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ORIGINAL ARTICLE

The physiologic effects of a new generation conducted electrical weapon on human volunteers at rest

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Abstract

Axon Enterprise, Inc. (Axon) released its newest generation conducted electrical weapon (CEW), the T7, in October 2018. In order to compare the effects of this new CEW to prior generations, we used our previously described methodology to study the physiologic effects of CEWs on human volunteers at rest. This was a prospective, observational study of human subjects consisting of two parts. Part 1 was testing a single cartridge (2-probe) exposure. Subjects received a 10-s exposure from the T7 to the back with a 30 cm (12 in.) spread between the two probes. Part 2 was testing a simultaneous two-cartridge (4-probe) exposure. Subjects received a 10-s exposure from the T7 to the back with two cartridges with a 10 cm (4 in.) spread between each probe pair. The probe pairs were arranged cephalad to caudal such that the distance between the top probe of the first cartridge and the bottom probe of the second cartridge was 30 cm (12 in.). Vital signs were measured immediately before and after the exposure. Continuous spirometry was performed. ECG monitoring was performed immediately before and after the exposure. Venous pH, lactate, potassium, CK, catecholamines, and troponin were measured before and immediately after the exposure, at 1-h post-exposure, and again at 24 h. 11 subjects completed part 1 of the study. 9 subjects completed part 2 of the study. No subjects had a dysrhythmia or morphology change in the surface ECG. There were no statistical changes in vital signs pre- and post-exposure. While subjects did not have a statistical change in spirometry parameters pre-exposure to exposure except for a small drop in $PETCO_2$, there was an increase in minute ventilation after the exposure that could have several explanations. A similar pattern was seen with prior generation weapons. No subject had elevated troponin levels. Other blood parameters including venous pH, lactate, potassium, CK, and catecholamines had changes similar to prior generation weapons. Comparison of the data for the single-cartridge exposures against the simultaneous two-cartridge exposures yielded no difference in vital signs, but the minute ventilation was higher for the two-cartridge exposures. The blood data, where there was a difference, was mixed. In our study, the physiologic effects of the Axon T7 are modest, consistent with the electrically-induced motor nerve-driven muscle contraction, and were similar to prior generation weapons.

Keywords Axon · CEW · Conducted electrical weapon · Conducted energy weapon · Physiology · TASER

Introduction

Conducted electrical weapons (CEW) are now ubiquitous in policing in the United States (U.S.) and many western countries due to their unique ability to temporarily immobilize subjects from a distance. The primary mechanism by which Axon Enterprise, Inc. (Axon), formerly TASER International (TASER), Scottsdale, AZ, CEWs are thought to operate is through the electrical stimulation of motor neuron axonal projections to skeletal muscles. Motor neurons within sufficiently strong electric fields established by the wire-tethered probes are stimulated at a sub-tetanic rate (19–22 pulses per second (PPS) depending on the CEW) leading to involuntarily fused muscle contractions and therefore incapacitation of activated muscle groups [1]. Because CEWs electrically capture excitable tissues, most of the direct safety concerns have centered on direct cardiac stimulation, respiratory impairment from

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respiratory muscle contraction, and other physiologic effects related to sustained muscle contraction (e.g. acidosis, rhabdomyolysis, and hyperkalemia). Known, but more obvious direct risks, include dart puncture wounds (especially to sensitive structures such as the eye) and minor skin burns due to electrical arcing. Indirect risks include head injuries from unprotected falls, and the possibility of severe burns from the ignition of flammable materials. Concerns have also focused on the indirect risk of increased "stress" particularly in vulnerable subjects.

We have previously described a methodology for studying the physiologic effects of CEWs on human volunteers at rest [2, 3]. Axon released its newest generation CEW, the T7, in October 2018. In order to compare the physiologic effects of this new CEW to prior generations, we have used our prior method here. Because the T7 is a two-bay/cartridge weapon, we tested both single-cartridge (2-probe) and simultaneous two-cartridge (4-probe) exposures. This is the second study in the literature to study the effects of simultaneous two-cartridge exposures from a handheld CEW and the first to study the new T7.

