



Cardiovascular Risk and the TASER®: A Review of the Recent Literature

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Although credited with saving hundreds to thousands of lives, controversy exists regarding an increasing number of deaths temporally associated with TASER® use [1,2,3,4]. Investigative reports have uncovered undisclosed relationships between TASER International and purportedly independent studies on the safety of the TASER®, raising questions of bias [5,6,7,8]. In this climate of uncertain risk, law enforcement and the public at large are left with many unanswered questions regarding the safety of the TASER®.

The only federal regulatory review of stun gun safety was performed by the Consumer Products Safety Commission in 1976, on the first generation TASER® [3]. The technology was deemed "not likely to be lethal to normally healthy adults". No formal FDA safety review has ever been performed, as the TASER is not classified as a medical instrument. The purpose of this paper is to review recent studies involving the TASER, and to place them in the context of the increasing number of deaths.

Overview

The TASER® conducted energy weapon system has been commercially available and used by law enforcement since 1974 [9]. First and second generation TASER® systems affected only the sensory motor system, and were designed to gain suspect compliance through painful stimuli. In contrast, newer generation systems (3rd generation M26, 1999, and 4th generation X26, 2003) affect both the sensory and motor nervous systems, through a mechanism TASER International refers to as electro-muscular disruption (EMD). As a result, compliance is achieved primarily by muscular incapacitation rather than by noxious stimuli [9].

Much press has surrounded the fact that third and fourth generation TASER® systems have a peak voltage of 50,000 volts. However, both systems operate at relatively low amperage. TASER International reports that the average current of the TASER® X26 is 2.1 mAmps [1,9,10]. As a consequence, the X26 delivers 0.36 joules per pulse. The predominant question concerning the safety of the TASER® has revolved around whether this unsynchronized energy discharge is capable of precipitating a malignant ventricular dysrhythmia, effectively causing an R-on-T PVC phenomenon.

But does this actually happen?

Animal Studies

Initial safety experiments utilized an open chest canine model. Investigators were unable to induce ventricular fibrillation even with electrodes implanted directly into the myocardium and the administration of epinephrine, ketamine, and isoproterenol as pro-dysrhythmic agents [10,11]. However, dose and route of administration remain unclear.

In November 2004, the US Air Force Research Laboratory (AFRL) presented results of a study (Technical Report AFRL-HE-BR-TR-2004-0094, September 2004) at the Non Lethal Technology and Academic Research (NTAR). Unfortunately, dissemination of the study is restricted under Department of Defense directive 5230.25, and as a result full details of the study have never been made publicly available [3,12]. The study came to public attention when CBS News reported that a study "found that repeated shocks from a Taser stun gun led to heart damage in pigs"[13]. Although study information is sparse, it involved a pig model of repeated TASER® discharge. Each animal received a total of 18 5-second TASER® discharges over a 3-minute period, and the process was repeated after a 60-minute rest period. [3,12].

No animal died during the study. Troponin T was found to be elevated after the study, but the elevation was not significantly different from that seen in control swine [14]. Actual troponin T levels have never been released for review. Pathology purportedly failed to demonstrate evidence of myocardial injury. CBS News acknowledged that the levels were statistically not significant, but called "the very existence of troponin T medically significant" [14]. While the number of shocks employed in this study falls well above the norm, cases of multiple TASER® shocks have been reported in the press.

The most recent animal safety study is the 2005 PACE study [15]. A custom-built device was used to simulate the effects of TASER® X26 deployment in a porcine model of ventricular fibrillation (VF) induction. The device was designed to produce discharges at the standard TASER® X26 level and beyond. A total of 9 pigs were used, and a safety index was calculated from the ratio of minimum VF induction threshold to standard TASER® X26 discharge level. The authors concluded that "discharge levels for standard electrical NMI [neuromuscular incapacitation] devices have an extremely low probability of inducing VF."

Several potential problems exist with the PACE study. The first involves validity in using a custom-built device rather than an actual TASER® X26. While necessary for the experimental design, one might argue that the experimental device is not the device used by law enforcement on the street. The second is that animals were maintained under isoflurane anesthesia, potentially blunting central sympathetic response to the TASER® device. In no case did the experimental device induce VF at discharge levels equivalent to those produced by the TASER® X26. However, with only 9 pigs, the 95% confidence intervals for VF induction at that discharge level range from 0% - 29.9%. This begs

the question: What defines "extremely low probability"? Furthermore, the study was never designed³ to "address the safety index as it relates to individuals with arrhythmias, pacemakers, or implantable cardiac defibrillators." As such, it may not reflect the effects of TASER® discharge on an individual under the influence of an arrhythmogenic agent, such as cocaine. From the standpoint of potential bias, three of the four authors either worked directly for or consulted with TASER International.

One aspect of this study does deserve closer attention. The authors found that weight was an independent factor in predicting VF inducibility. As weight increased from 30 to 117 kg, the safety index increased from 15X to 42X ($p < 0.001$). As such, the use of the TASER® against smaller adults and children may pose a greater risk of harm than use against larger suspects. Police in Miami, FL, report at least 31 deployments on individuals 17 years of age or younger since 2003, including a deployment against a 6 year-old in 2004 [16].

Human Studies

Human data on the medical effects of TASER® use are equally sparse. TASER International reports that at least 100,000 volunteers (including this author) have received a TASER® discharge. To date, no death has been reported. TASER International uses this data to support the safety of its product. Although not specifically described as such, TASER International is effectively performing an epidemiological cohort outcome study on the safety of the TASER®. However, one must use caution in analyzing the data in this manner, as the potential exists for bias through the healthy worker effect. One must presumably be free of significant mental illness (eg. schizophrenia), and not be under the influence of mind-altering substances such as cocaine or PCP, in order to function as a peace officer and undertake TASER® training. As such, results in this population may not translate to the population likely to require TASER® deployment.

