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Electrical weapons, hematocytes, and ischemic cardiovascular accidents

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ABSTRACT

Background: There have been case reports following the use of a conducted electrical weapon (CEW) suggesting that these devices might affect coagulation or thrombosis in at-risk individuals. The aim of this manuscript therefore is firstly to explore this hypothesis by reviewing each of these cases and secondly to report the results of a prospective study exploring *a priori* the effects of electrical weapons on hematocytes in a group of human volunteers.
Methods: First, we systematically reviewed all cases of adverse outcomes following CEW discharge that could be due to an effect on coagulation or thrombosis, with particular focus on the clinical scenario and its relationship with the weapon discharge. Second, we assessed hematocyte levels in venous blood from 29 volunteers before, 5 min after, and 24 h after receiving a full-trunk 5-s TASER® X26(E) CEW exposure.
Results: Following extensive review of the literature, we found 3 relevant case reports of possible vascular thromboembolic clinical events after CEW exposure, specifically a case of ischemic stroke, and 2 cases of ST-segment elevation myocardial infarctions. Review of these published cases failed to establish a plausible linkage to the CEW beyond a temporal association with significant emotional and physiological stress from a violent struggle.
Our prospective study of biomarker change following CEW discharge revealed acutely increased values for WBC (white blood cells), specifically lymphocytes and monocytes, and a raised platelet count. Neutrophil levels decreased as a percentage of WBC. While these changes were statistically significant at 5 min, all results remained within established reference ranges. At 24 h, all values had returned to baseline except total WBC which decreased to slightly below baseline but was still within the normal reference range.
Conclusions: A review of clinical cases, of ischemic or thrombotic events revealed no direct association with the CEW discharge. A full-trunk electrical weapon exposure did not lead to hematocyte changes beyond normal clinically expected variations in similar acute response scenarios. The case report and biomarker data do not support the hypothesis that a CEW discharge is associated with changes likely to promote coagulation or thrombus formation.

1. Introduction

The handheld conducted electrical weapon (CEW) deploys small probes to deliver short-duration (50–100 μs) electrical pulses to control skeletal muscle contractions. This typically leads to a loss of regional muscle control and a fall to the ground to end a violent confrontation or suicide attempt. The benefits and primary complications of the CEW are well established in numerous large studies and papers as summarized in Table 1. The aim of the present report is to review and summarize reported cases of thrombotic or ischemic vascular events in people receiving a CEW discharge. There is substantial literature describing the possibility of electrocution (CEW-induced ventricular fibrillation) and this will not be reviewed in this paper.[1–4] We also present the results of a biomarker study that explored whether changes in hematocytes might underlie any temporal relationship between a CEW discharge and ischemic or thrombotic events occurring in the field.

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Table 1
Known benefits and complications of electrical weapons.

Benefits & Complications	n	Effect Size
Reduction in any suspect injury[32]	24 380	65%
Reduction in injury requiring medical attention[33]	16 918	78%
All-cause mortality reduction[4]		59–66%
Fatal shooting reduction[34]		67%
Fatal brain or neck injury from fall[4,35]	18	4.6/million
Fatal burn injury[4,36,37]	8	2.1/million
Blinding from probe injury[38,39]	20	5.1/million

1.1. Clinical cases

We have reviewed each of the 3 case reports of unexpected vascular sequelae temporally related to the use of a CEW and summarized them:

