Can TASER Electronic Control Devices Cause Cardiac Arrest?

TASER Electronic Control Devices and Cardiac Arrests: Coincidental or Causal?

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Being arrested is a highly emotional event and can result in a fatal, adrenergically supercharged physiological state.[1] The exertion of arrest-related struggle is several-fold greater than that seen with normal exercise and leads to numerous extreme metabolic and electrolytic derangements, including elevated levels of lactate, CO_2, potassium, creatine kinase, and myoglobin.[2] Only 1.6% of US law-enforcement interactions involve the use or threats of force, and annually there are ≈700 000 cases in which force is used or threatened.[3] There are ≈700 arrest-related deaths per year in the United States, yielding a mortality rate of ≈1 in 1000 for a law-enforcement interaction associated with force.[4]

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The electronic control device (ECD) has gained widespread acceptance as the force option for law enforcement because of its dramatic reduction in both suspect and officer injury. At the same time, advocacy groups post statements on the Internet listing the hundreds of arrest-related deaths after ECD use with the implication that the ECD involvement was causal. Studies covering a total of >48 000 forceful arrests have consistently found suspect injury rate reductions of ≈65%.[5,6] Of the 250 000 annual ECD field uses in the United States, only 1 in 4000 is involved in an arrest-related death. This reduction in fatality rate is consistent with published data showing that 5.4% of ECD uses "clearly prevented the use of lethal force by police."[7]

Of the >3 million total ECD applications, there have been 12 published case reports suggesting a potential cardiac arrest link, giving an incidence of 4×10^{-6} per application.[8–13] In most cases, those authors did not consider important factors that are now better understood. These include separating postural from cardiovascular collapse, the latency of electrically induced ventricular fibrillation (VF), the presence of significant cardiac pathology, failure of prompt defibrillation, the duration of documented breathing, the distance of the ECD electrode from the heart, and the stability of electrically induced VF. We have thoroughly investigated these cases as either TASER scientific advisors or expert witnesses. The goal of this article is to resolve the confusion about these cases by introducing more complete data and by using a consistent case-report scoring methodology.

Methods

Understanding the Confounders

Postural Versus Cardiovascular Collapse

In the normal course of life, an exertional postural collapse is, correctly, often associated with a cardiac arrest. However, it is sometimes forgotten that the ECD design goal is to cause a postural collapse to stop aggression. A sternal rub response is often blunted by the presence of alcohol, illegal drugs, psychotic break, and endorphins from the struggle. Hence, nonresponsiveness is also more difficult to evaluate in the law-enforcement scenario.

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Latency and Temporality

Another common error is the assumption that a cardiac arrest minutes after an electric exposure is temporally related to that exposure. Because most illnesses have latency periods measured in days, not seconds, it is common for even physicians to significantly overestimate the latency period for electrocution.[8,13] However, these latency periods are well established and are summarized in the online-only Data Supplement. Some of the cited case studies confuse loss of consciousness minutes later as "precisely" temporal to an alleged electrocution.[13,14] Loss of consciousness actually occurs 13±4 seconds after electrically induced VF.[15] It has also been suggested that an increased latency (of, say, 60 seconds) might be due to the induction of a ventricular tachycardia (VT) and that this would allow the detection of an intercurrent pulse.[13] This intermediate VT induction hypothesis is problematic for the 5 basic reasons discussed in the online-only Data Supplement.

Confusing Electrically Induced VF With Ischemically Induced VF

Ischemically induced VF is more difficult to defibrillate than electrically induced VF, which is reliably reversed with defibrillation.[16] With any chest compressions, defibrillation has a 90% success rate after 10 minutes of electrically induced VF with ≤3 shocks.[17] Hence, the failure of prompt defibrillation tends to exculpate an electric cause for VF.

Misappropriating Normal Clinical Judgment to a Violent Arrest

A cardiologist seeing a patient in the clinic typically has a respectful, cooperative, and peaceful subject. The subject of a violent law-enforcement encounter is often antisocial, defiant, violent, intoxicated, or schizophrenic. The rate of mortality during a clinic visit is essentially zero, whereas that of a violent arrest is 1 in 1000. As mentioned above, sudden falls and nonresponsiveness have different positive predictive values in an arrest compared with in the clinic. The use of subjective "clinical impression" is a major cause of erroneous case reports involving arrest-related deaths.