Methods

This was a prospective, observational study of human volunteers consisting of two parts. Part 1 tested a one-bay exposure, and part 2 tested a simultaneous two-bay exposure with an over-all similar effective probe spread. The institutional review board at Hennepin County Medical Center (HCMC) (Minneapolis, MN) approved the study. The study was conducted at an Axon facility in Scottsdale, AZ on two different dates (one part per date). The subjects were a convenience sample of primarily law enforcement, military and security officers but also civilian volunteers. Subjects were compensated with an Axon CEW for their participation.

Subjects completed a medical screening questionnaire that was reviewed by a study physician and provided informed consent (an HCMC representative was brought in specifically to do the consenting process). Pregnancy was the only specific exclusion criterion.

A commercial scale (Omron Full Body Sensor HBF-5168, Omron Healthcare, Inc., Bannockburn, Illinois) was used to determine participant weight that was combined with stated height to determine body mass index (BMI).

Baseline creatine kinase (CK), potassium, pH, lactate, troponin, and catecholamines (epinephrine, norepinephrine and dopamine) levels were drawn. The pH, lactate, troponin, and potassium levels were immediately analyzed after the draw using the Abbott Point-of-Care i-STAT (East Windsor, NJ) and CG4, Chem8+, and cTnI cartridges. The blood was spun on site by a contracted laboratory technician, and delivered later to Lab Corps (Phoenix, AZ) for CK and catecholamines analysis (note in part 2 of the study, the cTnI cartridges were unavailable and troponin was sent to Lab Corps).

Subjects were laid supine on a padded table for testing. An "off-the-shelf" Axon T7 was attached by hand-placing the probes to full dart depth (11.5 mm). Axon engineers confirmed the CEW to be operating according to manufacturer specifications prior to testing. The top probe was placed 15 cm (6 in.) below the C7 prominence and 10 cm (4 in.) laterally on the back, and, in part 1, the bottom probe was placed 30 cm (12 in.) caudally. Laterality was alternated between subjects. The 30 cm (12 in.) distance was chosen based on prior work that showed that 30 cm (12 in.) was the minimum spread between the probes that would successfully incapacitate a human subject [4]. In part 2, two cartridges simultaneously were tested, rather than a single cartridge. In part 2, the top probe of the first cartridge had the same placement as above. However, the bottom probe of the first cartridge was placed 10 cm (4 in.) caudally. The top probe from the second cartridge was placed caudally an additional 10 cm (4 in.), and the bottom probe from the second cartridge was placed caudally an additional 10 cm (4 in.) such that the distance from the top probe of cartridge 1 to the bottom probe of cartridge 2 was 30 cm (12 in). See Fig. 1.

Baseline vital signs (blood pressure, heart rate, and pulse oximetry) were taken with a portable monitor (Nonin 2120, Plymouth, MN). Subjects were connected to a spirometer, and continuous respiratory data was collected with a breath-by-breath gas exchange system (Ultima Cardio 2 PFX, Med Graphics, Minneapolis, MN). The spirometer measured oxygen and carbon dioxide concentrations of breathed air, the respiratory rate, and the tidal volume on a breath-by-breath basis. Sparks Systems (Phoenix, AZ) was contracted to collect and process the spirometry data. A wireless acquisition module (Mortara, Milwaukee, WI) was used to integrate continuous ECG before and after the exposure (ECG during exposure was not possible due to electrical noise and body movement). Once baseline data were collected over about 5 min, subjects had a 10-s continuous exposure from Axon T7 CEW.

Vital signs were repeated, and labs were drawn within 1–2 min after the exposure. Labs were drawn again at 1 h, and at 16–24 h. Spirometry data were collected for approximately 5 min after the exposure.

Data were compiled in an Excel spreadsheet (Microsoft Corporation, Redmond, WA) and exported into Stata (Version 15, StataCorp, College Station, TX) for analysis. Median and interquartile range values were calculated for vital signs, laboratory values and respiratory values for each part. Part 1 and 2 were compared with a t-test for each value. Data were further analyzed by comparing change from baseline to point of highest clinical significance.