Case 1: Bell reported on a 32-year-old male who developed an ischemic stroke following electronic control.[5] He presented to the emergency department following a CEW discharge to the forehead during an altercation with the police. The patient became briefly nonresponsive during the incident. Upon arrival to the emergency department, the patient had a persistent change in mental status with speech difficulty. Physical examination revealed abrasions on the forehead from the probe and generalized right-sided weakness. The past medical history was significant only for bipolar and schizoaffective disorder. The initial work-up included normal electrolytes and normal sinus rhythm on electrocardiogram (EKG). Upon further evaluation, a computed tomogram (CT) of the head demonstrated a non-hemorrhagic acute infarct along the left middle cerebral artery (MCA) territory with surrounding edema and associated mass effect. CTA and MRI/MRA of the head and neck were subsequently performed which demonstrated filling defects in the distal M1 and proximal M2 segments of the left middle cerebral artery with restricted diffusion in the MCA territory. There were no other intracranial or cervical vessel abnormalities. Subsequent work-up including tests for infections, inflammatory states, drug screening and coagulation abnormalities were normal except for mildly decreased protein S levels. Transesophageal echocardiogram was normal and HbA1c and lipid studies were negative. History of tobacco use was the only relevant cardiovascular risk factor. Heart rhythm monitoring during the hospitalization was normal. Bell suggested that the temporal relationship of the development of a stroke and probe discharge on the head in an otherwise young healthy individual presented the possibility of electrical injury-induced stroke from a combination of vasospasm and endothelial thermal injury.

Case #2: Belen reported a 37-year-old male patient with no prior cardiac history who, after an altercation with the security personnel of a hotel, collapsed after application of a CEW in probe mode to the anterior thoracic wall with pain in all parts of the body and spasms.[6] One CEW probe entrance wound was 1 cm left of the left midclavicular line on the 6th intercostal space, and the other was 2 cm right of the anterior axillary line near the nipple level. While the patient was able to stand up with support, a crushing-like pain in the chest lasted for approximately 30 s following CEW application. The patient was admitted to the emergency service. EKG showed elevated ST segment in leads II, III and AVF, and reciprocal ST depression in leads I and AVL. The patient was conscious and had no past medical history or family history of relevance for ischemic heart disease. Physical examination showed no signs of traumatic injury or bleeding. The patient was transferred to coronary intensive care. Early laboratory tests were all normal including a drug screen. The patient was treated with aspirin, clopidogrel, isosorbide dinitrate, metoprolol, and diazepam (for sedation), and transferred for coronary angiography. Coronary angiography, 3 h after the CEW discharge, was normal and his further recovery was uncomplicated. The chest pain lasted for a total of 5 h, and the EKG normalized completely, while echocardiography showed minimal levels of inferior wall hypokinesia. The patient's cardiac markers peaked at 12 h: CK (1250 U/L), CK-MB (150 U/L), and troponin I (9 ng/mL). The patient was discharged on medication for secondary prevention and remained without symptoms at follow-up. In this case, it is possible that the effects of extreme stress could have led to a form of stress-related cardiomyopathy (similar to Takutsubo syndrome).[7]

Case # 3: Baldwin reported a case of a 20-year-old man involved in a brawl who received a TASER® X26 discharge to the upper right posterior part of the thorax and in the right buttock consisting of two 5-s activations of the device.[8] The man did not sustain a significant fall, but was brought to the hospital in custody an hour later to have the probes removed whereupon, having initially been pain free, he developed burning retrosternal pain accompanied by dyspnea. He was treated with aspirin and nitroglycerin paste, and his symptoms subsided. The initial EKG showed ST elevation in the inferior leads. Urgent coronary arteriography revealed normal coronary arteries but left ventriculography showed hypokinesis of the distal inferior wall, and a normal ejection fraction. Four hours after the onset of chest pain, the EKG showed evolution of an inferior infarct. The initial serum troponin was 0.66 ng/mL and peaked at 10.73 (reference < 0.04); serum creatine kinase (CK) was 373 U/L and peaked at 1016 (reference < 230); and CK-MB was 7.3 ng/mL and peaked at 52.5 (reference < 7.7). The patient was taking no prescribed medications, and a drug screen was normal, but he reported regular use of Finaflex (Redefine Nutrition), an over-the-counter anabolic steroid supplement for muscle building. There were no other risk factors for ischemic heart disease although throughout his hospitalization, his blood pressure was elevated in the range of 146/90–190/98 mmHg. He had no further symptoms, and he was discharged with secondary prevention medications and was advised to stop taking the anabolic steroid supplement.

