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REVIEW

Cardiac and skeletal muscle effects of electrical weapons

A review of human and animal studies

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Abstract
Conducted Electrical Weapons (CEWs) are being used as the preferred non-lethal force option for police and special forces worldwide. This new technology challenges an exposed opponent similarly to the way they would be challenged by physical exercise combined with emotional stress. While adrenergic and metabolic effects have been meta-analyzed and reviewed, there has been no systematic review of the effects of CEWs on skeletal and cardiac muscle. A systematic and careful search of the MedLine database was performed to find publications describing pathophysiological cardiac and skeletal muscle effects of CEWs. For skeletal muscle effects, we analyzed all publications providing changes in creatine kinase, myoglobin and potassium. For cardiac effects, we analyzed reported troponin changes and arrhythmias related to short dart-to-heart-distances. Conducted electrical weapons satisfy all relevant electrical safety standards and there are, to date, no proven electrocution incidents caused by CEWs. A potential cardiovascular risk has been recognized by some of the experimental animal data. The effects on the heart appear to be limited to instances when there is a short dart-to-heart-distance. The effect on the skeletal muscle system appears to be negligible. A responsible use of a CEW on a healthy adult, within the guidelines proposed by the manufacturer, does not imply a significant health risk for that healthy adult.

Keywords Forensic medicine · Conducted electrical weapon · TASER · Ventricular fibrillation · Electrocution

Introduction

Conducted Electrical Weapons (CEWs) are now being used as the preferred force option for police and special forces worldwide. This new technology challenges an exposed opponent similarly to the challenge they would experience if they were undertaking physical exercise combined with emotional stress. There has been concern whether the application of CEW-waveforms could cause cardiac or skeletal muscle problems. In order to answer this question, a systematic and careful search of the MedLine database was performed to find publications describing pathophysiological cardiac and skeletal muscle effects of CEWs. Additional publications were collected through a manual search of the reference lists in retrieved articles. Search terms included "Conducted Electrical Weapon", "CEW", "TASER", "Electroshock Weapon", "Electrical Weapon" and "Stun Gun". Papers published in languages other than English were excluded. We then removed publications that were unsuitable (i.e. unrelated) based on their title and abstract. Out of the remaining papers we chose experimental (computer, animal and human), overview articles, as well as case studies.

Where there was sufficient human data – such as with troponin or potassium, we focused on the human papers. Further review of the relevance of the articles to the topic at hand resulted in the inclusion of 78 articles. These included 3 papers on the issue of swine arrhythmia sensitivity and 3 on electrical safety standards. The earliest work was published by

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Koscove in 1985, in which he reviews fatality rates with firearms and CEWs [1]. The latest article was by Stopyra reporting on a clinical study with pacemaker and implantable defibrillator patients [2]. The rate of publications in this field is decreasing slightly, with a yearly average of 12 experimental and 21 non-experimental studies published over the last 10 years.

Cardiac effects of CEW

The primary cardiac effect of concern is the induction of VF (ventricular fibrillation) that can ultimately lead to death (electrocution).

Electrical safety standards

Current models of CEWs (X2 and X26P) deliver a safe level of electrical power as specified by the Underwriters Laboratory (UL) and International Electrotechnical Commission (IEC) electric fence standards [3–5]. The X26E CEW delivers ~ 1.8 watts which satisfies the 2.5-watt IEC electrical fence safety limit as well as the 5-watt UL limit for 100 μs pulses [3–5]. Newer CEW models deliver similar power levels. The earlier M26 model (no longer sold) delivered ~10 watts and thus did not satisfy the IEC electric fence standards, but did satisfy the higher UL limit for the 10 μs pulses. Note that the popular X26 is now referred to as the X26E to differentiate it from the newer "X26P". The M26 used high-frequency short pulses, which were relatively ineffective at pacing the heart [6].

Modern CEWs deliver an electrical current at around 1.8 mA pulsed DC and thus satisfy the ANSI CLPSO 17 limit of 2.2 mA of aggregate current. The 1.8 mA is equivalent to 13 mA of AC. This level (13 mA) is substantially lower than the 40 mA IEC safety limit used with residual current limiters and thus satisfy IEC 479–1. Present CEWs thus satisfy all relevant electrical safety standards [7].