Postulated Diagnosis of Exclusion

Another common error is to blame the ECD because other typical causes of death (eg, a drug overdose) are not present. This is erroneous for 3 reasons:

1. Arrest-related death is a well-recognized syndrome often with no clear single pathological mechanism.[18,19]
2. The majority of arrest-related deaths do not involve an ECD.[19,20]
3. The battery-operated ECD satisfies all relevant safety standards, including those for electric fences, and thus its inclusion should be questioned and its exclusion favored.[21,22]

Procedure

We searched for published case studies reporting a cardiac arrest after an ECD application and found 12 such incidents.[8–13]

We obtained autopsy reports, emergency medical services run sheets, law-enforcement records, medical records, and deposition transcripts. We then used objective electronic records such as the ECD download (with clock drift correction), radio logs, 9-1-1 dispatch records, and audio and video recordings to build detailed timelines of each incident. All such data were released by the subject or family as part of litigation. In the majority (6 of 9) of fatal cases (cases 6 and 8 through 12), myocardial tissue was analyzed by a cardiac pathologist (J.R.S.).

It has been suggested that case reports of adverse reactions can be analyzed most objectively by the use of a Naranjo-style algorithm.[23] Such a methodology assigns points for each of a set of predictors to provide a consistent framework across all cases. We scored the cases by the criteria listed in Table 1. If a criterion favored ECD-induced cardiac arrest, it was scored as +1; if not it was scored as −1. If the value for the criterion was unknown or inapplicable, it was scored as 0. The range of possible scores was from −7 (probably not ECD-induced) to +7 (probable).

A presenting cardiac rhythm of VF (or an automated external defibrillator finding of a shockable rhythm) was scored as +1 because asystole and pulseless electric activity are not inducible with electric stimulation.[24] Asystole is rarely confused by emergency medical services personnel—and very rarely if multiple leads are used.[25] Electrically induced VF has not been seen (in published studies) to deteriorate to asystole in swine in <20 minutes, and the median time for deterioration is 34 minutes.[26] Asystole and pulseless electric activity were scored as −1. For the 5 non-VF cases, the time from cardiac arrest to rhythm documentation was 6.3±3.7 minutes (range, 3.0–12.5), thus making deterioration from VF unlikely.

We used a liberal 8-mm criterion for the dart-to-heart distance because this was the maximum reported in swine for inducing VF (mean, 5.8±2.1 mm).[27] The Ideker group (Walcott, Kroll, Ideker, manuscript under review) has shown that swine are 3 times as sensitive to electric currents for the induction of VF as humans. This suggests a 3.1±1.1-mm distance in humans even with high catecholamine levels[28,29] (see the online-only Data Supplement for details). The dart-to-heart distance was calculated from autopsy reports, photographs, or cardiac magnetic resonance imaging. If it was unknown, it was scored as 0.

The presence of a pulse after the ECD application was scored as −1. Although false-negative pulse findings are common, false positives are not.[30] In 2 cases (Z0 and Z3), there were contradictory pulse findings; hence, this was scored as 0.

Loss of normal breathing in <1 minute and loss of agonal breathing in <6 minutes after the ECD application were scored as +1.[31,32] After a cardiac arrest, normal breathing ceases in 12 to 60 seconds.[31,32] However, some subjects will also have agonal breathing for a maximum total of 6 minutes.[33]

Successful prompt defibrillation (with ≤3 shocks) was scored as +1.[17] Findings of significant cardiac pathology or long QT (Z1) were scored as −1.

We also elected to include the medical examiner findings. Although not specialists in bioelectricity or electrophysiology, medical examiners tend to investigate arrest-related deaths carefully and have no financial bias. If the autopsy report blamed the ECD as a primary cause of death, this was scored as +1. If there was no autopsy (nonfatal case) or if the report stated that the ECD could not be eliminated, this was scored as 0; otherwise, it was scored as −1.