This subject had been engaged in a physical altercation prior to his myocardial infarction.[9] Although a 10-s TASER exposure can lead to nominal cardiovascular response, this is much less than simulated combat and fleeing.[9] Furthermore, anabolic steroid use and other fat-burning supplements are associated with premature cardiovascular complications including acute myocardial infarctions or coronary vasospasm.[10] Nevertheless, the latter 2 reports hint at the possibility of acute myocardial injury due to coronary vasospasm in the presence of normal coronary arteries.

In addition to a case review, we also wanted to establish whether there are any effects on blood contents, specifically hematocytes, as a result of a CEW discharge that might predispose at-risk individuals to sustain coagulation or thrombotic-related sequelae.

2. Methods

2.1. Study design

Participants were cadets from the Austin (Texas) Police Academy who had previously volunteered to undergo CEW exposure. The CEW exposure was performed by Academy staff as part of their normal training methods to ensure that cadets were aware of the effects of the device. Thus, a primary inclusion criterion was that the subject was participating in police-officer use-of-force training which implied that they were capable of physical exertion. The study was approved by the Institutional Review Board of Texas A&M University. Written, informed consent was obtained from all volunteers confirming their ability to withstand physical exertion. Exclusion criteria were: recent illness, musculoskeletal injury, pregnancy, lactation, or any known cardiovascular, pulmonary, or hematologic condition. A 12-lead EKG was administered as part of the baseline evaluation before the CEW exposure.

2.2. CEW application

Each subject was positioned prone on a padded mat. The 2 alligator clips were connected to clothing at the subject's shoulder area and waist. Electrical current delivery, from an X26(E), lasted for a duration of a standard 5 s cycle (single pull of the trigger), as used in training and in the field.

2.3. Serum biomarker protocol

A 20 mL venous blood sample was taken before, 5 min after, and at 24 h following the CEW exposure. All phlebotomies were performed by certified emergency medical technicians using routine venipuncture practices, wherein a sterilized intravenous catheter was placed in the vein of the anterior forearm for ease and repeatability. All drawn blood specimens were labelled, collected, and transported to an off-site facility by an independent laboratory (Laboratory Corporation of America, Austin, TX).

2.4. Statistical analysis

Comparisons were done by a paired Student's t-test for both baseline to post and baseline to 24-h. Post-hoc analysis demonstrated that the study was powered to detect p = .05 difference with a 90% likelihood for each biomarker. Because of the many comparisons, we used the Holm-Bonferroni correction for significance testing. For baseline-to-post this only affected the MCHC and MCV comparisons as seen in Table 2. The Holm-Bonferroni correction affected the RBC and hemoglobin 24-h comparisons as also seen in Table 2.

3. Results

A total of 29 subjects (26 male) participated and provided blood samples before, 5 min after and 24 h after the CEW exposure. Subject ages ranged from 21 to 55 years.

At 5 min following the discharge, there were increases in total WBC, platelets, lymphocytes (% and absolute), and monocytes as shown in Table 2. The absolute neutrophil count was increased but the *proportion* of neutrophils decreased to due greater relative increases in lymphocytes (Table 3). At 24 h, the only change was a slight decrease in the total WBC from baseline (6.35 ± 1.26/nL vs 5.93 ± 1.41/nL, p = .002) whereas all other blood cell values had returned to pre-test values. Importantly, although these changes were statistically significant, the magnitude of change did not lead to a shift beyond normal values for any of the hematocyte subfractions.

The 7 biomarkers with post-exposure shifts are detailed in Table 3. The greatest shift was in the lymphocyte level which was 0.83 sigma (standard deviations of baseline) or 21%. Platelets were increased 0.16 sigma or 2%. All levels were within typical clinical reference levels.

4. Discussion

We believe that this is the first publication of the effects of modern electrical weapons on hematocyte concentrations in humans. Our study demonstrates that exist transient changes in hematocyte levels which were not of a magnitude for any of the cellular subfractions to take them beyond the normal range.

