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Electromuscular Incapacitating Devices Discharge and Risk of Severe Bradycardia

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Abstract: Electromuscular incapacitating devices (EMDs) are high-voltage, low-current stimulators causing involuntary muscle contractions and sensory response. Existing evidence about cardiac effects of EMD remains inconclusive. The aim of our study was to analyze electrocardiographic, echocardiographic, and microvolt T-wave alternans (MTWA) changes induced by EMD discharge.

We examined 26 volunteers (22 men; median age 30 years) who underwent single standard 5-second duration exposure to TASER X26 under continuous echocardiographic and electrocardiographic monitoring. Microvolt T-wave alternans testing was performed at baseline (MTWA-1), as well as immediately and 60 minutes after EMD exposure (MTWA-2 and MTWA-3, respectively).

Mean heart rate (HR) increased significantly from 88 ± 17 beats per minute before to 129 ± 17 beats per minute after exposure ($P < 0.001$). However, in 2 individuals, an abrupt decrease in HR was observed. In one of them, interval between two consecutive beats increased up to 1.7 seconds during the discharge. New onset of supraventricular premature beats was observed after discharge in 1 patient. Results of MTWA-1, MTWA-2, and MTWA-3 tests were positive in one of the subjects, each time in a different case.

Standard EMD exposure can be associated with a nonuniform reaction of HR and followed by heart rhythm disturbances. New MTWA positivity can reflect either the effect of EMD exposure or a potential false positivity of MTWA assessments.

Key Words: electromuscular incapacitating devices, heart rate, heart rhythm, microvolt T-wave alternans, sudden cardiac death, ventricular fibrillation

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Electromuscular incapacitating devices (EMDs) are frequently used by law-enforcement officers or private persons worldwide. They are high-voltage (peak voltage, 1200 V), low-current (peak current, 3 A) stimulators that cause involuntary muscle contractions in combination with sensory response. The electrical stimuli are in the form of high short-duration (10–100 μseconds) and repetitive (10–19 per second) pulses.[1]

Existing data concerning adverse cardiac events of EMD including incidental deaths are still inconclusive. A number of animal[2] and human[3–7] studies have reported relative safety of EMD exposure. In contrast, some reports of severe adverse events including ventricular fibrillation[8–13] or other effects on myocardium[14] from EMD application exist.

Heart rate (HR) acceleration before and after TASER exposure has been repeatedly described.[4,5,15,16] However, it is difficult to determine exact HR dynamics during exposure and whether the EMD application caused cardiac electrical capture owing to the fact that EMD discharge causes substantial electrical interference with the electrocardiographic (ECG) recording. Although clearly visible abnormalities in ECG after EMD application have not been described,[15] very little is known about subtle repolarization changes in relation to EMD. Such changes could be revealed by using microvolt T-wave alternans (MTWA). This method allows us to reveal a subtle ECG pattern in which the amplitude of T-wave varies every other beat, and it is linked to arrhythmogenic mechanism.[17]

The aims of our study were to analyze ECG, echocardiographic, and presence of MTWA before, during, and after EMD exposure and to evaluate the potential cardiac electrical capture during TASER application using continuous echocardiography.

MATERIALS AND METHODS

We analyzed ECG recordings and MTWA tests in a group of healthy volunteers who underwent single standard 5-second duration exposure to TASER X26 (Taser International, Scottsdale, Ariz) discharge. The study protocol took place in parallel with routine training of EMD user license. The EMD energy application was performed by an authorized TASER instructor who was responsible for training according to standard training protocol. Independently of the training procedures, we obtained a detailed medical history of our volunteers and performed a baseline physical examination. In addition, we recorded a standard 12-lead ECG as well as 2-dimensional and Doppler echocardiography. The study criteria excluded subjects with potentially significant clinical abnormalities as detected by these methods. However, none of the study participants had a relevant ECG and/or echocardiographic pathology. The local ethics committee gave us approval of the protocol. All subjects signed an informed consent to the study. Only healthy volunteers older than 18 years were included in this study. The following study exclusion criteria were set: family history of sudden cardiac death, personal history of structural heart disease and serious cardiac arrhythmias, history of neurological or psychiatric disease, cardiac medication use, drug or alcohol abuse, as well as pregnancy.