The application of electrical safety standards to CEWs is partially limited as these standards assume external skin contact. The heart is closest to the skin in the 4th or 5th left parasternal intercostal spaces with a depth as small as 18 mm in a thin male [8]. The minimum recorded depth for females is 12 mm. The apex can also be this close to the skin but since the myocardium is much thicker there, and hence has a higher VF threshold, this is less relevant to a discussion regarding possible electrocution [9, 10]. Therefore, the electrical safety standards implicitly set an upper bound of ~10 mm for the DTH (dart to heart) distance for VF induction, since 10 mm is less than the skin depth of the right ventricle.

Accordingly, a probe, penetrating the skin directly over the ventricles, could theoretically induce VF, even when the device satisfies these safety standards, if the probe was nearly touching the ventricular epicardium. Horowitz [9, 11] found that the induction of VF in humans by right-ventricular epicardial bursts required pulse charges of 97 μC (= 24.3 mA • 4 ms) which suggests that the 100 μC TASER X26 ECD charge would have to be delivered almost directly to the epicardium. For this reason, the relevant VF testing focuses on the critical DTH (dart to heart) distance for the tip of the probe. Since these studies are always done in swine, we must review these swine studies.

Relevance of swine results to humans

Studies on animals (and in the context of CEW studies with pigs) are very limited in their validity and transferability onto humans. Swine have a different anatomic heart structure and electrophysiology. Due to longer QT intervals and intramural Purkinje fibers, the pig's heart is significantly more sensitive to external electrical stimulation than a human heart [12–15]. The primary utility of the swine studies is establishing the critical DTH distance. A secondary value is in providing estimates of the lower limits of body mass for safety.

Some swine studies have induced VF with the X26 CEW. The swine studies as a whole demonstrate that the theoretical risk of electrocution by CEW appears to be confined to very small or very thin humans. Walcott et al. have shown that swine are 3 times more sensitive to epicardial electrical current than humans [16]. The largest swine with intact electrophysiological properties that was electrocuted by a CEW, was reported by Valentino et al. and had a weight of 36 kg [17]. The levels of dangerous electrical current scale with body mass just like any drug dosage. Since swine are 3 times as sensitive to electrical current (as humans) we can roughly translate the Valentino 36 kg pig to a 12 kg (26 lb) human [16]. This calculation uses a direct -proportion relationship for VF threshold to the body mass. Some authorities have published that the danger level scales with the square root of body mass [18]. With such a relationship, the Valentino pig is equivalent to a 21 kg (46 lb) human. If we take the more conservative calculation, it is clear that the best evidence suggests that the risk of CEW electrocution is limited to humans with a body mass under 21 kg.

Nanthakumar et al. [6] were able to induce VF in a single 50 kg swine. However, in their study the animals were given epinephrine shortly before the electrical exposure, a drug that is known to significantly (but only temporarily) reduce the VF threshold (with high levels of simultaneously infused epinephrine or norepinephrine the VF threshold briefly drops by up to 26–28% [19, 20]).

The critical DTH probe distance was studied by the University of Wisconsin Biomedical Engineering Department using spacings of 2-12 mm [21]. They found that the probe tip had to be 5.8 ± 2.0 mm from the epicardium for induction of VF with an X26. Lakkireddy et al. (Cleveland Clinic) also

tested close-probe spacing to the heart (12–23 mm) without inducing VF [22, 23]. Based on these results, the probability of inducing VF (in swine) based upon dart-to-heart (DTH distance) can be estimated by logistic regression [24]. The probabilities are shown in Fig. 1.

The critical DTH distance in humans will obviously be less than that in swine since swine are more sensitive to external currents inducing VF [24]. This can be quantified as seen in Table 1.

A "linear" relationship between the current density and DTH would suggest dividing the 5.8 mm swine value by the 3× swine-to-human sensitivity ratio to get a predicted human DTH distance of 1.93 mm. However, the current density varies with the distance from the tip (of a percutaneous needle electrode) by a − 5/4 exponent so the correction is slightly smaller at 2.41 giving an expected DTH mean value of 2.41 mm [24, 25]. With high catecholamine levels, the DTH distance increases to 3.10 ± 1.09 mm as shown in the last row of Table 1, since that would decrease the VFT temporarily [19, 20].

