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REVIEW

Adrenergic and metabolic effects of electrical weapons: review and meta-analysis of human data

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Abstract

Introduction Electronic control with the CEW (conducted electrical weapon) has gained widespread acceptance as the preferred force option due to its significant injury reduction. However, a CEW application does stress the human body. In the case of the CEW, the human body response is similar to the challenge of physical exercise combined with emotional stress over a very short time interval. There has been concern whether the tension of the skeletal-muscle system together with the emotional stress of being exposed to the effects of a CEW, can lead to severe metabolic dysfunction.

Methods A systematic and careful search of the MedLine database was performed to find publications describing pathophysiological effects of CEWs. Additional publications were collected through a manual search of reference lists in retrieved articles. After preliminary exclusions, we carefully reviewed the remaining publications and found 24 papers reporting prospective human clinical research data on adrenergic, ventilation, or metabolic effects. Where there were multiple studies on the same endpoints, we performed meta-analyses.

Results A CEW exposure provides a clinically insignificant increase in heart rate (7.5 BPM) and a drop in both systolic and diastolic blood pressure. Alpha-amylase goes down but cortisol levels increase—both epinephrine and norepinephrine levels are increased by levels similar to mild exercise. A CEW exposure increases ventilation but does not appear to interfere with gas exchange. Lactate is increased slightly while the pH is decreased slightly with changes equivalent to mild exercise. The lactate and pH changes appear quickly and do not appear to be affected by increasing the exposure duration from 5 to 30 s.

Conclusions Thorough review and meta-analyses show that electrical weapon exposures have mixed and mild adrenergic effects. Ventilation is increased and there are metabolic changes similar to mild exercise.

Keywords Forensic medicine · Conducted electrical weapon · Metabolic effects · Catecholamines · Biomarkers

Introduction

Electronic control with the CEW (conducted electrical weapon) has gained widespread acceptance as the preferred force option due to its significant injury reduction. Large prospective studies have found suspect injury rate reductions of about 65% [1]. Of the 310,000 annual CEW field uses, only 1 in 3500 is involved in an ARD (arrest-related death) vs. the baseline ARD rate of 1:1000. This reduction in fatality rate is consistent with prospective published data, which showed that 5.4% of CEW uses "clearly prevented the use of lethal force by police." [2] It is also consistent with a 2/3 reduction in fatal police shootings where CEW usage is not overly restricted [3].

However, a CEW application does stress the human body. In the case of the CEW, the human body response is similar to the challenge of physical exercise combined with emotional stress over a very short time interval. The rapid increase in energy demands due to isometric muscle activity throughout the body activates the sympathetic nervous system, which stimulates glycogenolysis within the

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muscles. Simultaneously, the adrenocortical axis responds with rises in catecholamines (epinephrine, norepinephrine, and dopamine). In the context of the CEW, there has been concern whether the tension of the skeletal-muscle system together with the emotional stress of being exposed to the effects of a CEW, can lead to severe metabolic dysfunction.

The metabolic effects of different kinds of stress, physical activity, or trauma involve many interacting endocrinological, physiological, biochemical and central nervous system components. There has been no systematic review of the literature covering these possible adverse events from these weapons. A systematic and careful search of the MedLine database was performed to find publications describing pathophysiological effects of CEWs. Additional publications were collected through a manual search of reference lists in retrieved articles. Search terms included "Conducted Electrical Weapon," "CEW," "Electronic Control Device," "TASER," "Electroshock Weapon," "Electrical Weapon," and "Stun Gun." We found 525 matches and then removed publications that were unsuitable based on title and abstract.

We excluded papers on transanal submucosal endoscopic resection (TASER), the "TaSER" study (Targeting ultrasound remission in early rheumatoid arthritis), and "TASER-M" study of HIV infections. We removed 178 non-experimental articles which were mostly opinion articles (68%) as well as case studies (24%). Other articles excluded were retrospective case series of possible electrocutions, CEW-ignited fires, fall injuries, and experiments on obsolete older higher-power CEWs. We eliminated 30 papers reporting the results of computer simulations and 35 papers reporting the results of animal studies. Another 141 articles were removed for having no data or discussion on adrenergic, ventilation, or metabolic effects. We carefully reviewed the remaining publications and found 24 papers reporting prospective human clinical research data on adrenergic, ventilation, or metabolic effects.