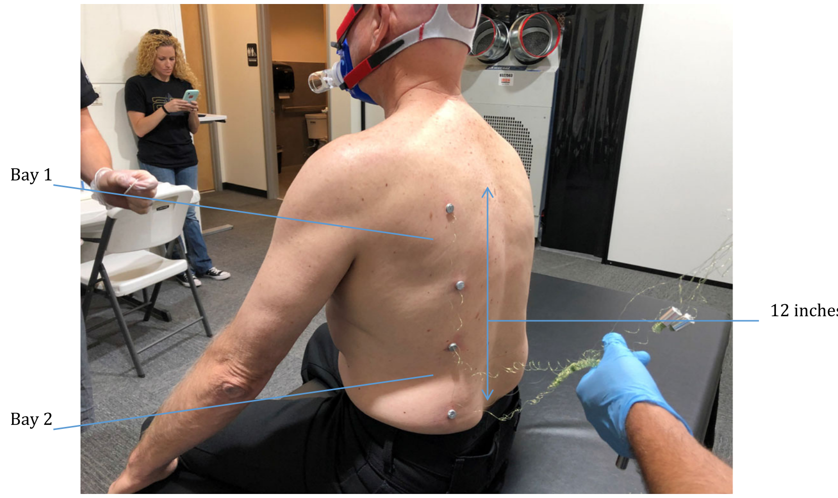

Fig. 1 Set up for part two (simultaneous two-bay exposures)

Results

In part 1, the single-cartridge exposures, 11 subjects were enrolled. No subjects were excluded. All the subjects were male. Subject health histories included: depression (1), chronic pain (1), high blood pressure (1), hernia surgery (1), orthopedic surgery (3), and multiple sclerosis (1). The mean age of the subjects was 38.5, with a range from 22 to 74. The mean height was 191 cm (70.5 in), weight 96.4 kg (212 lbs), and BMI of 29.6 kg/m^2.

In part 2, the simultaneous two-cartridge exposures, 9 subjects were enrolled. No subjects were excluded. 8 subjects were male, and 1 was female. Health histories included: diabetes (1), hypertension (4), GERD (2), abdominal surgeries (1) and orthopedic surgeries (3). The mean age of the subjects was 38, with a range from 19 to 59. The mean height was 178 cm (70 in), weight 98.6 kg (217.6 lbs), an BMI of 30.8 kg/m^2.

There were no important adverse events observed or reported. One subject in part 1 had vasovagal syncope immediately after the exposure (his pulse was checked during the syncope and he was bradycardic) but recovered quickly and reported no persistent symptoms. Another subject hyperventilated after but responded well to reassurance. One subject with a baseline CK of 672 had a CK of 2883 at 24 h. This subject was contacted by email 1 week later and had no complaints. He denied any symptoms or signs of rhabdomyolysis during the week after the exposure. He was on a cholesterol-lowering “supplement”. He was advised to have his CK (and renal function) rechecked. No additional follow up was attempted or requested.

A blinded emergency physician reviewed the ECGs from both parts. There were no changes in any subject ECGs except for small changes in heart rate (≤10–12 beats per minute (BPM)). No subjects had ectopic beats or a rhythm different from sinus rhythm at any point.

Data for CK, potassium, pH, lactate, troponin, and catecholamine levels for both parts is shown in Table 1. Only norepinephrine and epinephrine are given here for the catecholamines as dopamine was reported as “<30” pg/mL for 36/44 measurements in part 1 (the 8 exceptions ranged from 31 to 47 pg/mL) and for 34/36 measurements in part 2 (the 2 exceptions ranged from 36 to 61 pg/mL). While hemoglobin was not a primary outcome, we reported it here because one investigator had raised concerns regarding hemoglobin due to findings in swine, and because it has never been published previously. The comparative blood data is presented in Table 2. Statistical differences between the parts occurred in potassium, lactate, pH, and epinephrine. The clinical significance of these differences is uncertain, though unlikely to be important.

The spirometry data for both parts is presented in Table 3. The data presented is the respiratory rate, tidal volume, end tidal O_2, end tidal CO_2, and minute ventilation for pre-exposure, during exposure, the first minute post-exposure, and the second minute post-exposure. The data did not show a statistical change pre-exposure to exposure for either group (except a small change in $PETCO_2$). However, the minute ventilation increased after the exposure. Two subjects in the 2-cartridge group did not take any breaths during the exposures. The comparison spirometry data is presented in Table 4. Statistical changes occurred in $PETCO_2$ and minute ventilation.

Vital sign data are presented in Table 5 and comparison data are shown in Table 6. There were no statistically significant changes.

