surface to the cardiac surface. This produced a population distribution of skin-to-heart (STH) distances. These 2 distributions were then convolved to arrive at a probability of inducing VF for a typical human CEW application. With 900, 000 probe-mode field uses to date, epidemiological results have shown that these initial VF risk estimates were significant overestimates. We present model refinements that take into account the gender and body-mass-index (BMI) of the target demographics and produce VF risk estimates concordant with the epidemiological results. The risk of VF is estimated at 0.4 per million uses with males.

INTRODUCTION

The TASER® Conducted Electrical Weapon (CEW) is used by law enforcement agencies about 900 times per day worldwide and been shown to reduce suspect and officer injuries by about 65%. There are about 800 arrest-related-deaths (ARDs) in North America per year.[1] In the vast majority of these cases, no CEW was used during the arrest. However, since a CEW delivers rapid electrical pulses through injected probes, the risk of inducing ventricular fibrillation (VF) has been considered as a possible contributor. Animal studies have shown that a necessary component for the induction of VF is a small dart-to heart distance (DTH) on the order of a few mm (mean value 5.8 mm).[2]

M.W. Kroll is with the Biomedical Engineering Dept. at the University of Minnesota, Minneapolis, MN 55454. Member of TASER International, Inc. corporate and scientific board. Email: kroll051@umn.edu
D.R. Lakkireddy is with the University of Kansas Hospital, Kansas City, KS. Email: DLakkireddy@mac.md
P.S. Rahko is with the University of Wisconsin Department of Medicine, Madison, WI. Email: psr@medicine.wisc.edu
D. Panescu is with NewCardio, Inc., Santa Clara, CA 95054. Email: dpanescu@newcardio.com

This requires the combination of a low probability (of a probe landing in a small area directly over the right ventricle) and the low probability of a subject with a very thin skin-to-heart (STH) distance. The area of theoretical risk is shown in yellow in Figure 1.

The human heart is closest to the chest wall in the 4th intercostal space (between the 4th and 5th ribs) as shown in Figure 1. Bone is largely an electrical insulator and hence the ribs and sternum do not offer VF-sensitive landing areas. More than a few centimeters from the sternum, the heart is retracted from the inner chest wall and there is insulating lung tissue interspersed. This produces the sensitive zones as depicted in the simplified drawing of Figure 1.

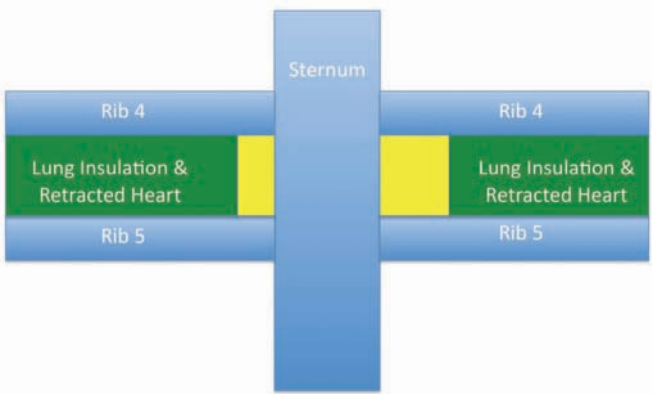

Figure 1. A necessary (but not sufficient) condition for the electrical induction of VF is that a probe lands in a small area in the 4th intercostal space near the sternum.

Early CEW VF risk models convolved distributions of the probe-landing data with the population distribution of chest-wall thickness to arrive at an estimated probability of inducing VF in a human. Present epidemiological data have demonstrated that the earlier risk models significantly overestimated the VF risk. We felt that refining these models with gender-specific male body habitus data, and correcting for the cardiac conduction differences between swine and humans might bring the model predictions more in line with actual field experience.

METHODS and RESULTS

Wisconsin Model

We began with the University of Wisconsin Biomedical Engineering Department model for CEW VF risk.[2, 3] They determined that the DTH distance for a TASER® X26™ CEW

to (directly induce VF) in 5 swine was 5.8 ± 2.04 mm (range 2-8 mm).[2] The DTH was determined twice in each animal and there was no difference in the values (p = .37 by paired T-test). Since the 8 mm value was both the extreme and the mode it dominates the modeling as will be seen. This is consistent with the Lakkireddy results that found no VF inductions in swine with a DTH range of 12.3-22.9 mm. (See Appendix)

Table 1. Distribution of CEW DTH Distances to Induce VF in 5 Swine in the Wisconsin Model.