Catecholamines

In the majority of cases, the pain component of electronic control is reduced due to drug or alcohol intoxication. A large study found that 71% of electronic control subjects had recreational drugs in their urine [4]. The reaction to pain in humans is complex. Pain does not always cause increases in blood pressure, heart rate, and catecholamine levels [5]. In fact, pain may lower blood pressure and heart rate. Studies of various types of pain in humans have demonstrated relatively few (and mostly transient) effects even in patients with severe hypertension [6]. One study even suggested that heart rate and blood pressure should not be used as reliable indicators of pain [7]. In addition, all of these studies measured the effects of painful stimulation over minutes, not seconds, as in electrical-weapon discharges.

In this section, we review all published peer-reviewed studies on various indicators of catecholamine effects from CEW usage.

Heart rate

We found 11 papers covering 352 subjects where heart rate was measured before and after exposure. The weighted average exposure time was 7.9 s and the heart rate increased an average of 7.5 BPM (see Table 1). Some of the papers provided standard deviations while others provided confidence limits or overall ranges. Thus, it was not possible to calculate a pooled standard deviation or confidence limits. There was no trend with respect to exposure times.

Blood pressure

We found seven papers covering 173 subjects where blood pressure was measured before and after exposure. The weighted average exposure time was 8.1 s. The systolic blood pressure decreased an average of 3.6 mmHg. The diastolic blood

Table 1 Heart rate effects

	Year	*n*	Exposure (s)	HR1	HR2	Delta (BPM)
Ho [8]	2007	34	15	104.7	116.3	11.6
Levine [9]	2007	105	5	122	137	15
Ho [10]	2008	34	10	108.7	94.1	− 14.6
Vilke [11]	2008	32	2.1	67.2	69.7	2.5
Bozeman [12]	2009	28	5	119.4	131.7	12.3
Dawes [13]	2010	9	5	91.7	86.8	− 4.9
Dawes [13]	2010	11	30	89.5	92.5	3
Ho [14]	2010	12	10	92	96	4
Ho [15]	2011	53	10	84.5	93	8.5
VanMeenen [16]	2013	23	5	110	121	11
Dawes [17]	2014	13	5	79	80	1

Table 2 Blood pressure effects

	Year	n	Exposure (s)	SBP1	SBP2	Delta	DBP1	DBP2	Delta
Vilke [18]	2007	32	5	139	128	− 11	84	83	− 1
Bozeman [12]	2009	28	5	138.6	145.8	7.2	82.8	85.6	2.8
Vilke [19]	2009	25	5	139	128	− 11	86	78	− 8
Dawes [20]	2010	11	30	141.3	142.9	1.6	81.8	76	− 5.8
Ho [14]	2010	12	5	139	141	2	88	84	− 4
Ho [15]	2011	53	10	149	147	− 2	86	83	− 3
Ho [21]	2014	12	5	127	128	− 11	82	74	− 8

All pressures in mmHg

pressure decreased an average of 3.0 mmHg (see Table 2). Some of the papers provided standard deviations while others provided confidence limits or overall ranges so we did not calculate a pooled standard deviation or confidence limits. As with heart rate, there was no trend with respect to exposure times.

Biomarkers

Dawes et al. measured alpha-amylase and cortisol levels before and after a CEW exposure [22]. These were also measured along with other analogs of law-enforcement control techniques. Subjects were randomized to one of the four interventions studied. Subjects received either a 5-s exposure from a TASER X26 CEW with the probes fired into the back from 2 m, a 5-s spray of pepper spray (a skin and mucous membrane irritant) to the eyes, a 45-s exposure of the hand and forearm in a 0 °C cold-water tank, or a 60-s defensive tactics (fighting) drill. Blood samples were taken at 10–15 min and also at 40–60 min.

Alpha-amylase had the greatest increase from baseline at 10–15 min (1st blood draw) with the defensive tactics drill. Cortisol had the greatest increase at 15–20 min with pepper spray at 0.50 μg/dL. Cortisol remained most elevated at 40–60 min in the defensive tactics drill group at 0.47 μg/dL. Alpha-amylase levels went down in the CEW group at both the first and second blood draws. In the CEW group, cortisol levels increased by 0.38 and 0.32 μg/dL at the first and second blood draws respectively.