Table 1. Diagnostic Criteria for an Electrically Induced Cardiac Arrest by an ECD

Item	Cutoff Value	Notes
Presenting rhythm	VF	Asystole and PEA are not inducible with electric stimulation
Dart-to-heart distance	8 mm	The critical dart-to-heart distance is 5.8±2.1 mm in swine
Documented pulse	After ECD	
Cessation of normal breathing	60 s	Agonal breathing to 6 min
Success of prompt defibrillation attempts	10 min	Electrically induced VF is defibrillated with a 90% success rate at 10 min with any chest compressions
Cardiac pathology		Severe long QT included
Medical examiner finding		Did the medical examiner find the ECD to be the primary cause of death?

ECD indicates electronic control device; PEA, pulseless electric activity; and VF, ventricular fibrillation.

Table 2. Case Study Summary

Case	Author	Age, y/Race	Total Score	Death	Presenting Rhythm	Dart-to-Heart Distance, mm	Pulse Found	Breathing, min	Failure of Defibrillation	Medical Examiner Agreement	Cardiac Pathology	Medical Examiner's Primary COD	Notes
1	K.F.	14/Black	–2	N	VF	UNK	Y	4	Y	NA	UNK	NA	Required 4 shocks for defibrillation
2	S.N.	18/White	–3	N	A	60	Y	6	NA	NA	None	NA	
3	S.F.	25/Black	–2	Y	VF	>20	N	UNK	Y	N	Hypertrophy, fibrosis	Physiological stress of a physical altercation, including use of ECD, and underlying heart disease	
4	Z0	31/Black	–4	Y	A	No probes	UNK	3.5	Y	N	None on autopsy	Undetermined	Varying reports on pulse
5	Z1	48/White	–1	N	VF	No penetration	N	UNK	Y	NA	Long QT	NA	Officer did not look for breathing, just noted nonresponsiveness
6	Z2	17/Black	–3	Y	VF	Right side	N	4	Y	N	HCM*	Agitation and stress; ECD listed as only adding stress	Video shows subject walking during ECD application
7	Z3	17/Black	–2	Y	VF	50	UNK	4	Y	N	None on autopsy	Uncertain COD with acute alcohol toxicity contributing	
8	Z4	24/White	–5	Y	VF	Probes missed	Y	9	Y	N	LM*	Acute alcohol intoxication, recent physical exertion	
9	Z5	33/White	–4	Y	A	70	Y	6	N	N	ARVC*/ normal	Seizure disorder	
10	Z6	24/Black	–6	Y	A/PEA	36	Y	5	Y	UNK	LM*	Nebulous autopsy report; could not rule out excited delirium or ECD in deposition	
11	Z7	16/Black	–3	Y	VF	55	N	See text	Y	N	ARVC*	ARVC with ECD being contributory	Agonal breathing at 8 min
12	Z8	23/White	–7	Y	A	50	Y	13	Y	N	LM*	Arrhythmia caused by epinephrine surge and respiratory issues related to struggle.	Medical examiner reported interstitial fibrosis

A indicates asystole; ARVC, arrhythmogenic right ventricular cardiomyopathy; COD, cause of death; ECD, electronic control device; HCM, hypertrophic cardiomyopathy; LM, lymphocytic myocarditis; PEA, pulseless electric activity; UNK, unknown; and VF, ventricular fibrillation.

*Represents findings of a cardiac pathologist (J.R.S.).

We did not score the presence of prescribed or illegal drugs because that was covered somewhat in the autopsy findings. We also did not score the duration of ECD application because no cases had durations long enough (>90 seconds) for a reduction in the VF threshold.[34]

Results

The results are summarized in Table 2. With 1 exception (case 8), all subjects had at least 1 probe (or drive-stun electrode) embedded in or in contact with the anterior thorax. The distances of the closest electrode to the ventricular epicardium are given in Table 2 in the dart-to-heart distance column.

Case 1 (K.F.)