There are previous human reports of changes with hematocytes following CEW discharge. Dawes collated hematocrit levels taken from 4 studies of 133 volunteer subjects including some intoxicated and exercising subjects following TASER X26(E) discharges,[11] and reported a statistically significant, but clinically insignificant decrease in hematocrit. Another human study used the Condor® Spark, a much higher-powered weapon (~7 W vs modern CEWs < 2 W) than that used in Dawes' study and ours, and reported a rise in hematocrit in 71 volunteers.[12]

Our prospective human data confirm previous swine studies, all of which have shown no consistent changes on RBC, platelets, neutrophils, lymphocytes, hematocrit, and hemoglobin following X26(E) discharges.[13–16] The minor increases in platelets and lack of changes in RBC or hematocrit seen in our volunteers suggest that thrombus generation from a CEW exposure is unlikely, making an embolic event highly unlikely. Our previous meta-analysis of 10 studies with a total of 421 subjects found an increase in troponin levels in only a single subject (at 24 h linked to a rigorous off-protocol workout).[17,18] Vilke found no changes in the 12-lead EKG at 1-h post-CEW exposure and no troponin I levels above 0.2 ng/mL at 6 h suggesting that there is no direct deleterious cardiac effect.[19]

In the present study, we did see a shift in the neutrophil/lymphocyte ratio. A shift of the ratio in this direction, but of much greater magnitude has prognostic significance in ischemic heart disease, heart failure and patients with arrhythmias.[20] The changes we saw may be due to the effects of increased cortisol and catecholamines following physiological stress on lymphocyte number and function, although the magnitude of the changes suggests minimal stress from the CEW exposure.[21,22] This is supported by our data from a related study in volunteers (n = 31) where we reported mild increases in adrenergic and metabolic stress markers: serotonin, cortisol, and lactic acid after a CEW exposure.[23]

The lack of stress-related changes in these data and our previous work could be due to the fact that all currently available TASER brand CEWs deliver less than 2 W, which is considerably less than the 5–7 W allowed by the Underwriters Laboratories (UL) electric fence standard.[24] Modern CEWs also satisfy the conservative IEC (International Electrotechnical Commission) 2.5 W limit.[25–27] Moreover, there is also an

Table 2
Statistical significance by Holm-Bonferroni test.

Parameter	Units	Baseline	Post	T-test	HB Limit	SS?	24 Hour	T-test*	HB Limit	SS?
WBC	/nL	6.35 ± 1.26	7.02 ± 1.35	0.0000	0.0028	Y	5.93 ± 1.41	0.002	0.0026	Y
RBC	/pL	4.47 ± 0.45	4.47 ± 0.42	0.91	0.03		4.40 ± 0.45	0.022	0.0029	
Hemoglobin	g/dL	13.8 ± 1.1	13.8 ± 1.1	0.45	0.01		13.6 ± 1.0	0.045	0.0033	
Hematocrit	%	40.2 ± 3.3	40.3 ± 3.0	0.70	0.01		39.6 ± 3.3	0.10	0.004	
MCV	fL	90.1 ± 3.8	90.5 ± 3.8	0.032	0.0045		90.5 ± 3.3	0.13	0.004	
MCH	pg	31.0 ± 1.2	30.9 ± 1.2	0.12	0.01		31.1 ± 1.4	0.27	0.006	
MCHC	g/dL	34.4 ± 0.5	34.2 ± 0.6	0.007	0.0042		34.4 ± 0.7	1.00	0.05	
RDW	%	13.5 ± 0.7	13.5 ± 0.7	0.88	0.02		13.5 ± 0.7	0.85	0.025	
Platelets	/nL	226 ± 34	231 ± 35	0.002	0.0038	Y	227 ± 32	0.67	0.013	
Neutrophils	%	60.7 ± 8.7	58.4 ± 9.0	0.0001	0.0031	Y	59.6 ± 7.2	0.39	0.007	
Lymphs	%	28.8 ± 8.1	31.2 ± 8.9	0.0000	0.0029	Y	29.1 ± 6.5	0.73	0.017	
Monocytes	%	7.69 ± 1.97	7.69 ± 2.00	1.00	0.05		8.03 ± 2.57	0.22	0.005	
Eos	%	2.28 ± 2.02	2.14 ± 1.79	0.29	0.01		2.59 ± 2.24	0.017	0.0028	
Basos	%	0.52 ± 0.51	0.55 ± 0.51	0.75	0.01		0.62 ± 0.49	0.41	0.008	
Neutrophils (Absolute)	/pL	3.91 ± 1.07	4.17 ± 1.13	0.0003	0.0033	Y	3.60 ± 1.28	0.031	0.0031	
Lymphs (Abs)	/pL	1.78 ± 0.44	2.14 ± 0.51	0.0000	0.0026	Y	1.69 ± 0.37	0.058	0.0036	
Monocytes (Abs)	/pL	0.48 ± 0.11	0.54 ± 0.16	0.002	0.0036	Y	0.46 ± 0.13	0.16	0.005	
Eos (Abs)	/pL	0.16 ± 0.14	0.17 ± 0.15	0.33	0.01		0.15 ± 0.14	0.33	0.006	
Baso (Abs)	/pL	0.04 ± 0.05	0.05 ± 0.05	0.49	0.01		0.05 ± 0.05	0.57	0.010	