Ho et al. and Dawes et al. measured epinephrine and norepinephrine levels before and after 10-s CEW exposures in two studies in 2010 [14, 23]. The norepinephrine values from the 12-subject 2010 Ho study were about 10 times higher than the other studies. After correspondence with the authors we felt that there had been an instrumentation scaling error so they were excluded as outliers. Ho published catecholamine values with a 5-s exposure in 2013 [24] (see Table 3 for summary).

The Ho paper compared the catecholamine increases between the CEW, pepper spray, simulated dog threat, heavy bag punching, and a 150-m sprint. The bag punching had the greatest increases in both epinephrine and norepinephrine immediately after and also at 2 and 4 min.

Summary

A CEW exposure provides a clinically insignificant increase in heart rate (7.5 BPM) and a drop in both systolic and diastolic blood pressure. Alpha-amylase goes down but cortisol levels increase—yet less so than with pepper spray or fighting. Both epinephrine and norepinephrine levels are increased but less so than with the physical exertion of punching a heavy bag and comparably to other police tools.

A challenge with these adrenergic studies is the anticipation effect on the baseline values. Most people fear electricity and this can increase baseline autonomic tone. Observing or hearing other volunteers experiencing a CEW exposure could raise the baseline values which could possibly lead to misleading post-exposure drops. For example, the weighted average systolic blood pressure was 141.3 mmHg before exposure which seems high for police officers. Vilke reported baseline minute ventilation of 15.5 ± 3.6 L/min in CEW volunteers just before their CEW exposure. When they came back the next day to be "controls" (without a CEW exposure) their minute ventilation was 13.9 ± 4.1 L/min [19]. However, this was not statistically significant with a t test ($n = 25$, $p = .15$), so it is hard to say if the pre-exposure anticipation biased the ventilation effects.

Table 3 Epinephrine and norepinephrine increase (pg/mL)

	Year	n	Immediate	2 min	4 min
Ho [14]	2010	12	270	150	80
Dawes [23]	2010	53	335	64	24
Ho [24]	2013	8	219	86	23
Epinephrine average			323	312	81
Dawes [23]	2010	53	214	94	65
Ho [24]	2013	8	257	147	51
Norepinephrine average			184	84	53

Table 4 Tidal volume before, during, and after CEW exposure

	Year	*n*	Exposure (s)	Tidal volume (L) before	Tidal volume (L) during	Delta	Tidal volume (L) after	Delta
Ho [8]	2007	34	15	1.1	1.8	0.7	1.7	0.6
Ho [8]	2007	18	3 × 5	1.4	1.5	0.1	1.9	0.5
Dawes [20]	2010	11	30	0.93	0.72	− 0.21	1.47	0.54

A CEW exposure has mixed autonomic effects which appear to be lower than those with many law-enforcement control techniques and tools.

Ventilation

Because of the desired effect of skeletal-muscle control, there has been concern that a CEW application could interfere with breathing by incapacitating the intercostal, oblique, and abdominal muscles. Indeed, swine studies have reported CEW interference with breathing [25, 26]. Those studies used anesthetized swine and turned the ventilator off during the CEW discharge and thus their relevance to the clinical situation is limited.

A CEW (drive-stun) application directly over the human phrenic nerves found no effect [27]. It has been suggested that any interference with thoracic contribution could be offset by an increasing diaphragmatic contribution and ventilation rate. We found five human studies of ventilation effect using instrumented volunteers. Of these, three involved only a 5-s exposure which we felt was insufficient to provide a true picture of the breathing cycle [16, 18, 19]. The VanMeenen study reported a decrease of tidal volume during the exposure but only provided a graph with no statistical summary [16]. Their graph appears to show a significant drop of inspiration with a mild drop of expiration during the exposure.

The Vilke resting study did not report ventilation values during the exposure but did show an increase of minute ventilation 60 s after the exposure [18]. The second Vilke study mixed in exercise, and thus, its results were not useful for our analysis since there was no measurement between the exercise and the CEW exposure [19].

This left the Ho and Dawes studies which used 15- and 30-s exposures [8, 20]. Ho's subjects either receive the 15-s exposure continuously or with three blocks of 5 s each and those group results are reported separately in the tables.