The oldest case (K.F.) was reported as follows[8]: "An adolescent was subdued with a TASER stun gun and subsequently collapsed. Paramedics found the adolescent to be in ventricular fibrillation and began performing cardiopulmonary resuscitation within two minutes after the collapse."

The violent psychiatric subject, a ward of the state, had a psychotic episode, punched through a glass door, and gave himself significant lacerations requiring emergency care. Paramedics and police were called. The subject refused medical care and jumped at a police officer, who then used an ECD to successfully

control him. The subject had effective loss of muscle tone and collapsed to the ground. This was misinterpreted as cardiovascular collapse in the case report. The subject was handcuffed to the paramedics' gurney and then stopped responding to communication. Whether he was faking or had fainted is not clear. Paramedics checked his pulse and respiration with a 15-second vitals check and recorded them as normal, with "breathing properly," respirations of 16 per minute, and a pulse of 100 bpm.

The subject was taken via elevator to the ground floor and placed in the ambulance for treatment of his lacerations, where a second vitals check was taken. Vitals again were found to be normal. Spontaneous VF followed, and the subject was defibrillated with 4 shocks after atropine, epinephrine, and chest compressions for postshock asystole. The delay to VF is an issue of controversy, lacking objective electronic records, but was clearly >2 minutes because of the number of intervening activities.

The cardiac rhythm strip shown in the letter (ostensibly demonstrating a return to sinus rhythm by a defibrillation shock) was cropped after what were actually 3 premature ventricular contractions followed by asystole, as shown in Figure 1. This report has been questioned elsewhere.[35]

Case 2 (S.N.)

This case was published twice by members of the same emergency department with contradictory statements.[9,10] The alcohol- and tetrahydrocannabinol-inebriated subject presented with asystole ≈6 minutes after his ECD application, which is consistent with his extreme blood alcohol concentration at 80% of the mean lethal level.[36] His mother was present throughout the incident (in her kitchen); thus, it is unlikely that the officers merely ignored any earlier loss of breathing or cyanosis. The asystole was later converted to VF with epinephrine, atropine, and chest compressions, whereupon he was defibrillated. The first publication correctly identified a presenting rhythm of asystole (consistent with its emergency medical services documentation in 3 leads).[9] The second publication incorrectly claimed it was VF.[10]

Case 3 (S.F.)

This case came out of a retrospective review of 200 ECD-involved arrest-related deaths from 2001 to 2008 with a methodical analysis of the 58 cases in which the presenting rhythm was ascertainable.[11] The authors (including D.R.L.) concluded that "only one death was suggestive of electrically induced VF." However, the probes landed in the electrically insulating sternum; thus, the dart-to-heart electric path distance was >20 mm. The subject also failed prompt defibrillation from an automated external defibrillator in the squad car. The medical examiner noted cardiac hypertrophy and fibrosis. The autopsy report had findings of ethanol and tetrahydrocannabinol metabolite and noted a history of cocaine abuse.

Case 4 (Z0)

This case was presented in a conference debate and represents the only case suggesting that either a TASER model M26 ECD or a drive stun (direct contact without probes) could have caused a cardiac arrest.[12] Swine studies have not found VF inductions with drive stuns.[37] Even in probe mode with electrodes near the heart, the M26 ECD has not induced VF in small swine with epinephrine infusion because of its low net charge and high-frequency oscillations.[38] The presenter of case Z0 did not discuss or include this case in his later case series.[13]

Case 5 (Z1)

The subject was a long-time alcoholic who had hypokalemia, hypomagnesemia, hypocalcemia, acquired QT prolongation (he was taking olanzapine for his schizophrenia), and tetrahydrocannabinol and alcohol intoxication (blood alcohol concentration, 0.35%, which is at the mean lethal level).[36] During his hospital recovery, his QT was 540 to 560 milliseconds. No ECD probes penetrated his skin, but some control was achieved by arcing through his shirt. There was no evidence of cardiac effects during the first 2 ECD applications, so it is unlikely that the third (with probes in the same locations) would have caused a cardiac arrest.[39]

Case 6 (Z2)