Discussion

Our results are consistent with the results of prior studies. Many of the same parameters have been studied by these authors and others across several CEW models including the

Table 1 Blood results

Laboratory Value	1 Cartridge Exposure				2 Cartridge Exposure				*P* Value*
	Median (IQR)				Median (IQR)				Pre - *
	Pre -Exposure	Post Exposure	1-Hour	Next Day	Pre -Exposure	Post Exposure	1-Hour	Next Day	1 Cartridge/2 Cartridge Exposure
K+	3.9	3.7 *	3.9	3.8	3.9	3.8*	3.9	3.9	0.002/0.64
(mmol/L)	(3.7–4.1)	(3.6–3.9)	(3.8–4.2)	(3.7–4.3)	3.6–4.0)	(3.6–4.1)	(3.7–4.3)	(3.8–4.1)	
Lactate	0.98	3.98*	1.12	0.88	1.10	2.10*	1.14	1.10	<0.001/0.005
(mmol/L)	(0.74–1.22)	(2.96–4.27)	(1.05–1.69)	(0.65–1.17)	(0.76–1.32)	(1.87–2.54)	(1.10–1.14)	(1.00–1.35)	
Hgb	15.3	15.3*	15.0	15.6	15.6	15.3*	15.3	15.6	0.34/0.01
(g/d/l)	(15.0–15.6)	(14.3–15.6)	(14.3–15.6)	(15.0–16.3)	(15.0–16.3)	(14.6–16.3)	(14.6–15.6)	(14.3–16.0)	
Troponin	0.00	0.00	0.00	0.00 *	0.00	0.00	0.00	0.00*	–/0.35
(ng/mL)	(0.00–0.00)	(0.00–0.00)	(0.00–0.00)	(0.00–0.00)	(0.00–0.00)	(0.00–0.00)	(0.00–0.00)	(0.00–0.00)	
pH	7.39	7.32*	7.36	7.37	7.36	7.32*	7.34	7.37	<0.001/0.12
	(7.36–7.41)	(7.27–7.33)	(7.35–7.38)	(7.35–7.41)	(7.33–7.36)	(7.30–7.35)	(7.33–7.35)	(7.33–7.38)	
CK	88	88	98	156*	159	152	163	294*	0.03/0.19
(U/L)	(79–143)	(76–141)	(79–134)	(183–371)	(88–198)	(92–199)	(122–202)	(145–489)	
NE	485	744*	547	526	532	682*	509	559	0.01/0.01
(pg/mL)	(390–715)	(516–924)	(453–690)	(359–665)	(362–580)	(565–1001)	(433–616)	(438–595)	
Epi	73	460*	38	27	62	666*	61	37	0.005/<0.00
(pg/mL)	(34–142)	(304–592)	(30–117)	(16–54)	(45–72)	(557–1194)	(45–71)	(26–78)	1

p values in the rightmost column are between the pre-exposure value and the value noted by *

CK creatine kinase, *Epi* epinephrine, *IQR* interquartile range, *NE* norepinephrine

Table 2 Comparative blood results

Comparative Blood Data	Comparison Points	Mean Change 1 Cartridge	Mean Change 2 Cartridges	Mean Difference (2 Cartridges-1 Cartridge)	*p* Value	95% CI
K+(mmol/L)	Pre-Exposure-Post Exposure	−0.15	+0.04	+0.19	0.05	0 to 0.38
Lactate (mmol/L)	Pre-Exposure-Post Exposure	+2.78	+1.39	−1.39	0.01	−2.33 to −0.44
Hgb (g/d/l)	Pre-Exposure-Post Exposure	−0.22	−0.21	+0.01	0.98	−0.51 to 0.53
Troponin (ng/mL)	Pre-Exposure – Next Day	+0.000	+0.001	+0.001	0.28	0 to 0.001
pH	Pre-Exposure – Post-Exposure	−0.07	−0.03	+0.04	0.02	0.01 to 0.08
CK (U/L)	Pre-Exposure – Next Day	+98	+341	+244	0.28	−220 to 707
NE (pg/mL)	Pre-Exposure-Post Exposure	+278	+253	−25	0.83	−265 to 214
Epi (pg/mL)	Pre-Exposure-Post Exposure	+347	+737	389	0.01	91 to 687

Means are measured as (2 cartridges – 1 cartridge)

first-generation Axon X26E, and the second generation X3 and (newer) X2.