While Dawes found a decrease in tidal volume during the exposure, the weighted average effect across all 63 subjects was an increase of 0.37 L in volume (see Table 4). The tidal volume after the exposure was increased by 0.56 L from baseline.

The breathing rate increased during and after the exposure and thus the minute ventilation was increased even more by a weighted average of 3.9 and 12.7 L per minute respectively as seen in Table 5.

The results with gas exchange are mixed. With a 5-s exposure, Vilke reported an increase of 1.0 mmHg in end-tidal CO_2 ($n = 32$) while Ho used a 15-s exposure and found an increase of 1.6 mmHg in 52 subjects and a decrease of 4.7 mmHg in end-tidal O_2 [8, 18]. Dawes found increases of 5.6 and 6.0 mmHg in pCO_2 and pO_2 respectively in 53 subjects with a 10-s exposure [23]. In his subjects, Vilke found increases of 0.01 and 2.1 mmHg in pCO_2 and pO_2 respectively in 32 subjects with a 5-s exposure.

A CEW exposure does not appear to interfere with breathing in humans.

Metabolic effects

The research group of Ho and Dawes along with Vilke et al. performed several metabolic studies on human volunteers with a CEW exposure. They examined the effects of the CEW after short, long-term, and multiple exposures with and without physical challenge and intoxication. Prolonged use of the CEW on already exhausted humans was also evaluated. Most analyses have shown a statistically significant lowered pH and elevated lactate-levels with a CEW exposure as shown in Table 6 and Table 7. However, no clinically significant metabolic changes could be identified.

Table 5 Minute ventilation before, during, and after CEW exposure

	Year	*n*	Exposure (s)	Minute ventilation (L/min) before	Minute ventilation (L/min) during	Delta	Minute ventilation (L/min) after	Delta
Ho [8]	2007	34	15	16.3	20.9	4.6	29.9	13.6
Ho [8]	2007	18	3 × 5	17.7	19.5	1.8	30.1	12.4
Dawes [20]	2010	11	30	15.1	20	4.9	25.7	10.6

Table 6 Change in pH immediately after exposure to CEW

	Year	*n*	Exposure* [s]	pH before*	pH after*	Delta	Range of pH level after
Dawes [23]	2010	53	10	7.40	7.36	− 0.04	7.24–7.46
Dawes [20]	2010	12	30	7.35	7.27	− 0.08	7.19–7.39
Dawes [28]	2010	16	5 (2–3 times)	7.35	7.33	− 0.02	7.27–7.38
Ho [14]	2010	12	10	7.37	7.29	− 0.08	7.24–7.35
Ho [24]	2013	8	5	7.37	7.37	0.0	7.35–7.39
Ho [29]	2009	38	15	7.38	7.23	− 0.15	6.99–7.35
Ho [30]	2009	10	15	7.37	7.35	− 0.02	7.30–7.39
Moscati [31]	2010	22	15	7.40	7.37	− 0.03	7.35–7.38
Vilke [18]	2007	32	5	7.45	7.42	− 0.02	****
Ho [30]	2009	10	15**	7.19	7.12	− 0.07***	7.01–7.23***
Vilke [19]	2009	25	5**	7.41	7.32	− 0.09***	****

*Values are medians except for the Vilke values which are means

**After exertion

***Includes exertion

****No range given

A total of 203 subjects had CEW exposures at rest and the weighted average pH decrease was 0.059 and there was no trend with exposure times from 5 to 30 s ($r^2 = .14$, $p = .31$). With 269 subjects, the weighted average lactate increase was 1.31 mmol/L and there was no trend with exposure time ($r^2 = .21$, $p = .18$).

This meta-analysis excludes the 10 Ho subjects and the 25 Vilke subjects with mixed exercise and CEW exposure [19, 29]. Ho's subjects did push-ups for 30 s and then ran on a treadmill to exhaustion before the CEW exposure. Vilke et al. carried out similar tests in which subjects had to complete an incremental cycling protocol to near-maximum effort, before being exposed to a 5-s CEW application. Comparable to the studies by Ho and Dawes, a rise in lactate could be seen, but no significant changes in acid-base status were detectable.

VanMeenen reported a mean increase of 1.1 mEq/L in bicarbonate with 118 subjects while Ho and Vilke reported decreases of 0.6 and 1.2 mEq/L respectively [18, 32, 33].