The incident was recorded on a store security video. The individual walked around during a 37-second ECD application (to his right chest), which is not consistent with hemodynamically unstable high-rate cardiac capture or VF. The probes penetrated only to a depth of 3 mm. Only a partial drug screen was performed despite the subject having 3 baggies of drugs in his socks. A cardiac pathologist (J.R.S.) diagnosed hypertrophic cardiomyopathy, but the published account states that a "plaintiff pathologist" found a normal heart.[13] Court records show that

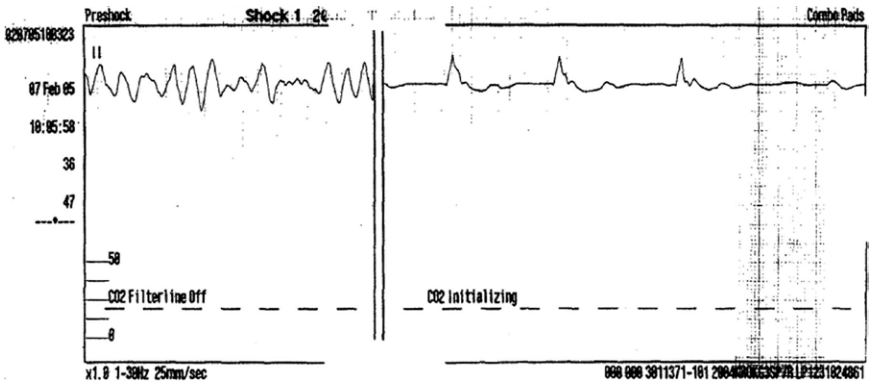

Figure 1. Actual strip of first shock in case 1 (K.F.) showing 3 premature ventricular contractions after shock followed by asystole.

the plaintiff pathologist was a person with an MD degree but no medical license and no board certifications.[40] This person testified that he has never called himself a pathologist, is not a forensic pathologist, and is not a cardiovascular pathologist.[40]

Case 7 (Z3)

The young subject was intoxicated with a blood alcohol level of 0.22% after binging on caffeinated alcoholic beverages now withdrawn from the market. The autopsy also found tetrahydrocannabinol metabolite. The subject had a radial and carotid pulse and was breathing for 4 minutes after the ECD application.

Case 8 (Z4)

The officer attempted to use an ECD to control an unsteady, extremely intoxicated man (blood alcohol concentration, 0.34%) but missed with the ECD probes, perhaps because the individual passed out and fell at the same time. This is clearly demonstrated by several items of objective evidence. The ECD probe has a channel near the back where it receives the wire much like the eye of a needle. Because the wire is only knotted, not soldered, there is substantial internal splatter, which is easily seen on scanning electron microscopy when the current is passed. The absence of such splatter demonstrated that no electric current was passed (Figure 2). In addition, a wire was broken, and microscopic analysis showed no arcing at the break (Figure 3), which again demonstrates that no current was passed. The ECD had a video camera, and this also showed that no probes contacted the subject's chest (see the online-only Data Supplement).

A police officer and a licensed emergency medical technician on scene verified a pulse and respirations for 9 minutes after the postural collapse.[41] A cardiac pathologist (J.R.S.) found lymphocytic myocarditis, but the published account states that a plaintiff pathologist found no specific pathology.[13] The court records show that the plaintiff pathologist is not board certified in pathology and lists himself as a general practitioner in state licensing records.[42]

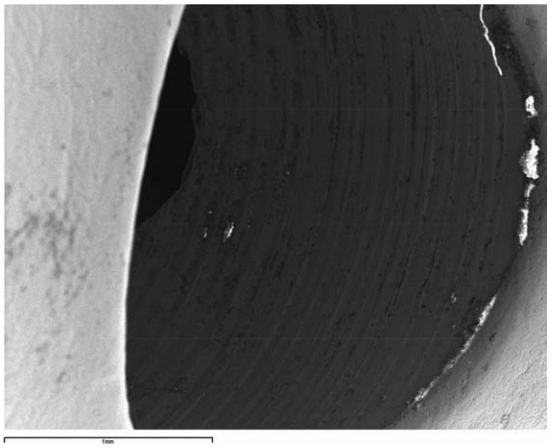

Figure 2. Scanning electron microscopy of the electronic control device probe-wire passage for case 8 (Z4) showing no arcing splatter. Only machining marks and nonconductive contamination (bright areas) are shown.