DTH (mm)	Frequency
2	1
4	2
5	1
6	2
7	1
8	3

Data from Wu et al.[2]

They then modeled the current distribution around a fully inserted 9 mm CEW probe to find the boundaries of the critical charge-density region. This critical charge density threshold was about 11 μC/cm^2. This boundary is given in Figure 2 and reflects (in the long axis) a 6 mm critical DTH distance in swine. The curve is dented near ribs (not shown).

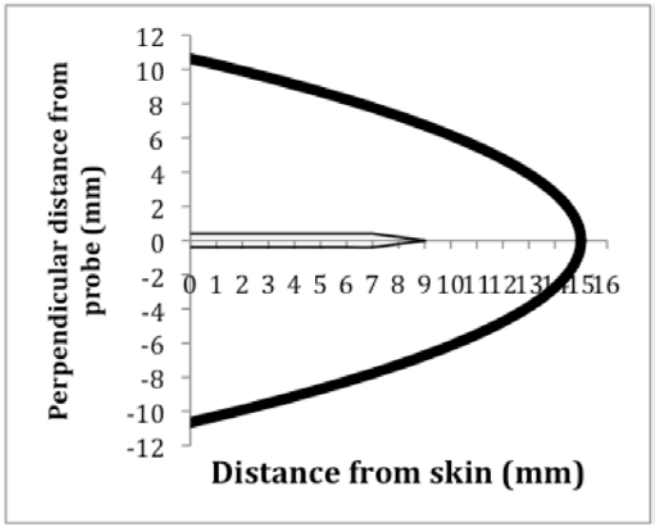

Figure 2. Boundary of critical charge density (thick line) and fully inserted CEW probe (thin lines) in assumed homogenous tissue.

This group then used the Utah rib-cage model and the Texas cardiac mesh model to calculate the VF risk-region area where a fully inserted 9 mm probe would produce the critical charge density to induce VF in the porcine model.[4, 5] In attempting to extrapolate to humans, the area of this risk-region was, of course, dependent on the STH distance which varies between subjects. For an extremely thin human (with a STH distance of 11 mm), there is a region on the chest where a fully-inserted perpendicular 9 mm probe could theoretically induce VF.

The condition to be satisfied was:

DTH = STH – 9 mm < 8 mm critical value.

2 mm = 11 mm (STH for thin subject) – 9 mm (probe length)

< 8 mm assumed critical distance

For example, in Figure 3, the "Highest Risk Region" refers to the skin directly over the heart, where the right ventricle is within 1 mm of the inner chest wall. This region would then (based on the Wisconsin group's assumptions) have VF induction for subjects with a STH distance of less than 16 mm.

16 mm (STH)
- 9 mm probe length
+ 1 mm maximum cardiac retraction
= 8 mm critical distance.

The critical skin area is actually slightly larger than the region where the heart is within 8 mm directly beneath the fully inserted CEW probe. That is because the width of the critical charge boundary (see Figure 2) allows the electrical field from the sides of the probe to capture the heart. This small effect was included in the Wisconsin Model.[3]

The size of the VF risk region was determined to be 3.33 cm^2 for an extremely thin human with a STH distance of 11 mm. In this theoretical model, the size of the VF risk region decreased rapidly for higher STH distances as shown in Table 2 which is based on the worst case — not the mean — value of an 8 mm VF DTH. The probability (in parts per million) of a CEW probe landing in this "sweet spot" was calculated, by the Wisconsin group, from field data on probe landings.

Table 2. Skin Area of the VF Risk Region Modeled as a Function Of STH Distance.

STH (skin-to-heart) in mm	Size of VF Risk Region (cm^2)	Probability (PPM) of probe landing in Risk Region
17	0	0.0
16	0.00716	2.7
15	0.0377*	15.1
14	0.161*	64.4
13	0.532	204.0
12	1.61	583.0
11	3.33	1200.0

* Interpolated by Size = 0.002587*STH^4; r^2=.998

Echocardiography was then used to measure the parasternal STH distance in 150 human subjects.[6] The STH distance was 32.1 ± 7.9 mm. From this it is easily seen that the likeli-

hood of a CEW probe having a sufficiently small DTH — even for landing in the highest risk region — is very small. Using the mean STH value (32.1 mm) and subtracting the assumed 9 mm probe penetration leaves a DTH value of 23.1 mm which is far in excess of the maximum 8 mm VF DTH distance for swine.