Table 7 Change in lactate immediately after exposure to CEW

	Year	*n*	Exposure time* [s]	Lactate before* [mmol/l]	Lactate after* [mmol/l]	Delta* [mmol/l]	Range of lactate level after [mmol/l]
Dawes [23]	2010	53	10	1.32	3.05	+ 1.73	1.31–6.81
Dawes [20]	2010	12	30	1.46	5.63	+ 4.17	1.47–17.29
Dawes [28]	2010	16	5 (2–3 times)	1.05	3.49	+ 2.44	1.41–4.49
Ho [14]	2010	12	10	1.30	5.49	+ 4.19	1.33–7.18
Ho [24]	2013	8	5	1.20	1.70	+ 0.50	1.00–2.40
Ho [29]	2009	38	15	1.65	8.39	+ 6.74	2.10–16.50
Ho [30]	2009	10	15	1.60	2.10	+ 0.50	1.80–3.30
Ho [32]	2006	66	5	1.75	2.74	+ 0.99	0.99–4.99
Moscati [31]	2010	22	15	0.95	1.98	+ 1.03	1.47–2.50
Vilke [18]	2007	32	5	1.4	2.8	+ 1.4	****
Ho [30]	2009	10	15**	9.10	11.60	+ 2.5***	7.80–15.00***
Vilke [19]	2009	22	5**	1.70	8.20	+ 6.50***	****

*Values are medians except for the Vilke values, which are means

**After exertion

***Includes exertion

****No range given

There were four reports covering a total of 224 subjects—all with 5-s exposures—with a weighted average of an increase of 0.24 mEq/L. Dawes tested 53 subjects with 10-s exposures and found an increase of 0.3 mEq/L [23]. Combining the 5- and 10-s exposure studies, we have 277 subjects with a weighted average increase of 0.25 mEq/L.

With the same subjects and exposures, VanMeenen reported a mean increase of 0.5 mg/dL in glucose while Ho reported a decrease of 0.6 mg/dL [32, 33]. Dawes found an increase in glucose of 3.5 mg/dL with his 53 subjects (10-s exposures). The weighted average change ($n = 237$) was 2.15 mg/dL.

Especially compared to the commonly employed uses of force, the metabolic effects of the CEW appear to be smaller and without any clinical consequences [14, 24]. An 18-m sprint generated greater pH and lactate changes than a 5-s CEW exposure [24].

Limitation

This meta-analysis of human research is limited by the listed journals in the search engine PubMed.

This paper is a presentation of the general assessment of the risk using CEW. In order to be able to estimate a possible individual risk, every aspect and variable of each specific case needs to be gathered, put into perspective, and analyzed, a task which cannot be answered by a review publication like this one. Furthermore, the assessment of CEW use in real-life police encounters is defined by the confined transferability of experimental research data.

Conclusions

Within study-design limitations, a CEW exposure appears to cause a mild increase in heart rate and a mild decrease in blood pressure. Catecholamines are increased similarly to mild exercise. Ventilation is enhanced and gas exchange is not decreased. Lactate is increased slightly while the pH is decreased slightly with changes equivalent to mild exercise. The lactate and pH changes appear quickly and do not appear to be affected by increasing the exposure duration from 5 to 30 s.

Compliance with ethical standards

Conflict of interest This paper is a result of literature research, which was not funded. Kunz SN, Calkins H, and Kroll MW are members of the scientific medical advisory board of Axon Int. (fka TASER). Kroll MW also is on Axon corporate board. Calkins H and Kroll MK have been expert witnesses in law-enforcement litigation and Calkins H has been an expert witness in cases of arrest-related death involving CEWs. Adamec J has no conflict of interest to declare.