Case 9 (Z5)

A trooper used his ECD to prevent a 100-kg (220-lb) postictal hit-and-run driver from running into freeway traffic. The case series report states that there were 62 seconds of ECD shocks, but this is based on the "trigger pull" record of the weapon and does not represent actual seconds of current delivery.[13] There appears to have been an initial delivery of 14 seconds of current to the subject's chest with no cardiovascular effect because the subject kept struggling for several minutes. The trooper was then unable to gain control, but a citizen driving by, who happened to be a physician, stopped and helped the trooper with the physical struggle to control the subject. There were 9 nonproductive trigger pulls (probably resulting from "sympathetic" trigger-finger contractions during the struggle) because a wire had broken when the subject rolled on the pavement. (Broken wires were found at the site.) This was followed by 2 drive stuns to the leg. The probe closest to the heart was lodged in the left shirt pocket and probably did not penetrate the skin.

The presenting rhythm was reported in the case series as fine VF/asystole. In fact, the firefighters' LifePak defibrillator found only asystole as the rhythm ≈5.5 minutes after the radio call was made reporting that the subject was nonresponsive. An ambulance crew arriving shortly later also found only asystole. The emergency department also recorded only asystole; their first strip is shown in Figure 4. The only reported rhythm in this incident was asystole.

One of us (J.R.S.) diagnosed arrhythmogenic right ventricular cardiomyopathy, but the subject's family retained a cardiac pathologist who reported a normal heart. Thus, this case was scored as 0 for cardiac pathology. The medical examiner ruled that the cause of death was seizure disorder.

Case 10 (Z6)

The presenting rhythm was reported as VT/VF.[13] The defibrillator annotated "VFIB/VTACH" at a point (Figure 5). The Philips device classifies a wide-complex rhythm >120 bpm lasting >4 seconds as VT/VF; thus, the VT annotation is suspect. Post hoc frequency spectrum analysis suggests that this tracing represents a 129-bpm cardiopulmonary resuscitation artifact; there were intercurrent chest compressions. Before

Figure 3. Microscopic image of a broken wire for case 8 (Z4) showing no arcing.

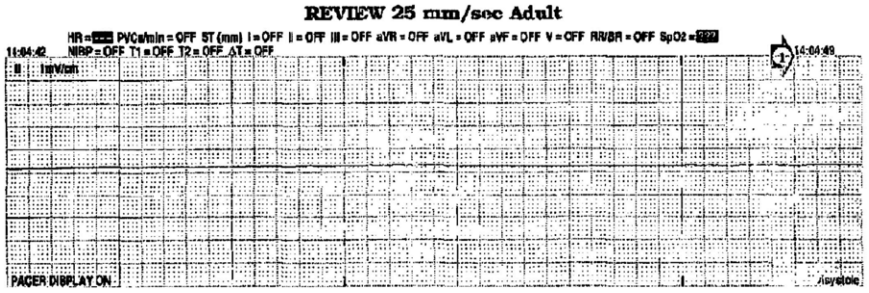

Figure 4. First recorded rhythm strip in the emergency department for case 9 (Z5).

and after this tracing, there were "pads off" warnings and clear tracings of loose-electrode noise (see strips 1 and 3 in the online-only Data Supplement). Spectrum analysis suggests that these strips represented noise on top of probable asystole, not VF.[43]

Case 11 (Z7)

The subject ran from police for 265 meters and went up and down stairs before being subdued by an ECD. About a minute later, he was nonresponsive, but cardiopulmonary resuscitation briefly restored breathing and a carotid pulse. Emergency medical services noted agonal breathing (2 breaths per minute) at 8 minutes after the ECD application. Defibrillation (4 shocks) resulted in both asystole and VF. The autopsy report noted arrhythmogenic right ventricular cardiomyopathy and tetrahydrocannabinol metabolite.