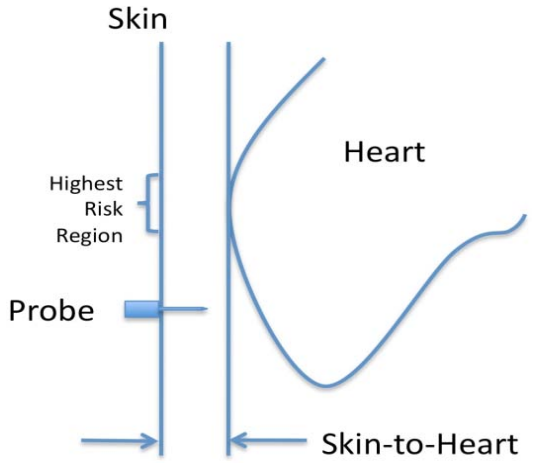

Figure 3. In humans, the highest risk region is where the heart (right ventricle) is within 1 mm of the inner surface of the ribs. With a probe landing away from this region the DTH distance grow rapidly.

The results shown in Table 2 were convolved with the STH distances obtained from the human echocardiography study. Note that only 8 of the 150 echocardiography subjects had small enough STH distances to be considered in the analysis. The overall sum of the probabilities was 16 PPM.

Table 3. Modeled Population Probability of Inducing VF in Swine Based on 8 mm VF DTH.

STH (mm)	Number of subjects	Probability of the STH (A)	Prob. of probe landing in Risk Region (PPM) (B)	Product A*B (PPM)
17	2	0.013	0	0.0
16	1	0.007	3	0.0
15	0	0.000	15	0.0
14	0	0.000	64	0.0
13	3	0.020	204	4.1
12	1	0.007	583	3.9
11	1	0.007	1200	8.0
Totals	8	0.054	2069	16.0

Only 3 of the 10 swine samples had a DTH as great as 8 mm (see Table 1). Thus the 16 PPM value given in Table 3 should be multiplied by 3/10 yielding a model theoretical VF estimate of:
4.8 PPM = 3/10 * 16 PPM

However, there is some small contribution from the single swine sample with VF DTH = 7 mm and from the 2 samples with VF DTH = 6 mm. So, the Wisconsin group performed the analysis shown in Table 3 for *each* of the swine VF DTH distances and then convolved those results with the probability of each VF DTH distance. The final result was a theoretical porcine model extrapolated VF probability of 6 PPM in humans. Thus the analysis using the 8 mm VF DTH distance contributed 78% of the VF probability estimate. (The swine data with 2-5 mm VF DTH distances made a negligible difference — because of the rarity of the requisite human STH distance and the small size of the sweet spot for that STH — and can be ignored.)

Gender Correction

ARD is uncommon in females. The Mumola study tabulated ARDs in 47 states over a 4-year (2003-2006) period and found only 112 (out of 2,686) were females.[1, 7] The Stratton study of excited delirium ARDs found only 1/13 was female.[8] The Ho study of ARDs had only 6/162 female.[9] In contrast, the echocardiography sample used in the Wisconsin model was 79/150 female. By chi-square analysis this difference was highly significant compared to the Stratton data ($p = .00186$), the Mumola data ($p = 2.6 \times 10^{-99}$), and the Ho data ($p = 2.9 \times 10^{-22}$)

It must be noted that only 1 of the 8 thin human subjects used in the Wisconsin Model was male (12.7 mm STH) and thus this demographic inconsistency had a major influence on the results. Finally, it is obvious that males and females have differing levels of chest musculature among other things. This suggested that the area ripest for refinement was an analysis for males.

The female subjects were then removed from the Wisconsin Model and the single male was left in the calculation as seen in Table 4. The probability (for this 13 mm STH) was then 0.014 = 1/71.