Several authors have studied ECG tracings before and after CEW exposure and found no significant morphologic changes or dysrhythmias [2, 3, 5–8]. The majority of these studies involved large probe spread back exposures, although one involved chest exposures (one involved abdomen to leg exposures). Likewise, our study found no important ECG changes associated with the exposures. While not a study objective, continuous ECG recording was done during spirometry except when disconnected for the exposure and no dysrhythmias or ectopy was noted in any subject after exposure. Based on animal studies, it is very unlikely to have direct cardiac effects with exposures that do not include the chest.

Several authors have studied spirometry. In a prior study by Ho et al. of 15-s trunk (front and back) X26E exposures, the study subjects had increased minute ventilation during the exposures (mean 20.9 L/min compared to 16.3 at baseline for the continuous exposures) [9]. Van Meenan et al., in a study of 5-s X26E back exposures, showed a decrease in tidal volumes (described as "cessation" of normal breathing) during exposures. However, only graphical data was presented with no statistical summary. In addition, compared to other studies, these exposures were short [10]. In a study by Dawes et al. of a 10-s X3 exposure to the chest, there was no statistical change in tidal volume, respiratory rate or minute ventilation (although minute ventilation was close to significant) during the exposure with a statistically significant increase in these parameters after the exposure [11]. In a study by Ho et al. of 10-s X2 exposures to the abdomen and leg, there were significant increases in the respiratory rate and minute ventilation but a decrease in the median tidal volume during the exposures [3]. Our data is not dissimilar to the prior X3 data. There were no statistically significant changes in spirometry parameters (except for $PETCO_2$) in both groups. In both groups, minute ventilation rose in the post-exposure period. There are several possible explanations for this. One explanation is that there was some limitation on subject breathing during the exposure to prevent full respiratory compensation for the muscle-contraction induced acidosis and this was "make up" ventilation; or simply a pain response. The significance of these findings is likely low in practice. Most field exposures are less than 15 s. In Bozeman et al. only 7% of field uses included over 3 exposures (not necessarily continuous) [12]. In a study by Ross and Hazlett of 1085 "violent" arrests over 12 months with 17 agencies, the average "trigger pulls" was 2 totaling 10 s (30% one trigger pull, 55% two, and 15% 3) [13]. It would be very unlikely even in physiologically compromised subjects for short exposures (less than 15 s), even if they caused cessation of all breathing, to cause any important effects. Of particular note, the clinically important patient-oriented outcomes of $PETCO_2$ and $PETO_2$ did not show hypercarbia or hypoxia. It is not clear why the 2-cartridge group had an increase in minute ventilation compared to the 1-cartridge group. Given the rapidity of the pulses, it would not be expected that the brief "short path" time and pulse changes would make a clinical difference in respiration but this is possible. It is also not clear why the data is different from the original X26E data (the X2 data did not involve the thorax). Exposures were more lateral in the original study that could mean less bilateral thorax involvement. The difference with the Van Meenan data may be due to the difference in duration as previously hypothesized.

Several authors have also studied serum markers, including troponin. None of our subjects had a positive troponin (the LabCorp reference range is 0.00–0.04). Multiple authors have measured serum troponin after exposures and none have found a troponin elevation except one 24-h reading in Ho et al. That subject had in-hospital monitoring and testing and the result was deemed spurious [2, 3, 7, 8, [14]. Back exposures would not be expected to have direct cardiac effects but indirect effects may be possible. The data here, including the ECG data, does not suggest any indirect effects other than the possibility of vasovagal induced syncope.