Exposition to TASER Energy

The EMD energy was applied in supine position with arms parallel to the side of the body. The EMD probes were manually positioned on the subject's body in predefined positions: 1 electrode in the second intercostal space in the right midclavicular line and second electrode in the fifth intercostal space in the left anterior axillary line. The distance between the probes was at least 30 cm. The probes were attached to the skin with a plaster to achieve maximal (probe to skin) contact. The single standard 5-second duration exposure by TASER X26 was delivered. The subject was maintained in the lying position until sinus tachycardia, rhythm disorders, or breathing changes disappeared, at least

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for 3 minutes. Upon completion of the application protocol, the electrodes were removed and the site of contact was checked for possible injuries.

The Study Protocol

The analysis of digital recordings of standard 12-lead ECG was performed. The recordings were monitored using Prucka Cardiolab 7000 (GE Healthcare, United Kingdom). The baseline ECG was analyzed at the beginning of all procedures during standard physical examination. Periprocedural continuous 12-lead ECG measurement was recorded. The recordings were initiated 1 minute before exposure and concluded at the end of the third minute after EMD energy delivery. Another ECG measurement was recorded 60 minutes after the EMD shock delivery. Heart rhythm, heart rate (HR_{ECG}), conduction intervals, P wave, QRS complex, ST-T segment morphology, as well as arrhythmia occurrence were analyzed.

Echocardiography (Vivid q; GE Healthcare, United Kingdom) performed to reveal potentially significant clinical pathology before including the subjects in the study. Therefore, echocardiography was used for documentation of echocardiographic HR (HR_{ECHO}) during TASER discharge during which frequent electrical artifacts occur and make ECG analysis impossible. Cine loops were obtained in modified apical 4-chamber or subcostal view in lying subjects. The recording was initiated 5 seconds before discharge and terminated 10 seconds after discharge. The HR_{ECHO} was calculated as the frequency of systolic contraction of the lateral wall of the left ventricle during a 5-second period of EMD exposure. Owing to limited echocardiographic window, no other parameters were consistently documented during the discharge.

Simultaneously, MTWA testing by treadmill exercise was performed at baseline (MTWA-1), immediately, and after 60 minutes after stun gun delivery (MTWA-2 and MTWA-3, respectively). The spectral method by graded exercise protocol was used. In brief, after careful skin preparation, high-resolution ECG leads (Cambridge Heart, Inc, Tewksbury, Mass) were placed on the standard precordial positions and in an orthogonal configuration. Exercise protocol consisted of gradually increasing workload so that constant HR_{ECG} was archived. The HR_{ECG} was measured between 100 and 110 beats per minute for 150 seconds; then subsequently, the HR increased to zone 110 to 120 beats per minute for 90 seconds. The MTWA test was interpreted with previously described criteria.[18] The test result was classified as positive, intermediate, or negative. *Positive result* was defined as the presence of sustained MTWA for at least 1 minute with alternans voltage of 1.9 μV or greater, K ratio less than 3, and an onset HR_{ECG} of 110 beats per minute or less in any orthogonal leads or in 2 adjacent precordial leads. *Negative test* was defined by unmet criteria for positivity in case HR_{ECG} was greater than 105 beats per minute. In case the test did not meet the criteria for positivity or negativity, it was classified as intermediate. Aside to standard classification, any presence of MTWA criteria was recorded.

Statistical Analysis

All continuous variables were expressed as mean ± standard deviation or as median with range for abnormally distributed variables. The Wilcoxon paired test was used to compare measured values when appropriate. *P* values less than 0.05 was considered to be significant. All analyses were performed with STATISTICA 6.1 package (StatSoft, Inc, Tulsa, Okla).

RESULTS

The data were obtained from 26 healthy volunteers (22 male and 4 female, mean age of 30 ± 8; median age 30, range 19 – 46 years). The baseline clinical, ECG, and demographic data are shown in Table 1.

As indicated in Table 2, the median of HR_{ECG} immediately before EMD exposure increased significantly from baseline level ($P < 0.01$). Another considerable increase in HR_{ECG} was noted immediately after the EMD administration ($P < 0.001$ in comparison with preshock values). The mean HR_{ECG} decreased back to the baseline level in all patients within 3 minutes after the EMD exposure. The analysis of HR_{ECHO} was available only in 19 subjects (73%). Remaining recordings were invalid because of strong muscle contraction and loss of optimal acoustic window during the EMD exposure. Values of HR_{ECHO} identified by echocardiograph corresponded to those determined by ECG.