Table 4. Modeled *Male* Human Population Probability of VF Induction Based on 8 mm VF DTH

STH (mm)	Number of subjects	Probability of the STH (A)	Prob. of probe landing in Risk Region (PPM) (B)	Product A*B (PPM)
17	0	0.000	0	0.00
16	0	0.000	3	0.00
15	0	0.000	15	0.00
14	0	0.000	64	0.00
13	1	0.014	204	2.87
12	0	0.000	583	0.00
11	0	0.000	1200	0.00
Totals	1	0.014	2069	2.87

As discussed in the previous section, the human population VF probability based *solely* on the 8 mm VF DTH provides an overestimate of the overall probability since it does not consider the lower probabilities obtained with the 6 and 7 mm VF DTH distances. The final probability estimate of 6 PPM for the mixed-gender group was 37.5% (i.e. 6/16 * 100) of that of the 8 mm VF DTH analysis alone. We calculated that using this percentage for arriving at new estimates for the human VF probability would contribute a relative error of only 5.5%, which is immaterial for the purposes of this analysis.

Hence, the estimated probability of VF in a human male population would be:

1.1 PPM = 2.87 PPM * .375

Balancing BMI

Strote studied the autopsies of ARDs in which a CEW had been involved at some point.[10] He found a BMI of 30.8 ± 5.5 kg/m^2. The BMI of the males in the echocardiography sample was 27.9 ± 4.3 kg/m^2 (p = .0032 vs. Strote data). Since the STH distance is highly correlated to the BMI, this also presented a difference of clinical significance.

Since the Wisconsin group analysis (for male predictions) was essentially based on a single male we decided to add samples to improve statistical power. The Cleveland Clinic calculated the STH distances in 42 males using CT scans and provided the raw data to us.[11] We were concerned that echo probe pressure might slightly reduce the STH distance and the supine position, in the CT scanner, might also reduce the STH distance slightly. From the 108 data, 8 subjects were eliminated as being Tukey outliers (within their own modality class) primarily for BMI. (3 from the echo set and 5 from the CT scan set) We performed a multiple regression analysis on the 100 non-outlier subjects using age, height, weight, BMI, and imaging modality to predict the STH. While BMI and age were significant predictors of the STH (p < .0001 and p = .04 respectively) the modality (CT vs. echo) had no effect (p = .91). Thus we felt comfortable combining the data sets.

The consolidated group had a BMI of 28.4 ± 4.9 kg/m^2 (p = .014 vs. Strote data) so we sequentially removed alternating subjects beginning with the lowest BMI. This was done without regard to the STH distance. (The extreme echo case with STH of 12.7 mm survived the trimming.) This trimming process was stopped when the BMI reached 29.6 ± 4.2 kg/m^2 (p = .224 vs. Strote data). This left 69 subjects with a STH distance of 32.39 ± 7.44 mm. These data were well fit by a normal distribution (p =.477 by Shapiro-Wilk).

To avoid the sensitivity problems incurred in the original Wisconsin Model (from the use of a single male) we elected to generate a more continuous distribution of STH distance from these parameters. The results of convolving in this distribution are seen in Table 5.

At this point, the estimated probability of VF in a human male population would be:

0.88 PPM = 2.36 PPM * .375

The difference between this value and the value derived from the single male (1.1 PPM) is not clinically significant.

Table 5. Male VF Theoretical Probability From Parametric STH Distribution.

STH (mm)	Probability of the STH (A)	Prob. of probe landing in Risk Region (PPM) (B)	Product A*B (PPM)
17	0.00631	0	0.00
16	0.00474	3	0.01
15	0.00349	15	0.05
14	0.00253	64	0.16
13	0.00180	204	0.37
12	0.00125	583	0.73
11	0.00086	1200	1.03
Totals	0.021	2069	2.36

Porcine Model to Human Adjustment

It is well recognized that swine are the easiest mammal to fibrillate (least current required) and thus present a conservative model for estimating electrocution risk.[12-14] In canines and humans the Purkinje fibers are confined to a very thin (few mm) endocardial layer.[15] In swine they cross the entire ventricular wall.[16] It has been recently demonstrated that activation in swine proceeds from the epicardium to the endocardium while in canines and human it proceeds in the reverse direction.[17] Thus swine hearts are literally wired outside-in compared to humans and are considerably more sensitive to external (extra-cardiac and transcutaneous) electrical currents. In addition they have significant ion channel differences.[18]