Because of the human anatomy, such a constellation is very unlikely in the case of a healthy adult. However, a correspondingly low DTH is possible in children with a cachectic body habitus with a direct hit over the heart. Risk calculations, using a distribution of body habitus and echo and CT scan data have been performed [26, 27]. The VF risk is estimated at approximately 1 in 3 million probe uses.

The primary utility of the swine studies is establishing the critical DTH distance at approximately 3 mm in humans. A secondary value is in providing estimates of the lower limits of body mass for safety at approximately 21 kg in humans.

Finite element modeling

A number of computer simulation models have been published assessing the risk of VF resulting with various probe placements, body habitus, and CEW waveforms [26–32].These publications are listed for reference, but are not reviewed further in this paper.

Human results

There have been 66 humans monitored continuously during a CEW discharge with precordial probes (Table 2). In these 66 cases there was no VF induction and there was only 1 recorded case of cardiac pacing. The pacing report was with an experimental prototype CEW which was never manufactured. However, we are conservatively listing it here.

The margin between cardiac pacing and the induction of VF is quite high and typically found to be about 12:1 [35]. Hence, these clinical data suggest a very low risk of VF induction even with precordial probes.

As of 1 September 2017, the primary manufacturer of CEWs reports 3.44 million field uses of CEWs. In 49% of field uses a probe lands in the front chest [36]. Thus, we estimate that there have been 1,690,000 field uses with a probe in the chest. There have been 12 published case studies suggesting electrocution by a CEW giving a potential incidence of 3.4×10^{-6} per field use and 7.1×10^{-6} per precordial probe application [37–42]. A total of 9 of these 12 reports were from the paid expert witnessing activities of a single retired cardiologist [39, 40]. Only 7 of these cases presented in VF. Another 2 can be eliminated as they included a case where the

Fig. 1 Probability of VF induction vs dart-to-heart distance in swine. Human values are lower

Table 1 Dart-to-heart distance (mm) for VF in X26 ECD

Condition	VFT Ratio	DTH Ratio	DTH mean	DTH stdev	Maximum	Notes
Swine			5.8	2.04	8.0	Wu-Webster [21]
Human	3.0	2.41	2.41	0.85	3.32	Walcott [16]
With maximum catecholamines	0.73	0.78	3.10	1.09	4.27	Han 26% & Papp 28% VF threshold reduction [19, 20]

VFT VF thresholf, *DTH* dart-to-heart distance

probes missed the subject, and another case that had a documented pulse afterwards. See Table 3 for details of the remaining cases.

Electrocution can be diagnostically eliminated in these cases by [43–45]:

1. Dart-to-heart ≥20 mm (vs. 3 mm): 5/5
2. Failure of prompt defibrillation: 5/5
3. Breathing >1 min: 3/5 (2 Unknown)

The success rate and elapsed time duration for defibrillation of VF can be used to assess its origin. Electrically-induced ventricular fibrillation is easier to defibrillate than ischemically-induced VF [43]. All of the alleged VF case reports were studied by the Canadian Council of Science [46]. Their report was produced by a deliberative panel that included numerous Canadian and U.S. experts on electrical weapons and arrest-related-death, that itself was extensively peer-reviewed. This panel, without equivocation, especially dismissed the controversial expert witness case series [39].

> "The study by Zipes is particularly questionable since the author had a potential conflict of interest and used eight isolated and controversial cases as part of the analysis."

In summary, the known scientifically sound data suggest that the risk of CEW-induced VF is extremely low. It appears to be confined to an extremely small or thin subject with a probe nearly touching the right ventricle. To date, no reported cases of electrocution have withstood careful scrutiny [47].

Table 2 Human testing with precordial probes and continuous monitoring

Author	Year	n	Exposure (s)	Monitoring	Capture	VF
Stopyra [2]	2017	3*	5	Electrogram	0	0
Dawes [33]	2010	10	5	Echo	0	0
Ho [34]	2011	53	10	Echo	1	0

*Stopyra had 4 subjects but the pacemaker prevented recording in a single case

Troponin changes

We found 10 studies with a total of 421 subjects with troponin measurements before and after a CEW exposure. Details are in Table 4. With a single exception (1), there were no increases above the testing cutoff level. Ho reported a single subject with an elevation at the 24-h post-exposure period [55].This subject was evaluated in a hospital by a cardiology team and no clinical evidence of acute myocardial infarction was identified and no evidence of cardiac disability was demonstrated. The troponin I value returned to normal within 8 h of its reported elevation. This subject was a very fit and athletic individual and had performed a rigorous aerobic workout regimen without difficulty about 3 h before the CEW exposure. There were several explanations offered as possible causes by the consulting cardiologists. These included laboratory error, delayed clearance of troponin related to subject baseline physiology, or idiopathic and indeterminate etiology. There was agreement that there was no indication of myocardial damage or ischemia, and the subject was allowed to return to regular duty without limitations.