References

1. MacDonald JM, Kaminski RJ, Smith MR (2009) The effect of less-lethal weapons on injuries in police use-of-force events. Am J Public Health 99(12):2268–2274. https://doi.org/10.2105/AJPH.2009.159616
2. Eastman AL, Metzger JC, Pepe PE, Benitez FL, Decker J, Rinnert KJ, Field CA, FRIESE RS (2008) Conductive electrical devices: a prospective, population-based study of the medical safety of law enforcement use. J Trauma 64(6):1567–1572. https://doi.org/10.1097/TA.0b013e31817113b9
3. Ferdik FV, Kaminski RJ, Cooney MD, Sevigny EL (2014) The influence of agency policies on conducted energy device use and police use of lethal force. Police Quarterly 17(4):328–358. https://doi.org/10.1177/1098611114548098
4. Strote J, Walsh M, Angelidis M, Basta A, Hutson HR (2010) Conducted electrical weapon use by law enforcement: an evaluation of safety and injury. J Trauma 68:1239–1246
5. Burton AR, Fazalbhoy A, Macefield VG (2016) Sympathetic responses to noxious stimulation of muscle and skin. Front Neurol 7:109
6. Finsterer J (2004) Effect of needle-EMG on blood-pressure and heart-rate. J Electromyogr Kinesiol 14(2):283–286. https://doi.org/10.1016/j.jelekin.2003.08.002
7. Lindgren K, Miner J, McGill J (2003) Correlation of heart rate and systolic blood pressure with reported pain. Paper presented at: ANNALS OF EMERGENCY MEDICINE 2003
8. Ho JD, Dawes DM, Bultman LL, Thacker JL, Skinner LD, Bahr JM, Johnson MA, Miner JR (2007) Respiratory effect of prolonged electrical weapon application on human volunteers. Acad Emerg Med 14(3):197–201. https://doi.org/10.1111/j.1553-2712.2007.tb01772.x
9. Levine SD, Sloane CM, Chan TC, Dunford JV, Vilke GM (2007) Cardiac monitoring of human subjects exposed to the TASER. J Emerg Med 33(2):113–117. https://doi.org/10.1016/j.jemermed.2007.02.018
10. Ho JD, Dawes DM, Reardon RF, Lapine AL, Dolan BJ, Lundin EJ, Miner JR (2008) Echocardiographic evaluation of a TASER-X26 application in the ideal human cardiac axis. Acad Emerg Med 15(9):838–844. https://doi.org/10.1111/j.1553-2712.2008.00201.x
11. Vilke GM, Sloane C, Levine S, Neuman T, Castillo E, Chan TC (2008) Twelve-lead electrocardiogram monitoring of subjects before and after voluntary exposure to the Taser X26. Am J Emerg Med 26(1):1–4. https://doi.org/10.1016/j.ajem.2007.01.005
12. Bozeman WP, Barnes DG Jr, Winslow JE 3rd, Johnson JC 3rd, Phillips CH, Alson R (2009) Immediate cardiovascular effects of the Taser X26 conducted electrical weapon. Emerg Med J 26(8):567–570. https://doi.org/10.1136/emj.2008.063560
13. Dawes DM, Ho JD, Reardon RF, Miner JR (2010) Echocardiographic evaluation of TASER X26 probe deployment into the chests of human volunteers. Am J Emerg Med 28(1):49–55. https://doi.org/10.1016/j.ajem.2008.09.033
14. Ho JD, Dawes DM, Nelson RS, Lundin EJ, Ryan FJ, Overton KG, Zeiders AJ, Miner JR (2010) Acidosis and catecholamine evaluation following simulated law enforcement "use of force" encounters. Acad Emerg Med 17(7):e60–e68. https://doi.org/10.1111/j.1553-2712.2010.00813.x
15. Ho JD, Dawes DM, Reardon RF, Strote SR, Kunz SN, Nelson RS, Lundin EJ, Orzco BS, Miner JR (2011) Human cardiovascular effects of a new generation conducted electrical weapon. Forensic Sci Int 204(1-3):50–57. https://doi.org/10.1016/j.forsciint.2010.05.003
16. Vanmeenen KM, Lavietes MH, Cherniack NS, Bergen MT, Teichman R, Servatius RJ (2013) Respiratory and cardiovascular response during electronic control device exposure in law

enforcement trainees. Front Physiol 4:78. https://doi.org/10.3389/fphys.2013.00078