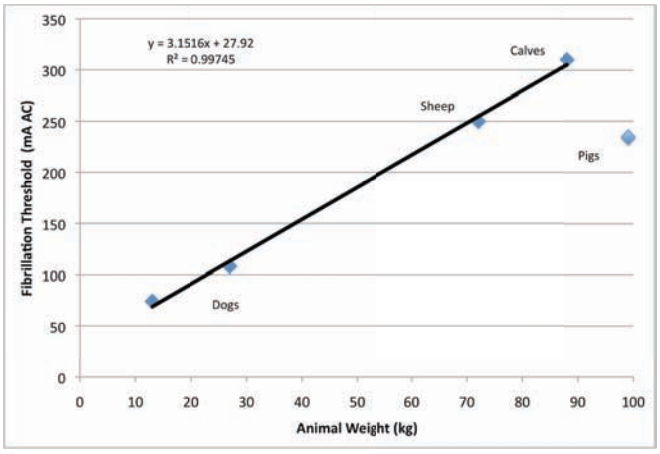

Figure 4. Swine fibrillate at 68% of the current required for other mammals of the same body weight.

Data compiled by Dalziel show that swine fibrillate at 68% of the current of other mammals when corrected for body weight as depicted in Figure 4.[13] No study has done a direct

comparison of the VF threshold (VFT) between swine and humans and thus there is no justification for a direct extrapolation from swine to humans. Hence, the best approximation we have to use for a correction between swine and humans is the 68% fraction of the swine VFT versus other mammals.

The fall-off of current density from the metal CEW probe tip is shown in Figure 5 of the Sun paper.[3] This followed an inverse 5/4 power relationship. The current density was given by:

$$J\ (A/m^2) = 10467 * d^{-1.26}\ (r^2 = .9996)$$

where d is the distance from the metal probe tip in mm.

The 32% lower swine VFT is equivalent to having the probe 1.96 mm closer to the heart. Thus the species effect can be modeled by requiring the STH distance to be 2 mm less to obtain a theoretical human VF risk estimate. Hence, the STH values were shifted by 2 mm and then convolved with the existing probability values as shown in Table 6.

Table 6. Modeled Male-Human VF Probability Based on Correction From Swine Data.

Swine STH (mm)	Required STH for humans	Probability of the STH (A)	Prob. of probe landing in Risk Region (PPM) (B)	Product A*B (PPM)
17	15	0.00349	0	0.00
16	14	0.00253	3	0.01
15	13	0.00180	15	0.03
14	12	0.00125	64	0.08
13	11	0.00086	204	0.18
12	10	0.00058	583	0.34
11	9	0.00038	1200	0.46
Totals		0.011	2069	1.1

The estimated theoretical probability of VF in a male *human* population would be:

$$0.41\ PPM = 1.09\ PPM * .375$$

Table 7 depicts the evolution of the swine-based computer models for CEW VF risk prediction. The first 2 results were based on a swine study that used a gel tunnel to provide a direct (albeit moderate resistance) pathway to the heart.[19] They are not discussed further in this paper.

DISCUSSION

As of 31 March 2011 there have been an estimated 1,27 million (±2%) field uses of CEWs.[20] Bozeman found that field usage involved successful probe landings 70.8% of the time.[21] The reason why probe-mode usage is not higher is that many CEW field uses are drive-stun only. This gives an estimated 900 000 probe uses on human beings in the field.

There are 2 published anecdotes suggesting that a TASER CEW caused VF in a human being. The best known is the Kim-Franklin letter to the New England Journal of Medicine.[22] Since the subject was 14 years old at the time of the incident it was impossible to get details of the incident until recently. The violent psychiatric subject had a normal pulse and respiration (as reported by paramedics in sworn deposition) after the CEW was used and thus no dysrhythmia or cardiac arrest was induced by the CEW.[23, 24] It had also been demonstrated that the cardiac rhythm strip shown (supposedly demonstrating a return to sinus rhythm by a defibrillation shock) was cropped after what were actually 3 PVCs followed by asystole. This anecdote has been challenged in the literature. [25, 26]

Table 7. The Evolution of Theoretical VF Risk Probability Modeling.