The HR_{ECG} changes in all subjects are shown in Figure 1. Out of the total cohort, 2 male subjects manifested a different pattern of HR_{ECG} dynamics during the discharge. In those subjects, HR_{ECG} paradoxically decreased during and immediately after the EMD exposure. The HR_{ECHO} profile was documented

TABLE 1. Clinical and Demographic Data (n = 26)

Male/female	22 (85%)/4 (15%)
Age, y	30 (19–46)
Baseline systolic/diastolic blood pressure, mm Hg	125 (105–140)/75 (65–90)
Body mass index, kg/m^2	25 (20–32)
Arterial hypertension	0 (0%)
Diabetes mellitus	0 (0%)
Hypercholesterolemia	2 (8%)
Current smokers/past smokers	2 (8%)/0 (0%)
Structural heart disease	0 (0)
Sinus rhythm at baseline	26 (100%)
Atrioventricular block or complete bundle branch block at baseline	0 (0%)
Incomplete right bundle branch block at baseline	5 (19%)
Presence of any VPB on baseline ECG	1 (4%)
Presence of any SPB on baseline ECG	0 (0%)
Repolarization abnormalities at baseline on ECG	0 (0%)
Local skin injury after EMD exposure	0 (0%)

Values are expressed as n (%) or median (range).

TABLE 2. ECG Parameters at Baseline, During, and After EMD Discharge

Parameter	Baseline (n = 26)	10 s Before EMD Discharge (n = 26)	10 s After the End of EMD Discharge (n = 26)	5 min After the End of EMD Discharge (n = 26)
HR_{ECG}, beats per minute	89 (65–125)	101 (71–133)*	130 (71–153)*,†	89 (55–113)
PR, ms	160 (120–190)	150 (134–180)	150 (130–180)	160 (134–190)
QRS, ms	90 (71–110)	87 (70–100)	85 (66–100)	90 (77–110)
QT, ms	340 (281–440)	340 (273–400)	320 (270–380)	340 (300–380)
QTc, ms	410 (349–462)	430 (375–477)	484 (365–605)	405 (349–467)
New onset of ECG changes	—	No	1 (4%) person manifested frequent SPB	No

Data are expressed as n (%) or median (range).

*$P < 0.01$ – comparison with baseline.

†$P < 0.001$ – comparison with value time point: imminent before EMD discharge.

in 1 of those 2 subjects. His HR_{ECHO} suddenly slowed down at the beginning of EMD energy administration and reached bradycardia with the longest interval between two consecutive beats of 1.7 seconds. Decrease in HR_{ECG} was followed by spontaneous rapid return to sinus tachycardia within the 30 seconds after exposure. In the second case, similar bradycardia was suspected, but the echocardiographic recording was not fully valid.

More details of serial ECG parameters are listed in Table 2. Frequent monomorphic ventricular premature beats (VPBs) with left bundle branch block morphology and inferior axis were noted at baseline in 1 case. Besides VPB absence at the 30-second period after EMD exposure, their frequency was not changed after the EMD shock delivery. The new onset of frequent supraventricular premature beats (SPBs) was detected after EMD exposure in 1 case. The SPBs were not accompanied by any symptoms and spontaneously disappeared within 5 minutes. No other changes in ECG parameters and morphology as well as no severe cardiac arrhythmias were found after the EMD exposure.

The MTWA protocol was performed in 21 (81%) volunteers. The detailed results are summarized in Table 3. Results of MTWA tests at the baseline (MTWA-1), immediately after (MTWA-2), and after 60 minutes after (MTWA-3) TASER shot gun delivery were negative in 16 (76%), 14 (67%), and 15 (71%) cases, respectively. All results of the MTWA tests 1, 2, and 3 were positive in 1 (5%) of 21 subjects, different person on each occasion. Remaining tests (24% of all tests) were indeterminate. The reason for the indetermination was low quality of signals in 10 cases and missing criteria of full negative or positive result in 3 cases.