Case 12 (Z8)

The case report stated that the subject was "said to be breathing."[13] In fact, a police dashboard camera video recording shows breathing at 14 breaths per minute until ≈13 minutes after the ECD discharge, which clearly eliminates agonal breathing.[32,33]

The medical examiner found that the sternum was between the ECD probe and the heart, thus precluding a sufficiently short current path for VF induction.

Summary of Results

The mean totaled score for this study cohort was −3.5±1.8 (range, −7 to −1). There were no positive scores.

Many of the case reports confused a postural collapse or syncope with a cardiac arrest, which is problematic because several cases involved extreme alcohol intoxication. In 9 cases, there was no consideration of the time to breathing cessation, which is critical because the mean time was 6.1±3.1 minutes compared with the maximum of 60 seconds ($P<0.0001$ by t test) for normal breathing after a cardiac arrest.[31,32] There was typically no mention (9 cases) of the failure of prompt defibrillation as indicating a nonelectric source for the cardiac arrest.[16,17]

Almost all cases (11 of 12) ignored the critical dart-to-heart distance, which is well established in the literature for the induction of VF.[27] The unsupported implied inclusion criterion of any chest ECD exposure (regardless of the dart-to-heart distance) was surprising in view of the negative epidemiological association found between ECD chest exposures and mortality.[44,45] Bozeman et al[44] reported that 49% (424 of 874) of probe-mode cases involved a probe in the chest (not to be confused with the much smaller percentage of total probes found in the chest). White et al[45] found that only 36% (57 of 158) of ECD-involved arrest-related deaths had a chest probe ($P=0.004$ by χ^2), thus disproving the hypothesis that an application anywhere on the chest presents a risk of VF.

A surprising finding was that none of the fatal case-report authors gave any weight to the opinions of the medical examiners (7 were board certified in forensic pathology, 1 was certified in pathology, and 1 had training but no certifications). In 8 of those cases, the medical examiner did not list the ECD as a primary cause of death; a single case (Z6) had a nebulous autopsy report. Is the implication that forensic pathologists are truly unqualified to rule on cases of possible electrocution?

Discussion

The main findings of the study are as follows:

1. The demonstrated incidence of ECD-induced cardiac arrest is extremely low, if not zero.
2. Conclusions of a connection between ECD use and cardiac arrest are speculative at best.
3. The role of several non-ECD confounding factors explaining cardiac arrests are not accounted for in published case reports.

Although controversial case reports may generate useful discussion, they bring with them a great deal of personal interpretation and speculation by their authors. They need to be

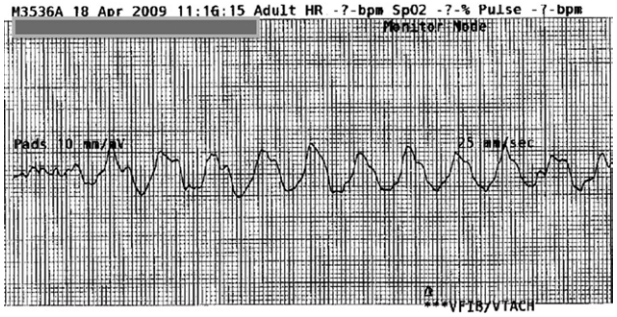

Figure 5. Strip 2 showing 129-bpm cardiopulmonary resuscitation artifact (on pads) incorrectly interpreted as ventricular tachycardia in case 10 (Z6).