Paper	Improvements over previous model	VF probability prediction (PPM)
Wu[19]	Used gel tunnel to heart for VF DTH determination.	172
Sun[3]	Used gel tunnel VF DTH but improved FEM.	1000
Sun[3]	Used intact swine (no gel tunnel) for VF DTH.	6
present	Using male gender as seen in ARDs.	1.1
present	Using BMI seen in ARDs.	0.88
present	Extrapolated to humans using swine VF sensitivity adjustment.	0.41

A more recent case also suggested that an older teenager had VF induced by an CEW.[27] This report was completely contradicted by the emergency medical services (EMS) records and sworn paramedic testimony.[28] One of the co-authors admitted under oath that the cardiac rhythm was actually asystole "or fine VF."[29] Fine VF is highly unlikely as the paramedic monitored 3 leads. Fortuitously, this anecdote had been repudiated by treating physicians at the *same hospital*. The treating physicians published that the subject, presented with asystole — not VF — consistent with his extreme levels of ethanol.[30]

The Swerdlow study analyzed the presenting ARD cardiac rhythms in which a CEW was used.[31] A single case of VF was found with timing consistent with the electrical induction of VF. That case can be demonstrated to not be CEW-induced VF due to the probe locations, failure of prompt defibrillation, and other factors.

Thus, out of more than 900 000 probe-mode uses, there has yet to be a confirmed case of VF in humans induced by a CEW. By Hanley's "Rule of 3" this means that the 95% confidence range for the risk of VF in humans is [0-3.4 PPM]. The first estimates from the Wisconsin Model were outside of this epidemiologically established limit. With corrections for male gender, and human versus swine differences, the model produces a more refined theoretical human VF risk estimates comporting with the epidemiological estimates of VF risk.

This and previous models assume that the metal CEW probes were fully inserted when they generally do not

actually penetrate fully. It also assumes no clothing over the heart. Correcting for these factors would have reduced the VF risk estimates further. This modeling would also exaggerate the risks for non-metallic TASER CEW probes.

This modeling is based on the "direct" induction of VF. We, and others, have published on the induction of VF from long-term (typically 90-second) cardiac capture.[32-35] Capture obtains with greater DTH distances than does the direct induction of VF. For multiple reasons, discussed in an earlier paper, the long-term capture risk of VF does not appear to apply to law-enforcement situations.[33]

The animal results used here were all based on the TASER X26 CEW. Other models, with different charges and pulse durations, may have different risk profiles.

CONCLUSIONS

Sophisticated published computer models have estimated the risk of ventricular fibrillation for conducted electrical weapons. A growing body of epidemiological data has now shown that these models produced significant overestimates. With the use of male body habitus data, and correcting for the differences between swine and humans the models now give a theoretical VF risk estimate of about 0.4 PPM or 1 per 2.5 million. This is consistent with the epidemiological findings to date.

APPENDIX

Both Wu and Lakkireddy measured the DTH distance in swine VF experiments.[2, 36, 37] Wu used 5 animals and a special probe that could be advanced towards the epicardium beginning with a 12 mm DTH with a CEW exposure at each position. This procedure was repeated once.

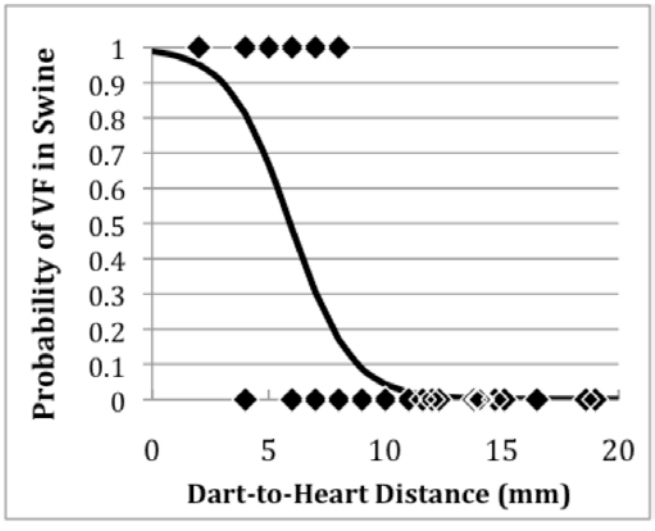

Figure 5. Probability of inducing VF in swine with CEW application as a function of the DTH distance. The diamonds reflect actual data while the curve is a logistic regression fit to the data.

Lakkireddy used 13 animals with a standard probe (fully inserted over the point of minimum STH distance) and delivered 3 applications of CEW current in each animal. A total of 84 tests were made (39 by Wu and 45 by Lakkireddy) and there were 10 inductions of VF (Wu). These inductions are listed in Table 1 and represented by the diamonds seen at the top of Figure 5.