As of today, CEW exposure does not appear to increase troponin levels. Creatine kinase and myoglobin levels are covered in the skeletal muscle section below.

Pacemaker & ICD interactions with CEW

By the IEC (International Electrotechnical Commission) standard 60,601, any implantable medical device must withstand a 360-joule (J) external defibrillation shock [56, 57]. The popular TASER X26E CEW delivered pulses that were ~ 0.1 J and hence there is an extremely large safety (3600:1) margin for damage [58, 59]. Later CEW models, such as the X2 and X26P CEWs deliver similar energy pulses.

Resets of settings have not been reported with the CEW in either animal studies or human case reports [58–66]. It may also be possible that the implanted device temporarily operates in "noise-reversion", a mode of limited pacing capability caused by detection of high-frequency noise.

Table 3 Reported VF cases from CEW use

Age/ Race	DTH (mm)	Breathing (minutes)	Failure of prompt defibrillation	Cardiac pathology	Toxicology
25 B	>20	UNK	Y	Hypertrophy, fibrosis	alcohol (0.15%) & THC carboxylic acid
48 C	No pene-tration	UNK	Y	Acquired long QT from schizophrenia and low K^+, Mg^+, Ca^{2+}, and Na^+	alcohol (0.36%), THC & olanzapine
17 B	Right side	4	Y	Hypertrophic cardiomyopathy	THC suspected (not tested)
17 B	50	4	Y	none on autopsy	Alcohol (0.25%) & THC carboxylic acid
16 B	55	8	Y	Arrhythmogenic right-ventricular cardiomyopathy	THC & THC carboxylic acid

Pacemaker

Out of 3.44 million field uses there have been only a handful of reports of pacemaker patients receiving a CEW exposure. Cao reported on a rioting prisoner who received TASER probes in the chest but reported that he had not felt anything [62]. A later interrogation of the pacemaker showed that his heart was paced very rapidly during the TASER CEW application. Vanga reported on 2 pacemaker patients that had not experienced any rapid pacing and later pacemaker interrogation showed no effects on the pacemaker [61].

A legitimate reason for concern is that the pacemaker and the lead that connects it to the inside of the heart could act as an "antenna" and carry some of the CEW pulses to the inside of the heart, thus effectively reducing the dart-to-heart (DTH) distance to zero. This could rapidly "pace" the heart and this rapid pacing could theoretically induce a cardiac arrest.

Another rare, but potential concern, regards pacemaker "inhibition." This is only a concern in the 2–10% of pacemaker patients that need the pacemaker all the time. It is possible for the pacemaker to interpret the CEW pulses as cardiac QRS complexes and decide that pacing is not required and hence temporarily shut off the pacemaker. This was seen in the Lakkireddy swine study [58] but not in the larger Khaja swine study [65]. This was also not seen in the 2 pacemaker patients of the Vanga case series [61]. If this inhibition was to occur then the patient could have a temporary cardiovascular collapse, faint, and potentially suffer a head injury from the fall-related head impact. The cardiovascular collapse would stop the fight and an alert officer would presumably turn the CEW off immediately.

Thus, there is a theoretical risk of a serious adverse event in applying CEW probes to the chest of a pacemaker patient. However, the average pacemaker patient is in their 70's or 80's while the age of subjects receiving police force is 32.4 ± 11.2 and hence the population overlap is extremely small [67].