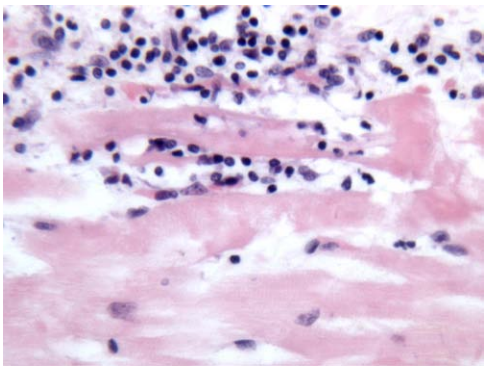

Figure 6. Histological image of a hematoxylin and eosin–stained slide of the heart from case 10 (Z6) showing lymphocytic myocarditis.

ratified by properly conducted studies for these hypotheses to become scientific facts. It is sobering to see that some of these published cases omitted dispositive facts (eg, documented later pulse, videotaped breathing 13 minutes later, and hard forensic evidence of missed probes). This is, however, consistent with the observation that adverse-event case reports are often poorly peer reviewed and may contribute more harm than good.[23]

Our scored analysis suggests that the authors of these case reports have not met their evidence burden because none of the cases had a positive score. Even if one would disagree with a given criterion, it would not change (with a single exception) the results to positive scores. Except for Swerdlow et al,[11] no authors described a systematic methodology to select cases and to maximize objectivity.

A key finding was that the majority (7 of 9) of fatal cases had significant cardiomyopathies; myocarditis was the most common. The lymphocytic myocarditis in these cases was characterized by multiple areas of infiltration by lymphocytes with injury to the cardiac muscle, as shown in Figure 6. The mean age of our cases was 24.2±9.6 years, which overlaps military training. Among the fatal cases, the incidence of myocarditis (3 of 9) was similar to that in case series of military recruit training sudden deaths. Amital et al[46] had 14 of 104 cases (P=NS by χ^2), and Phillips et al[47] had 8 of 53 cases (P=NS) with myocarditis.

Although there have been suggestions that a law-enforcement officer armed with an ECD must have an automated external defibrillator readily available, there was no incident in which an automated external defibrillator was, without advanced cardiac life support measures, successful in resuscitating a subject.[8] On the contrary, the failure of prompt defibrillation was a hallmark of these cases.

Current ECDs satisfy all relevant electric safety standards, including those for electric fences.[21,22] Human echocardiographic studies have not found cardiac capture with precordial electrodes with any commercially available ECD.[48–50] These data suggest that the threshold of factual evidence for blaming a cardiac arrest on an ECD should be set very high. The published case reports have not met that threshold.

Conclusion

A Naranjo-style case report scoring demonstrates the unreliability of case reports in identifying an ECD as the cause of cardiac arrests. This is consistent with the fact that existing ECDs satisfy all relevant electric safety standards, thus making electrocution extremely unlikely.

Disclosures

All authors have been expert witnesses for TASER International, Inc, which also provided funding for this work. Drs Kroll and Luceri are members of the TASER International, Inc Scientific and Medical Advisory Board. Dr Kroll is a member of their corporate board.

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Response to Kroll et al

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TASERS can cause cardiac arrest. My assignment was to validate this assertion, not to litigate these cases in *Circulation*. The courts are doing that. Kroll et al's case summaries attempt to try the cases but contain many misleading statements. For example, Kroll et al state that case Z1 had electrolyte abnormalities and long QT. True, but only after 3 TASER shocks, 5 automated external defibrillation attempts, and intravenous drugs and during profound cerebral hypoxia. Other assertions must be countered. Kroll et al stress an "8-mm criterion for the dart-to-heart distance" for TASER-induced VF to occur. False. This is based on 1 pig study in which a blunt dart was advanced through the anterior chest wall of 5 intubated pigs premedicated with tiletamine (a dissociative anesthetic) and isoflurane "to induce a surgical plane of anesthesia." Pigs received current from the X26 TASER lasting "≈5 seconds." Cardiac capture could not be determined because no ECG recording was done during the shock, only after stimulation to "check if the heart was beating normally." Dart-to-heart distances >8 mm have clearly caused cardiac capture in humans and produced VF in animals (see my article and the figures for cases Z3, Z7, and Z8). Kroll et al assert, "Failure of prompt defibrillation tends to exculpate an electric cause for VF." False. TASER-induced VF lasting 8 to 10 minutes can require repeated defibrillation and resuscitation attempts, especially if underlying heart disease is present. Unfounded entries invalidate the use of any scoring system. Other misleading or erroneous assertions by Kroll et al will be addressed in future venues.