The probability of inducing VF was then modeled by logistic regression using the study and the DTH distance as predictors. The study author (Wu vs. Lakkireddy) was not a predictor ($p = .96$) so the data were consolidated and the logistic regression run again using only the DTH distance. The VF data were well fit by the DTH distance ($r^2 = .49$ by U-test, $p = .0009$.) The model output is shown as the curve in Figure 5 and Table 8.

Table 8. Probability of Inducing VF in Swine

DTH (mm)	DTH (inch)	Probability
1	0.04	98%
2.5	0.10	93%
5	0.20	67%
10	~ 0.4	4%
25	~ 1.0	0.5 PPM
50	~ 2.0	2.8×10^{-15}
100	~ 4.0	8.6×10^{-32}

ACKNOWLEDGEMENTS

We wish to thank Dr. Pat Tchou (Cleveland Clinic) for his kindness in sharing unpublished data.

REFERENCES

1. Mumola C. Arrest-Related Deaths in the United States, 2003-2005. *Bureau of Justice Statistics Special Report* October 2007(NCJ 219534).

2. Wu JY, Sun H, O'Rourke AP, et al. Taser blunt probe dart-to-heart distance causing ventricular fibrillation in pigs. *IEEE Trans Biomed Eng.* Dec 2008;55(12):2768-2771.

3. Sun H, Haemmerich D, Rahko PS, Webster JG. Estimating the probability that the Taser directly causes human ventricular fibrillation. *J Med Eng Technol.* Apr 2010;34(3):178-191.

4. Klepfer RN, Johnson CR, Macleod RS. The effects of inhomogeneities and anisotropies on electrocardiographic fields: a 3-D finite-element study. *IEEE Trans Biomed Eng.* Aug 1997;44(8):706-719.

5. Zhang YL, Bajaj C. Finite Element Meshing for Cardiac Analysis. *Univ. of Texas at Austin: ICES Technical Report.* 2004 2004(4-26).

6. Rahko PS. Evaluation of the skin-to-heart distance in the standing adult by two-dimensional echocardiography. *J Am Soc Echocardiogr.* Jun 2008;21(6):761-764.

7. Mumola C. Arrest-Related Deaths in the United States, 2003-2006. *Bureau of Justice Statistics Special Report* 2011.

8. Stratton SJ, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden death of individuals requiring restraint for excited delirium. *Am J Emerg Med.* May 2001;19(3):187-191.

9. Ho JD, Heegaard WG, Dawes DM, Natarajan S, Reardon RF, Miner JR. Unexpected arrest-related deaths in america: 12 months of open source surveillance. *West J Emerg Med.* May 2009;10(2):68-73.