HB= Holm-Bonferonni. *From Baseline.

Table 3
Statistically significant changes from baseline to post-exposure by level and compared to reference limits.

Parameter	Units	Baseline	Post	*Reference Limits*	Delta	Z-score (Delta ÷ sigma)	Delta (%)
WBC	/nL	6.35 ± 1.26	7.02 ± 1.35	*6–18*	0.67	0.53	11%
Platelets	/nL	226 ± 34	231 ± 35	*140–400*	5.38	0.16	2%
Neutrophils	%	60.7 ± 8.7	58.4 ± 9.0	*45-70%*	−2.31	−0.27	−4%
Lymphs	%	28.8 ± 8.1	31.2 ± 8.9	*20-45%*	2.41	0.30	8%
Neutrophils (Absolute)	/pL	3.91 ± 1.07	4.17 ± 1.13	*1.8–7*	0.26	0.24	7%
Lymphs (Absolute)	/pL	1.78 ± 0.44	2.14 ± 0.51	*1–4*	0.37	0.83	21%
Monocytes (Absolute)	/pL	0.48 ± 0.11	0.54 ± 0.16	*0.2–0.8*	0.06	0.54	13%

electrical standard designed specifically for the CEW: ANSI CPLSO-17, requiring minimum outputs for effectiveness and maximum limits for safety which are satisfied by all current TASER® models. Our work was partly stimulated by the fact that these safety limits are based on the risk of VF and do not explicitly speak to coagulation or thrombosis.

Cardiac damage is sometimes seen with high-power electrical injury, and ST elevation has been reported with a lightning strike.[28–31] Although the low-power 2-W CEW effect on skeletal muscle activation appears dramatic, in the way a lightning strike might be imagined, the electrical power of the latter and power line injuries is much higher (1 kW–100 kW).

4.1. Limitations

It is possible that our search for vascular spasm or thrombotic events was incomplete and that we missed unpublished cases. We consider it unlikely however, that our approach would miss large numbers of these events, which are usually high-profile.

We only tested 29 individuals and because of the small sample of females, we were not able to make a baseline comparison between genders. Our prospective study did not measure fibrinogen or thrombin levels. We consider it unlikely that these humoral factors would be elevated — in isolation — in the absence of changes in cellular contents since changes in both are usual during and following spontaneous cardiovascular events. Our study was also unable to exclude stress-related vascular spasm as a result of the CEW discharge in susceptible individuals and although swine data have given no indication of this as a phenomenon, this could form the subject of a future study.

5. Conclusions

We have identified 2 cases in the literature of possible stress-related coronary artery vasospasm and although we could not exclude the TASER discharge as a contributory factor, the events leading up to each discharge were more likely to have played a dominant role. Our prospective data do not support the hypothesis that a CEW discharge is associated with changes likely to promote coagulation or thrombus formation.

Declaration of competing interest

MWK is a member of Axon's Scientific and Medical Advisory Board (SMAB) and corporate board. RML is a SMAB member and consultant to Axon. SNK is a SMAB member. KKW and JCC declare no conflicts. MWK and RML have served as litigation or inquest experts in multiple countries.

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