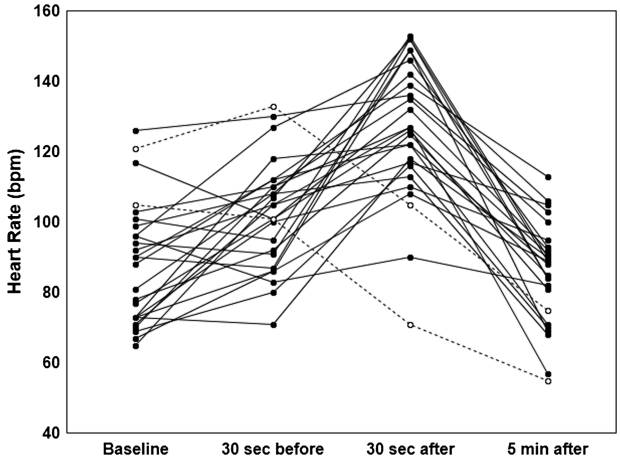

FIGURE 1. Heart rate profile in an individual. Heart rate was obtained from ECG recordings in predefined time points. The HR increased in majority of cases after EMD discharge (close circles and full line). Two abnormal case profiles are highlighted (open circle and dashed line).

DISCUSSION

The major finding of our study is the detection of nonuniform reaction of HR in response to EMD exposure. Although HR acceleration was present in the majority of subjects, bradycardia was detected during exposure to EMD in 1 case with echocardiography. In another subject, a similar decrease in HR was visible immediately after terminating the EMD energy administration in ECG. In addition, in 1 case, we observed a series of asymptomatic frequent SPB after EMD exposure. All 3 MTWA tests had positive results in one of the tested subjects, different person on each occasion.

It is generally accepted that increased HR is triggered by physiologic stress accompanied with adrenergic stimulation owing to an anticipated physical inconvenience.[4,5,15,16,19] This hypothesis is supported by previously described systolic blood pressure and minute ventilation increase imminently before stun gun discharge.[15,20] In our opinion, the onset of frequent SPB after TASER exposure in 1 case may have an analogous adrenergic mechanism.[19] The dynamics of SPB frequency and their disappearance during the subsequent observation correlated with HR decrease during postexposure observation period. Of note, another volunteer presented frequent fully asymptomatic VPB during both pre-EMD and post-EMD periods. This rhythm disorder has been classified as idiopathic ventricular ectopy of the right ventricular outflow tract origin before EMD exposure and was considered benign. Both stress and adrenergic activation with increasing HR are responsible for shift in frequency of VPB. To the best of our knowledge, only 1 case of VPB present before shock that resolved afterward has been previously reported.[16] It seems that, in individuals without any evidence of organic heart disease, the VPB and SPB dynamics observed are without clinical significance and that their occurrence is not related to increased risk of serious tachyarrhythmias after EMD application.

We speculated that the bradycardia observed in relation to EMD exposure is likely explained as a vagally mediated response induced by the profound muscular contraction (Valsalva maneuver equivalent) and resembles mechanisms of neurally mediated syncope.[21] We believe that the direct effect of high-frequency stimulation causing suppression of sinus node, the similar phenomenon known from the assessment of sinus node recovery time, is highly unlikely, but it cannot be definitively ruled out. Because of existing electrical interference with the ECG monitoring,

TABLE 3. Microvolt T-wave Alternans Test Results

MTWA Test (n = 21)	Test Negative	Test Indeterminate	Alternans Present Without Fulfilling Test Positivity	Test Positive	Maximal Negative HR_{ECG}, beats per minute	Alternans Onset, beats per minute
Baseline	16 (76%)	4 (19%)	2 (9%)	1 (5%)	114 (92–117)	114 (106–114)
Immediately after exposure	14 (66%)	6 (29%)	0 (0%)	1 (5%)	113 (107–118)	107
60 min after exposure	15 (71%)	5 (24%)	0 (0%)	1 (5%)	112 (97–117)	108

Data are expressed as n (%) or median (range).

only limited data are known about cardiac capture during EMD application. Under certain experimental conditions, the cardiac capture during EMD application has been detectable in an animal model.[22–29] Although the chest and heart of humans and pigs differ greatly, 1 case of cardiac capture phenomenon during EMD exposure has been detected by using echocardiography[16] and another case revealed cardiac capture in a patient with implanted pacemaker when the device was interrogated.[30] However, the causality of sudden death and EMD use is not exactly established. The presenting rhythm in sudden cardiac death proximate to the use of EMD was asystole or pulseless electrical activity more frequently than ventricular fibrillation.[10] Pulseless electrical activity was also more likely than ventricular fibrillation in animals that died after repeated long-duration EMD exposures.[31] That is why any information about observed incidental bradycardia during EMD exposure may be of practical value.