Implantable cardioverter defibrillators

The potential complications with an Implantable Cardioverter Defibrillator (ICD) are significantly different from those with a pacemaker. To begin with, the antenna effect is not expected as the circuitry in an ICD is more sophisticated and should not pass any voltage on the housing down the lead into the heart [59]. However, an ICD can be confused by the rapid pulses from a CEW and falsely conclude that ventricular fibrillation (VF) is present [63, 64]. In the Vanga case series, this occurred in 1 of 4 ICD subjects that received a CEW application [61]. This was also seen in the ICD patients of Haegeli [63] and also Paninski [64]. In these

Table 4 Troponin testing

Author	Year	Number of subjects [n]	Exposure (s)	Troponin Increase (ng/mL)	Cutoff (ng/mL)
Ho [46]	2006	66	5	single case with 0.6	0.3
Vilke [48]	2007	32	5	0	0.2
Sloane [49]	2008	66	4.4	0	0.2
Dawes [50]	2009	16	5	0	UNK
Ho [51]	2011	25	15	0	UNK
Moscati [52]	2010	22	15	0	0.3
Dawes [33]	2010	11	30	0	0.08
Ho [53]	2010	12	5	0	0.2
VanMeenen [54]	2010	118	≤ 5	0	0.1
Ho [34]	2011	53	10	0	0.04

cases, the ICD then began charging the shock capacitor and then double-checked the cardiac rhythm after the capacitor was fully charged. It takes an ICD about 8–10 s to recognize VF and to then charge the capacitor. Since this process required more than the 5-s standard CEW application there was no defibrillation shock delivered by the ICD. Limiting the CEW-exposure duration to 5 s should thus minimize the risk of an ICD shock. However, longer CEW discharges are likely to trigger such shocks.

Acute clinical cardiac data of CEW

Due to recruitment challenges, there are minimal prospective clinical data on implantable device interactions. The single study is that by Stopyra et al. [2], which involved 4 patients. Adults scheduled to undergo diagnostic electrophysiology studies or replacement of an implanted cardiac device were enrolled. A total of 157 subjects were reviewed for possible inclusion and 21 were interviewed. Among these, 4 subjects agreed and completed the study protocol. Sterile subcutaneous electrodes were placed at the right sternoclavicular junction and the left lower costal margin at the midclavicular line to simulate CEW probes. A model X26 CEW was attached to the subcutaneous electrodes and a 5 s discharge was delivered. Continuous surface and intracardiac EKG monitoring was performed. All subjects tolerated the 5 s CEW discharge without clinical complications. There were no significant changes in mean heart rate or blood pressure. Interrogation of the devices after CEW discharge revealed no ventricular pacing, dysrhythmias, damage or interference with the implanted devices.

One large retrospective study on clinical data covered emergency department patients, who were subjected to CEW [68]. Out of 1123 individuals against whom a CEW was used, only 4.4% were admitted for medical reasons. In 2 cases, elevated potassium levels were recognized and 11 had increased creatine kinase. The majority of patients (93.9%) either had a history of drug and/or alcohol abuse or a psychiatric diagnosis and over 70% of the tested individuals were positive for recreational drugs.

Impact of CEW on the musculoskeletal system

The electrical waveform of CEW creates an electric field between the dart electrodes, which stimulates type A-α motoneurons, initiating a tonic muscle contraction throughout parts of the body. Such tension of the musculoskeletal system could lead to muscle cell damages with release of the intracellular muscle enzyme creatine kinase (CK), myoglobin, and potassium. In this context, the major concern is, whether this can cause an extreme increase of CK with medical complications, such as rhabdomyolysis with the danger of kidney failure.

There are a small number of experimental studies on this matter. A few published animal studies by Jauchem et al. found a correlation between CEW-exposure and an increase in CK-levels [69–71]. While no significant increases in CK could be found after multiple 5 s exposures with a 5 s pause between the discharges [69, 70], a continuous 30 and 60 s application [71] showed relevant changes. These results could not be observed by a similar study performed by Dennis et al. [72]. This research group exposed 11 pigs to an 80 s discharge (2 x 40s) from a CEW. Creatine values did not change significantly after the exposure and they did not exceed normal levels in any of the animals.

We found 3 papers regarding CK levels covering 241 patients. VanMeenen reported on results with 65 volunteers [54]. Dawes et al. reported on human studies with a total of 156 volunteers [73]. They both found an increase in CK levels after a CEW exposure (Table 5), but no clinically significant signs or symptoms were noted. In 2014, Ho et al. [74] reported on 10 subjects with essentially no pooled median change in CK, but the median pairwise shift was −5.5. A challenge in these studies is that subjects sometimes disregard protocol instructions and then exercise vigorously between CEW exposure and the blood sample taken the next day and this can give an artificially increased change. Similarly, some subjects had exercised before the CEW exposure and they thus had an elevated "baseline" value so that they showed a decrease in CK the next day.