Table 3 Spirometry results

Respiratory Value	1 Cartridge Exposure				2 Cartridge Exposure				*P* Value
	Median (IQR)				Median (IQR)				Pre – Exposure to Exposure
	Pre - Exposure	Exposure	1st minute post	2nd Minute post	Pre - Exposure	Exposure	1st minute post	2nd Minute post	1 Cartridge/2 Cartridge Exposure
Respiratory Rate (bpm)	13 (11–19)	15 (11–18)	19 (13–27)	18 (12–24)	1 (13–19)	2 (9–27)	2 (17–23)	17 (16–27)	0.64/0.65
Tidal Volume (L)	0.98 (0.71–1.27)	0.88 (0.78–1.21)	1.65 (1.31–2.05)	1.27 (1.03–2.49)	0.84 (0.62–0.95)	1.43 (0.52–1.50)	1.75 (1.44–2.07)	1.17 (0.99–1.59)	0.32/0.37
PETO2 (mmHg)	104 (101–107)	105 (103–112)	105 (99–112)	109 (107–111)	105 (103–108)	112 (108–113)	105 (103–109)	112 (106–114)	0.10/0.32
PETCO2 (mm Hg)	34 (33–36)	34 (28–35)	34 (32–37)	34 (33–36)	34 (31–36)	30 (27–32)	35 (32–35)	35 (32–36)	0.04/0.04
Minute ventilation (L)	12.8 (11.6–14.7)	11.8 (10.2–14.8)	30.1 (24.5–36.4)	23.0 (21.9–28.5)	12.5 (11.1–14.4)	21.9 (13.7–31.2)	35.3 (29.2–38.2)	25.7 (24.6–27.6)	0.41/0.10

Bpm breaths per minute

Our study did not suggest that the T7 would cause clinically significant changes in CK with 10-s exposures. We would possibly expect some CK elevation due to the motor-nerve mediated muscle contractions caused by the CEW. Axon CEWs can induce normalized contraction forces of 46% [1]. However, none were clinically important, and only the 1-cartridge exposure group (part 1) had a statistically significant change at 24 h, but the median was only 156. One subject, in the two-cartridge exposure group, who had a high baseline CK of 672 (and was on a cholesterol “supplement”), had a CK at 24 h of 2883. He was followed up by email 1 week later and had no clinical symptoms or signs of rhabdomyolysis during the week after the exposure. The lack of clinically important rises in CK is consistent with other studies. In Ho et al. study subjects exposed to a 5-s X26E had a baseline mean CK of 185 that rose to 242 at 24 h; clinically insignificant [2]. Van Meenan et al. also with 5-s X26E exposures, had a mean change from 174 to 181 [7]. In Dawes et al. the baseline CK was 143, rising to 649 at 24 h but this study was confounded by subjects in the study participating in a physical training course concurrently in the AZ heat [11]. In another Ho et al. study, the median change in CK at 24 h was 313; clinically unimportant. This study also suffered from compliance issues with participants who exerted between the initial and final blood draw [3]. In another study by Ho et al. comparing use-of force scenarios, the CK rose to 241 from a baseline of 184 after a 10-s X26E exposure [15]. In this study, only the sprint group had a statistically significant rise in CK; although none of these were clinically important. The comparative analysis did not show a statistical change between the two groups. High CKs found post-exposure in the field are much more likely to be related to the subject’s overall exertion, hyperthermia, and drug effects.

Our study subjects had small changes in pH and lactate, similar to other studies. In a study by Vilke et al. of 5-s exposures from the X26E, the pH dropped from a baseline of 7.45 to 7.42 at 1 min post-exposure. Lactate rose from a baseline of 1.4 to 2.8 at 1 min. In a study by Dawes et al. of 10-s exposures from an X3, the pH dropped from 7.4 at baseline to 7.36 immediately post-exposure and 7.35 at 2 min. The lactate rose from a baseline of 1.32 to 3.05 post-exposure and to 4.52 at 2 min [11]. In a study by Ho et al. of 10-s X26E exposures, the pH dropped from 7.37 at baseline to 7.29 post-exposure and 7.29 at 2 min. The lactate rose from a baseline of 1.30 to 5.49 post-exposure and 5.52 at 2 min. This was in contrast to a drop in pH of 7.36 at baseline to 7.04 and 7.01 post-exposure and at 2 min, respectively, for a 45-s heavy bag exertional activity. In the heavy bag subjects, the lactate rose from a baseline of 1.44 to 15.46 and 17.22 post-exertion and at 2 min, respectively [15]. In a study by Ho et al. a 10-s X2 exposure to the abdomen and thigh resulted in a median drop in pH of −0.031 and a rise in lactate of 1.2 [3]. The comparative analysis showed that the 1-cartidge group had statistically greater changes in pH