In agreement with previous data in human studies,[3–7,15,16] we observed neither clinically significant ECG morphology changes nor any clinically relevant arrhythmias detected after EMD exposure. Despite the observed increase in HR, our data fail to confirm the previously described sympathetically mediated slight shortening of PR and QT intervals after the stun gun discharge.[15] The observed trends in our study did not reach statistical significance.

The MTWA testing was used in our study as a method for detecting potential subclinical repolarization changes induced by EMD discharge. The value of MTWA in predicting ventricular tachyarrhythmia was shown in populations with structural heart disease.[32–39] Very few data are known about MTWA prognostic value in apparently healthy populations where positive MTWA has been sporadically detected with a prevalence range between 2% and 5%.[40,41] Such cases resemble one of our study participants who had a positive test result before the EMD discharge already. It is not clear whether the new onset of MTWA positivity detected in our study was given only by repolarization change after EMD exposure. Observation of the new MTWA positivity in 2 of 21 subjects may merely reflect a low reproducibility of MTWA assessment.[42] On the other hand, development of repolarization abnormalities after EMD exposure could reflect an objective physiological response. Even if we exclude a potential direct impact of EMD exposure, as discussed previously, both anger and stress followed by induction of a substantial sympathetic response may have direct impact on myocardial electrophysiological properties. It has been speculated that the repolarization heterogeneity is influenced indirectly by changes in the tone of sympathetic nervous system,[43,44] which can be potentiated by physical resistance.[19] This hypothesis is supported by the data that show that β-blockers are able to reduce MTWA positivity during anger in experimental animal studies.[45] The potential deleterious effects of sympathetic overstimulation are very obvious in stress-induced Tako-Tsubo cardiomyopathy, which is an example of heart functional deterioration with potentially malignant course.[46] Stress and its consequences can persist longer than any repolarization changes induced directly by cardiac capture; therefore, it would be inappropriate to conclude that EMD exposure cannot lead to delayed adverse cardiac consequences. This may be particularly important in settings where EMD is being used in subjects under drug influence and exposed to subsequent law-enforcement procedures. This may explain the occurrence of fatal outcomes occurring in time delay of several minutes after the EMD exposure.[47] However, it needs to be mentioned that MTWA testing may be unsuitable for proarrhythmogenic risk detection of EMD because this method predicts the risk of ventricular tachycardia and fibrillation not of asystole and pulseless electrical activity.

Study Limitations

There have been several limitations to our study. The extent of study population and its restriction to healthy and mostly well-trained individuals makes an extrapolation to field use of EMD very problematic. However, it is particularly difficult to get a significant group of volunteers in case of the anticipated inconvenience of the procedure. Any testing on subjects with any structural heart disease is not acceptable. Several technical difficulties should be mentioned. The character of artifacts did not allow continuous detection of ECG signals during the discharge. Our attempt to overcome this limitation by using echocardiography was limited by the restricted acoustic windows in subjects in lying position. Moreover, the muscle contraction during TASER exposure caused a misplacement of the probe in some subjects and it led to the loss of some data. The nature of the MTWA protocol leads inevitably to a delay (up to 10 minutes) between the discharge and acquisition of the first postshock recordings.

CONCLUSIONS

Standard EMD exposure was not associated with any clinically relevant ECG changes except the significant sinus tachycardia in the majority of subjects and the new onset of frequent SPB in 1 case, which was possibly induced by stress reaction due to stun gun shock. The observation of the 2 extraordinary cases in which the EMD discharge induced a brief but profound bradycardia possibly related to vagal stimulation by holding breath and muscular contraction is of particular importance. The new MTWA positivity detected in 2 of the 21 subjects after the EMD exposure may be caused by its direct effect on the myocardium or by sympathetic activation induced by stress, pain, and anger related to the procedure but may be also caused by a potential false positivity of MTWA assessment.

REFERENCES

1. Murray J, Resnick B. *A Guide to Taser Technology.* Whitewater, CO: Whitewater Press; 1997.
2. McDaniel WC, Stratbucker RA, Nerheim M, et al. Cardiac safety of neuromuscular incapacitating defensive devices. *Pacing Clin Electrophysiol*. 2005;28:S284–S287.