Other contributing factors, such as extreme physical exertion [73] or intoxication [23] might additionally elevate CK-levels. Sanford et al. [75] presented two patients, who were controlled with a CEW and afterwards developed symptoms

Table 5 Change of creatine kinase 24 h after exposure to CEW

	Number of subjects [n]	Exposure time [s]	Change from baseline [U/l]
VanMeenen [54]	65	5	7.3 (mean)
Dawes [73]	81	5	26.5 (median)
Dawes [73]	64	10	303.0 (median)
Dawes [73]	11	30	47.0 (median)
Ho [74]	10	10	0.5* (median)

*The median pairwise shift was −0.45 but the pooled median difference was 0.5

Table 6 Change of potassium immediately after exposure to CEW

	Number of subjects [n]	Exposure time [s]	Change from baseline [mmol/l]
Vilke [48]	32	5	+0.1 (mean)
VanMeenen [54]	65	5	+0.2 (mean @24 h)
Ho [55]	66	5	−0.2 (median)
Dawes [77]	53	10	+0.2 (median)
Ho [78]	38	15	−0.4* (median)

*Subjects initially engaged in vigorous exercise, which raised their baseline K^+

of rhabdomyolysis. They reached CK-levels of 3166 U/l and 8086 U/l. Since one of them was under the influence of cocaine and the other one involved in a prolonged physical confrontation with the police, other causes for the clinical symptoms have to be taken into account [76].

The only paper reporting myoglobin levels was by Ho et al. [55]. After a 5 s exposure, myoglobin increased from 32.4 ± 15.1 ng/ml to 45.5 ±27.1 and was then 51.3 ± 29.8 at 24 h.

When muscle tissue is stressed, potassium leaks out of exerting muscle tissue and hyperkalemia can occur. After the physical activity, the circulating catecholamines can reverse this effect and cause a temporary hypokalemia. Both conditions could affect the heart and cause deadly cardiac arrhythmias. There has been concern that the application of a CEW could release large amounts of potassium into the human body, causing a cardiac arrhythmia. However, the 5 human studies on this topic showed no clinically important changes of potassium (Table 6).The largest change was in the 2009 Ho paper [78], in which subjects exercised to exhaustion before the blood sample and CEW exposure. At that point, their K+ levels were probably renormalizing and thus the CEW exposure found a decrease of 0.4 as shown in the last row of Table 6.

In summary, recent clinical research could not prove a direct link between CEWs and the development of rhabdomyolysis or hypo−/hyperkalemia. Even though an increase in CK cannot be excluded, no clinical signs have been noted.

Conclusions

Electrical weapons are weapons and as such there are risks associated with their usage. These weapons satisfy all relevant electrical safety standards and there are, to date, no proven electrocution incidents, in spite of millions of exposures.

A potential cardiovascular risk has been recognized by some of the experimental animal data. However, the majority of human research data suggests that cardiac capture is directly related to the dart-to-heart distance and as such is too large to be of risk for an exposed healthy adult.

There is a theoretical risk of a serious adverse event when applying CEW probes to the chest of a pacemaker patient, but no incident to this effect has been reported in police use of CEWs. There are no demonstrated risks to the skeletal muscle system as creatine kinase, myoglobin, and potassium are not significantly affected.

A responsible use of a CEW, within the guidelines proposed by the manufacturer, on a healthy adult, does not imply a significant health risk for the exposed healthy adult.

Key points

1. Data considering CEW use could not demonstrate relevant pathophysiological cardiac changes or arrhythmias in patients.
2. The risk of CEW-induced VF is extremely low and related to the dart-to-heart distance.
3. No direct link could be found between CEWs and the development of rhabdomyolysis or hypo−/hyperkalaemia.
4. A theoretical influential risk of CEW on a pacemaker or defibrillator exists, but no incident has been reported in police use.
5. A responsible use of CEW does not imply a significant health risk for an exposed healthy adult.

Compliance with ethical standards

Conflict of interest This paper is a result of literature research, which was not funded. SNK, HC, and MWK are members of the scientific medical advisory board of Axon Int. (fka TASER). MWK also is on Axon corporate board. HC & MWK have been expert witnesses in law-enforcement litigation and HC has been an expert witness in cases of arrest related deaths involving CEWs.

J. Adamec has no conflict to declare.

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