17. Dawes DM, Ho JD, Vincent AS, Nystrom PC, Moore JC, Steinberg LW, Tilton AM, Brave MA, Berris MS, Miner JR (2014) The neurocognitive effects of simulated use-of-force scenarios. Forensic Scio Med Pathol 10(1):9–17. https://doi.org/10.1007/s12024-013-9510-y
18. Vilke GM, Sloane CM, Bouton KD, Kolkhorst FW, Levine SD, Neuman TS, Castillo EM, Chan TC (2007) Physiological effects of a conducted electrical weapon on human subjects. Ann Emerg Med 50(5):569–575. https://doi.org/10.1016/j.annemergmed.2007.05.004
19. Vilke GM, Sloane CM, Suffecool A, Kolkhorst FW, Neuman TS, Castillo EM, Chan TC (2009) Physiologic effects of the TASER after exercise. Acad Emerg Med 16(8):704–710. https://doi.org/10.1111/j.1553-2712.2009.00458.x
20. Dawes DM, Ho JD, Reardon RF, Miner JR (2010) The cardiovascular, respiratory, and metabolic effects of a long duration electronic control device exposure in human volunteers. Forensic Sci Med Pathol 6(4):268–274. https://doi.org/10.1007/s12024-010-9166-9
21. Ho J, Dawes D, Miner J, Moore J, Nystrom P (2014) Neurocognitive effect of simulated resistance and use of force encounters on standardized field sobriety testing. J Emerg Med 46:283
22. Dawes D, Ho J, Miner J (2009) The neuroendocrine effects of the TASER X26: a brief report. Forensic Sci Int 183(1-3):14–19. https://doi.org/10.1016/j.forsciint.2008.09.015
23. Dawes DM, Ho JD, Reardon RF, Strote SR, Nelson RS, Lundin EJ, Orozco BS, Kunz SN, Miner JR (2010) The respiratory, metabolic, and neuroendocrine effects of a new generation electronic control device. Forensic Sci Int 207:55–60
24. Ho JD, Dawes DM, Nystrom PC, Collins DP, Nelson RS, Moore JC, Miner JR (2013) Markers of acidosis and stress in a sprint versus a conducted electrical weapon. Forensic Sci Int 233(1-3):84–89. https://doi.org/10.1016/j.forsciint.2013.08.022
25. Jauchem JR, Sherry CJ, Fines DA, Cook MC (2006) Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of Sus scrofa following repeated TASER exposures. Forensic Sci Int 161(1):20–30. https://doi.org/10.1016/j.forsciint.2005.10.014
26. Dennis AJ, Valentino DJ, Walter RJ, Nagy KK, Winners J, Bokhari F, Wiley DE, Joseph KT, Roberts RR (2007) Acute effects of TASER X26 discharges in a swine model. J Trauma 63(3):581–590. https://doi.org/10.1097/TA.0b013e3180683c16
27. Ho J, Lapine A, Joing S, Reardon R, Dawes D (2008) Confirmation of respiration during trapezial conducted electrical weapon application. Acad Emerg Med 15:398
28. Dawes DM, Ho JD, Reardon RF, Sweeney JD, Miner JR (2010) The physiologic effects of multiple simultaneous electronic control device discharges. West J Emerg Med 11(1):49–56
29. Ho JD, Dawes DM, Bultman LL, Moscati RM, Janchar TA, Miner JR (2009) Prolonged TASER use on exhausted humans does not worsen markers of acidosis. Am J Emerg Med 27(4):413–418. https://doi.org/10.1016/j.ajem.2008.03.017
30. Ho JD, Dawes DM, Cole JB, Hottinger JC, Overton KG, Miner JR (2009) Lactate and pH evaluation in exhausted humans with prolonged TASER X26 exposure or continued exertion. Forensic Sci Int 190(1-3):80–86. https://doi.org/10.1016/j.forsciint.2009.05.016
31. Moscati R, Ho JD, Dawes DM, Miner JR (2010) Physiologic effects of prolonged conducted electrical weapon discharge in ethanol-intoxicated adults. Am J Emerg Med 28(5):582–587. https://doi.org/10.1016/j.ajem.2009.02.010
32. Ho JD, Miner JR, Lakireddy DR, Bultman LL, Heegaard WG (2006) Cardiovascular and physiologic effects of conducted electrical weapon discharge in resting adults. Acad Emerg Med 13(6):589–595. https://doi.org/10.1111/j.1553-2712.2006.tb01016.x
33. VanMeenen KM, Cherniack NS, Bergen MT, Gleason LA, Teichman R, Servatius RJ (2010) Cardiovascular evaluation of electronic control device exposure in law enforcement trainees: a multisite study. J Occup Environ Med 52(2):197–201. https://doi.org/10.1097/JOM.0b013e3181cc58ba