3. Levine SD, Sloane CM, Chan TC, et al. Cardiac monitoring of human subjects exposed to the Taser. *J Emerg Med*. 2007;33:113–117.
4. Vilke GM, Sloane CM, Bouton KD, et al. Physiological effects of a conducted electrical weapon on human subjects. *Ann Emerg Med*. 2007;50: 569–575.
5. Ho JD, Miner JR, Lakireddy DR, et al. Cardiovascular and physiologic effects of conducted electrical weapon discharge in resting adults. *Acad Emerg Med*. 2006;13:589–595.
6. Ho JD, Dawes DM, Reardon RF, et al. Echocardiographic evaluation of a TASER-X26 application in the ideal human cardiac axis. *Acad Emerg Med*. 2008;15:838–844.
7. Sloane CM, Chan TC, Levine SD, et al. Serum troponin I measurement of subjects exposed to the Taser. *J Emerg Med*. 2008;35:29–32.
8. Kim PJ, Franklin WH. Ventricular fibrillation after stun-gun discharge. *N Engl J Med*. 2005;353:958–959.
9. Sadhu S, Leal S, Herrera CJ, et al. Ventricular fibrillation and death after TASER injury. *Heart Rhythm*. 2006;3:S72–S73.
10. Swerdlow CD, Fishbein MC, Chaman L, et al. Presenting rhythm in sudden deaths temporally proximate to discharge of TASER conducted electrical weapons. *Acad Emerg Med*. 2009;16:726–739.
11. Schwarz ES, Barra M, Liao MM. Successful resuscitation of a patient in asystole after a Taser injury using a hypotermia protocol. *Am J Emerg Med*. 2009;4:515.
12. Naunheim RS, Treaster M, Aubin C. Ventricular fibrillation in a man shot with a Taser. *Emerg Med J*. 2010;8:645–646.
13. Zipes DP. Sudden cardiac arrest and death following application of shocks from a TASER electronic control device. *Circulation*. 2012;125:2417–2422.
14. Richards KA, Kleuser LP, Kluger J. Fortuitous therapeutic effect of Taser shock for a patient in atrial fibrillation. *Ann Emerg Med*. 2008;52:686–688.
15. Vilke GM, Sloane C, Levine S, et al. Twelve-lead electrocardiogram monitoring of subjects before and after voluntary exposure to the Taser X26. *Am J Emerg Med*. 2008;26:1–4.
16. Ho JD, Dawes DM, Reardon RF, et al. Human cardiovascular effects of a new generation conducted electrical weapon. *Forensic Sci Int*. 2010;204: 50–57.
17. Pastore JM, Girouard SD, Laurita KR, et al. Mechanism linking T-wave alternans to the genesis of cardiac fibrillation. *Circulation*. 1999;99: 1385–1394.
18. Bloomfield DM, Hohnloser SH, Cohen RJ. Interpretation and classification of microvolt T wave alternans tests. *J Cardiovasc Electrophysiol*. 2002;13(5):502–512.
19. Ho JD, Dawes DM, Nelson RS, et al. Acidosis and catecholamine evaluation following simulated law enforcement "use of force" encounters. *Acad Emerg Med*. 2010;17:60–68.
20. Dawes DM, Ho JD, Reardon RF, et al. The cardiovascular, respiratory, and metabolic effects of a long duration electronic control device exposure in human volunteers. *Forensic Sci Med Pathol*. 2010;6:268–274.
21. Bondar RL, Dunphy PT, Moradshahi P, et al. Cerebrovascular and cardiovascular responses to graded tilt in patients with autonomic failure. *Stroke*. 1997;28:1677–1685.
22. Hick JL, Smith SW, Lynch MT. Metabolic acidosis in restraint-associated cardiac arrest: a case series. *Acad Emerg Med*. 1999;6:239–243.
23. Dennis AJ, Valentino DJ, Walter RJ, et al. Acute effects of Taser X26 discharges in a swine model. *J Trauma*. 2007;63:581–590.
24. Nanthakumar K, Billingsley IM, Masse S, et al. Cardiac electrophysiological consequences of neuromuscular incapacitating device discharges. *J Am Coll Cardiol*. 2006;48:798–804.
25. Wu JY, Sun H, O'Rourke AP, et al. Taser dart-to heart distance that causes ventricular fibrillation in pigs. *IEEE Trans Biomed Eng*. 2007;54:503–508.
26. Roy OZ, Podgorski AS. Tests on a shocking device-the stun gun. *Med Biol Eng Comput*. 1989;27:445–448.