Table 4 Comparative spirometry results

Comparative Respiratory Data	Mean Change 1 Cartridge	Mean Change 2 Cartridges	Mean Difference (2 Cartridges-1 Cartridge)	*p* Value	95% CI
Respiratory Rate	+0.5	+2.1	+1.6	0.71	−7.23 to 10.45
Tidal Volume	−0.76	+0.19	+0.26	0.19	−0.14 to 0.65
PETO2	+2.4	−18.3	−20.7	0.20	−53.1 to 11.7
PETCO2	−2.2	−9.6	−7.3	0.06	−15.1 to 0.4
VE	−0.6	+8.8	+9.4	0.04	0.2 to 18.5

Respiratory Comparison points are measured between pre-exposure and exposure values

and lactate. This may have to do with the brief "short path" time and pulse changes due to the cross connect algorithm. However, these changes are all within the norms from prior studies.

Changes to catecholamines in our study are generally consistent with prior studies. In a study by Ho et al. of 5-s TASER X26E exposures, norepinephrine rose from 374 at baseline to 631 post-exposure, and epinephrine rose from 79 to 298 post-exposure [16]. In a Dawes et al. study of 5-s X3 exposures, norepinephrine rose from 295.5 to 546 post-exposure, and epinephrine rose from 55 to 402 post-exposure [11]. In Ho et al. after a 10-s TASER X26E exposure, the change in norepinephrine was similar to our current values; in that same study, the change in norepinephrine, by way of reference, was in the thousands for a 45-s exertion activity (heavy bag) [15]. Here our mean change in norepinephrine was very similar to previous studies. The mean change in epinephrine was higher for the two-cartridge subjects for unclear reasons. However, most subjects reported the T7 to be more painful, likely due to the increased pulse rate.

Our study subjects had no clinically important change in potassium. In a study by Ho et al. study subjects exposed to a X26E for 5-s had a mean baseline potassium of 4.1, dropping to 3.9 post-exposure [2]. Van Meenan et al., also with 5-s X26E exposures, had a mean potassium of 3.9 pre-exposure and 4.0 post-exposure [7]. In a study by Vilke et al. of 5-s X26E exposures, the mean pre-exposure baseline potassium was 4.2, dropping to 4.1 1-min post-exposure [17]. In a study by Dawes et al. of 10-s X3 exposures, the potassium changed from a mean of 3.8 pre-exposure to 4.0 post-exposure [11]. In a study by Ho et al. of 10-s exposures from the X2, the potassium changed from a median of 3.65 at baseline to 3.9 post-exposure [3]. In none of these studies was the change clinically important. Our comparative analysis did show a statistical change between the groups but this is clinically unimportant.

One prominent CEW animal researcher raised a concern about changes in hematocrit after exposures in swine [18]. We previously replied to this in a letter to the editor, however, this data has never been published apart from this letter so we elected to present the data here despite not being one of our planned outcome measures [19]. In this study there was no clinically important change in hemoglobin (or hematocrit).

In our study, vital signs showed no statistical changes. In Vilke et al., there was a statistical but not clinically significant drop in systolic blood pressure pre-exposure to post-exposure (first reading at 5 min) after a 5-s X26E exposure. There were no statistical differences in diastolic blood pressure, pulse rate or oxygen saturation [17]. In Bozeman et al. there was a

Table 5 Vital sign results

Vital Signs	1 Cartridge Exposure		*P* value	2 Cartridge Exposure		*P* value
	Pre-Exposure	Post-Exposure	Pre-Exposure to Post- Exposure	Pre-Exposure	Post-Exposure	Pre-Exposure to Post- Exposure
Heart Rate						
Beats per Minute	93 (72–108)	89 (79–92)	0.79	87 (75–92)	94 (92–104)	0.12
Oxygen Saturation (%)	98 (96–98)	98 (96–98)	1.00	98 (98–99)	98 (97–98)	0.83
Systolic Blood Pressure						
mmHg	159 (144–160)	164 (146–179)	0.12	150 (142–176)	145(143–176)	0.49
Diastolic Blood Pressure						
mmHg	100 (94–106)	100 (83–107)	0.38	86 (84–113)	92 (76–113)	0.50

All data are displayed as mean (IQR)