10. Strote J, Range Hutson H. Taser use in restraint-related deaths. *Prehosp Emerg Care.* Oct-Dec 2006;10(4):447-450.
11. Bashian G, Wagner G, Wallick D, Tchou P. Relationship of Body Mass Index (BMI) to Minimum Distance from Skin Surface to Myocardium: Implications for Neuromuscular Incapacitating Devices (NMID). *Circulation.* 2007;116:II-947.
12. Chilbert M. High-Voltage and High Current Injuries. In: Reilly J, ed. *Applied Bioelectricity: From Electrical Stimulation to Electrical Pathology*. New York: Springer-Verlag; 1998:412-453.
13. Dalziel CF, Lee WR. Reevaluation of lethal electric currents. *IEEE Transactions on Industry and General Applications.* 1968;IGA-4(5):467-476.
14. Kroll MW, Calkins H, Luceri RM, Graham MA, Heegaard WG. Sensitive swine and TASER electronic control devices. *Acad Emerg Med.* Jul 2008;15(7):695-696; author reply 696-698.
15. Schnabel PA, Richter J, Schmiedl A, et al. Patterns of structural deterioration due to ischemia in Purkinje fibres and different layers of the working myocardium. *Thorac Cardiovasc Surg.* Aug 1991;39(4):174-182.
16. Howe BB, Fehn PA, Pensinger RR. Comparative anatomical studies of the coronary arteries of canine and porcine hearts. I. Free ventricular walls. *Acta Anat (Basel).* 1968;71(1):13-21.
17. Allison JS, Qin H, Dosdall DJ, et al. The transmural activation sequence in porcine and canine left ventricle is markedly different during long-duration ventricular fibrillation. *J Cardiovasc Electrophysiol.* Dec 2007;18(12):1306-1312.
18. Li GR, Du XL, Siow YL, O K, Tse HF, Lau CP. Calcium-activated transient outward chloride current and phase 1 repolarization of swine ventricular action potential. *Cardiovasc Res.* Apr 1 2003;58(1):89-98.
19. Wu JY, Sun H, O'Rourke AP, et al. Taser dart-to-heart distance that causes ventricular fibrillation in pigs. *IEEE Trans Biomed Eng.* Mar 2007;54(3):503-508.
20. Brewer J, Kroll M. Field Statistics Overview. In: Kroll M, Ho J, eds. *TASER Conducted Electrical Weapons: Physiology, Pathology, and Law*. New York City: Springer-Kluwer; 2009.
21. Bozeman W, Winslow J, Hauda W, Graham D, Martin B, Heck J. Injury Profile of TASER®Electrical Conducted Energy Weapons (CEWs) *Annals of Emergency Medicine.* 2007;50:S65.
22. Kim PJ, Franklin WH. Ventricular fibrillation after stun-gun discharge. *N Engl J Med.* Sep 1 2005;353(9):958-959.
23. Chicago_Fire_Department. Paramedic Records for Case of Mr. Akeem Watson. Feb. 7 2005.
24. Hutchinson J. Deposition in Ortega Piron (Akeem Watson) v. City of Chicago. *Circuit Court of Cook County, Illinois, County Department, Law Division.* 21 June 2007(Case No. 05 L 1643).
25. Roberts J. Medical Effects of TASERs. *Emergency Medicine News.* Feb 2008;30(2):12-15.
26. Kroll MW, Calkins H, Luceri RM. Electronic control devices and the clinical milieu. *J Am Coll Cardiol.* Feb 13 2007;49(6):732; author reply 732-733.
27. Naunheim RS, Treaster M, Aubin C. Ventricular fibrillation in a man shot with a Taser. *Emerg Med J.* Aug 2010;27(8):645-646.
28. Beckenholdt M. Deposition in Fahy v. St. Louis Police Dept. *US District Court, Eastern District Of Missouri.* 15 Oct 2009;4:08-CV-1889.
29. Aubin C. Deposition in Fahy v. St. Louis Police Dept. *US District Court, Eastern District Of Missouri.* 12 July 2010;4:08-CV-1889.
30. Schwarz ES, Barra M, Liao MM. Successful resuscitation of a patient in asystole after a TASER injury using a hypothermia protocol. *Am J Emerg Med.* May 2009;27(4):515 e511-512.
31. Swerdlow CD, Fishbein MC, Chaman L, Lakkireddy DR, Tchou P. Presenting rhythm in sudden deaths temporally proximate to discharge of TASER conducted electrical weapons. *Acad Emerg Med.* Aug 2009;16(8):726-739.
32. Dennis AJ, Valentino DJ, Walter RJ, et al. Acute effects of TASER X26 discharges in a swine model. *J Trauma.* Sep 2007;63(3):581-590.
33. Kroll M, Panescu D, Hinz A, Lakkireddy D. A Novel Mechanism for Electrical Currents Inducing Ventricular Fibrillation: The Three-Fold Way to Fibrillation *Engineering in Medicine and Biology Society Proceedings.* Sept 2010:1990-1996.
34. Walter RJ, Dennis AJ, Valentino DJ, et al. TASER X26 discharges in swine produce potentially fatal ventricular arrhythmias. *Acad Emerg Med.* Jan 2008;15(1):66-73.
35. Nimunkar A, Wu J, O'Rourke A, Huebner S, Will J, Webster J. Ventricular fibrillation and blood chemistry after multiple Tasering of Pigs. *Physiol Meas.* 2011.
36. Lakkireddy D, Wallick D, Verma A, et al. Cardiac effects of electrical stun guns: does position of barbs contact make a difference? *Pacing Clin Electrophysiol.* Apr 2008;31(4):398-408.
37. Lakkireddy D, Wallick D, Ryschon K, et al. Effects of cocaine intoxication on the threshold for stun gun induction of ventricular fibrillation. *J Am Coll Cardiol.* Aug 15 2006;48(4):805-811.