27. 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*. 2006;48:805–811.
28. Walter RJ, Dennis AJ, Valentino DJ, et al. TASER X26 discharges in swine produce potentially fatal ventricular arrhythmias. *Acad Emerg Med*. 2008;15:66–73.
29. Dawes DM, Ho JD, Cole JB, et al. Effect of an electronic control device exposure on a methamphetamine intoxicated animal model. *Acad Emerg Med*. 2010;17:436–443.
30. Cao M, Shinbane JS, Gillberg JM, et al. Taser-induced rapid ventricular myocardial capture demonstrated by pacemaker intracardiac electrograms. *J Cardiovasc Electrophysiol*. 2008;18:876–879.
31. Jauchem JR, Seaman RL, Fines DA. Survival of anesthetized Sus scrofa after cycling (7-second on/3-second off) exposures to an electronic control device for 3 minutes. *Am J Forensic Med Pathol*. 2011;32:124–130.
32. Hohnloser SH, Ikeda T, Bloomfield DM, et al. T-wave alternans negative coronary patients with low ejection and benefit from defibrillator implantation. *Lancet*. 2003;362:125–126.
33. Bloomfield DM, Steinman RC, Namerow PB, et al. Microvolt T-wave alternans distinguishes between patients likely and patients not likely to benefit from implanted cardiac defibrillator therapy: a solution to the Multicenter Automatic Defibrilator Implantation Trial (MADIT) II conudrum. *Circulation*. 2004;110:1885–1889.
34. Narayan SM. T-wave alternans and the susceptibility to ventricular arrhythmias. *J Am Coll Cardiol*. 2006;47:269–281.
35. Chow T, Kereiakes DJ, Bartone C, et al. Prognostic utility of microvolt T-wave alternans in risk stratification of patients with ischemic cardiomyopathy. *J Am Coll Cardiol*. 2006;47:1820–1827.
36. Chow T, Kereiakes DJ, Onufer J, et al. Does microvolt T-wave alternans testing predict ventricular tachykarrhythmias in patients with ischemic cardiomyopathy and prophylactic defibrillators? The MASTER trial. *J Am Coll Cardiol*. 2008;52:1607–1615.
37. Constantini O, Hohnloser SH, Kirk MM, et al. The ABCD (Alternans before cardioverter defibrillator) trial. *J Am Coll Cardiol*. 2009;53:471–479.
38. Gupta A, Hoang DD, Karliner L, et al. Ability of microvolt T-wave alternans to modify risk assessment of ventricular tachyarrhythmias events: a meta-analysis. *Am Heart J*. 2012;163:354–364.
39. Merchant FM, Ikeda T, Pedretti RF, et al. Clinical utility of microvolt T-wave alternans testing in identifying patients at hight or low risk of sudden cardiac death. *Heart Rhythm*. 2012;9:1256–1264.
40. Weber S, Tillmanns H, Waldecker B. Prevalence of T wave alternans in healthy subjects. *Pacing Clin Electrophysiol*. 2003;26:49–52.
41. Grimm W, Liedtke J, Muller HH. Prevalence of potential noninvasive arrhythmia risk predictors in healthy, middle-aged persons. *Ann Noninvasive Electrocardiol*. 2003;8:37–46.
42. Bloomfield DM, Ritvo BS, Parides MK, et al. The immediate reproducibility of T-wave alternans during bicycle exercise. *Pacing Clin Electrophysiol*. 2002;25:1185–1191.
43. Kovach JA, Nearing BD, Verrier RL. Angerlike behavioral state potentiates myocardial ischemia-induced T-wave alternans in canines. *J Am Coll Cardiol*. 2001;37:1719–1725.
44. Rashba EJ, Cooklin M, MacMurdy K, et al. Effects of selective autonomic blockade on T-wave alternans in humans. *Circulation*. 2002;105:837–842.
45. Klingenheben T, Gronefeld G, Li YG, et al. Effect of metoprolol and sotalol on microvolt T-wave alternans. Results of prospective, double-blinded, randomized study. *J Am Coll Cardiol*. 2001;38:2013–2019.
46. Kurisu S, Sato H, Kawagoe T, et al. Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. *Am Heart J*. 2002;143:448–455.
47. Ordog GJ, Wasserberger J, Schlater T, et al. Electronic gun (Taser) injuries. *Ann Emerg Med*. 1987;16:73–78.