Table 6 Comparative vital signs results

Comparative Vital Sign Data	Mean Change 1 Cartridge	Mean Change 2 Cartridges	Mean Difference (2 Cartridges-1 Cartridge)	*p* Value	95% CI
Heart Rate Beats per Minute	−1	+8	+9	0.15	−4 to 22
Oxygen Saturation (%)	0	0	0	0.87	−0.01 to 0.01
Systolic Blood Pressure mmHg	+8	+3	−5	0.50	−19 to 10
Diastolic Blood Pressure mmHg	−4	−2	+2	0.66	−9 to 13

means are measured as (2 cartridges – 1 cartridge)

statistical but not clinically important rise in systolic blood pressure 1 min after a 5-s X26E exposure. There was no statistical change in diastolic blood pressure. There was a statistically significant rise in heart rate post-exposure (12 BPM) in these subjects [6]. In a Ho et al. study comparing intermediate use-of-force scenarios, there was no statistical change in blood pressure or heart rate post-exposure after a 10-s X26E exposure. Interestingly, there were statistical increases in blood pressure and heart rate after exposures to the sprint ("fleeing") scenario, the oleoresin capsicum (alternative intermediate force option) scenario (blood pressure only), the heavy bag ("fight") scenario, and the K-9 "bite" scenario [15]. In Dawes et al. and Ho et al., after 10-s exposures to the X3 and X2, respectively, there were no clinically important changes in vital signs [3, 11].

The Axon T7 has several advantages over the previous generation weapons. Like the second-generation X2, the T7 has two cartridges (or probe pairs) giving officers a backup shot in case of a missed probe, poor initial connection, disconnect, or suboptimal incapacitation due to a small spread or the location of the probes. This feature was an improvement over the single cartridge X26E. With the X2, however, the bays/cartridges functioned independently unless one probe was not connected. This is important because if a subject had suboptimal incapacitation due to a small-probe-spread exposure (e.g. close contact exposure), and the back-up shot also resulted in a small-probe-spread exposure (which would be expected if the officer remained close to the subject), there was no bay-to-bay (cartridge-to-cartridge) connection, the probe pairs were electrically isolated, and there would likely continue to be suboptimal incapacitation. The Axon T7 has new technology that enables adaptive cross connection between the two bays. With adaptive cross connection, the weapon rotates connections between all possible positive to negative current paths during an exposure. The weapon senses the return current (detecting bad current paths) and adapts the path between all possible positive to negative current paths to optimize the connection. Because of this, the pulse rate "seen" at a single probe can vary between 11 and 35 PPS. In the case of two suboptimal small probe-spread exposures, because of the rotation of the current paths, there would be current paths between the two bays that would increase the overall "effective" probe spread and improve the incapacitation performance of the CEW. In other words, the "effective" probe spread would be the longest distance between a positive and negative probe. This scenario of small-probe-spread exposures is anecdotally frequently a cause of suboptimal exposures with the X2 and X26E. Many encounters with subjects are at close range. In a study by White and Ready of the NYPD, the mean distance was 5.5 ft, and, in their study, one of the statistically significant predictors of an "ineffective deployment" was a distance of less than 3 ft between the subject and the officer [20]. A 3-ft distance would only give a probe spread of about 5 in.. As previously stated prior work by us has shown that probe spreads of ≥12 in. are generally are necessary for optimal incapacitation [4].

While this study did not study incapacitation performance, or cardiac safety, the physiologic effects of the new T7 appear to be similar to prior generation Axon CEWs.

Limitations

The small number of enrolled subjects limits our conclusions. Additionally, our study only used large-spread (or large spread "equivalent") back shots. Low center of mass exposures on the front (manufacturer recommended targeting) would have mechanistically had less of a respiratory effect given that thoracic muscles would not be stimulated. Because of our small study enrollment, we decided to not study these exposures. However, this may bias our study to more respiratory effects. Lastly, we did not test a probe scenario that would yield the highest PPS rate at a probe with the adaptive cross connect algorithm. We instead chose, in order to better compare both groups, exposures that would be relatively equivalent if the adaptive cross connect technology was working (two 30 cm (12-in) equivalent exposures).

Conclusions

In our study, the physiologic effects of the new generation Axon T7 are similar to prior generation weapons.

Key points

1) CEWs cause modest changes in physiology consistent with the electrically-induced motor-nerve mediated muscle contraction.
2) The T7 appears to have a similar physiologic profile to prior CEWs.

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