A 2005 study monitored the cardiovascular effects of the TASER® X26 in healthy human volunteers [17]. Twenty police officers had 3-lead ECG monitoring performed before, during, and immediately after TASER® deployment. Primary experimental end-points included changes in cardiac rate, rhythm, morphology, and intervals. Mean shock duration was 2.4 sec (range 1.2 - 5 sec). A 15 beat per minute (bpm) mean increase in heart rate occurred post-TASER® shock ($p < 0.001$). Interestingly, the mean heart rate prior to TASER® deployment amongst healthy police volunteers was 127 bpm, with a range of 80 - 160 bpm. No ventricular dysrhythmia occurred during the study, and QRS and QTC intervals did not change. The authors concluded that no significant cardiac dysrhythmias occurred immediately after receiving a TASER® shock.

This study is an extremely useful beginning in the attempt to understand the cardiovascular effects of the TASER®. However, it too has some potential flaws. The first is the fact that the 20 subjects were police volunteers, again raising issues of the healthy worker effect. Perhaps different results would be found in patients on psychotropic medications or under the influence of sympathomimetic drugs. The second is that the mean monitoring time post strike was only 16.3 seconds. Most TASER® deaths are delayed minutes to hours [12]. Lastly, as this is a negative study, the question of sufficient power is an

issue. Using an absolute QTc interval greater than 500 msec or a 60 msec change in QTc interval (⁴? QTc 60, the typical minimal significant increase used by the FDA in determining drug safety) as experimental end-points, and a one-sided t-test, a sample size of 20 would provide an 8.4% chance of an event even with a negative study (Table 1) [18,19,20]. In the setting of a negative study, a minimum sample size of 300 would be required to decrease the upper confidence limit to less than 1%, and a sample size of 3000 would be required to decrease the upper limit to 0.1%.

In-Custody Deaths Reconsidered

Why are there no deaths in the volunteer group (n = 100,000) and relatively few deaths in the operational deployment group (n = 50,000)? According to the published reports, the TASER® has now been temporally associated with the deaths of 120 individuals since 1999 [4]. In 17 of these cases, coroners cited the TASER as "a cause, a contributing factor, or could not be ruled out." To date, all the experimental studies examining the cardiovascular safety of the TASER have focused upon the potential of an electrical discharge to precipitate a malignant ventricular rhythm. The question becomes whether these studies are actually looking at the correct end-point. Consider these recent TASER deaths, as described in the lay press.

"A Waco man has died after being shot several times with a Taser by police... He was shot with Tasers about four times, none of which had any effect, according to Waco police spokesman Ryan Holt. "There was no sign that he was slowing down," he said. "He just kept fighting officers and kept fighting officers." Holt said [suspect] complained he was having trouble breathing after sitting down. Officers immediately called for an ambulance. By the time the ambulance arrived, [suspect] was not breathing, and officers were performing CPR on him, Holt said." [21]

"Police reports said [suspect] continued to fight even after police doused him with pepper spray and shocked him repeatedly with a Taser. Eventually, the officers wrestled him down in about 20 inches of swampy water, where, police reports state, he was hit with a Taser three more times. Police reports said [suspect] was still breathing after officers finally handcuffed him and pulled him onto dry land. But he quit breathing about a minute later. Rescue personnel and officers were unable to revive him." [22]

In each case, the decedent was behaving violently or erratically, necessitating law enforcement intervention. In each case, there was a violent struggle, in which the suspect seemed to have superhuman strength and be immune from less-lethal forms of restraint. Most importantly, in each case, the death was not a sudden event immediately at the time of TASER impact or at peak struggle, as would be expected from a malignant dysrhythmia. In fact, the death was a unresuscitatable respiratory arrest. These cases are all classic examples of excited delirium (ED).

As previously discussed, excited delirium is a hypermetabolic state with features similar to neuroleptic malignant syndrome (NMS) [23,24,25,26,27,28,29]. Lethal excited delirium follows a four-step sequence

of hyperthermia, agitated delirium, respiratory arrest and death [24]. Metabolic features include rhabdomyolysis, hyperkalemia, and profound metabolic acidosis [23,24,29,30]. In case reports in which the patient was monitored at the time of cardiac arrest, ventricular dysrhythmias accounted for only 1 of 14 deaths [26,31]. It may be for this reason that TASER® safety studies, have failed to detect any ventricular dysrhythmias, and that healthy human volunteer strikes have not resulted in death.

Excited Delirium, TASER®, and Use of Force

It is not surprising that excited delirium deaths are temporally associated with TASER® deployments. Individuals with excited delirium tend towards violent and erratic behaviors, and manifest feats of superhuman strength. The mean number of officers required to restrain an ED suspect is four [25]. The TASER® is likely to be deployed on these individuals, in an attempt to avoid use of lethal force. According to TASER International, 87% of police departments place TASER® before OC pepper spray on the use-of-force continuum. As such, these deaths may not reflect a cause and effect relationship between TASER® use and in-custody death, but rather be confounded by use of force spectrum response. Moreover, lethal excited delirium was first described in 1849, long before the TASER® existed [25]. Numerous ED deaths have occurred in the absence of TASER®, including a death in Cincinnati, OH which received national attention in 2003 [12,23].

Animal data demonstrate that restraint and increased circulating catecholamine levels, both of which occur during a violent struggle, increase the risk of death in cocaine-administered rats [24,32]. As such, one might speculate that it is the struggle which in turn necessitated TASER® deployment, rather than the actual TASER® discharge, that causes death in these individuals. Unfortunately, this has never been studied.

TASER® Safety and Excited Delirium

Does this therefore mean that the cause of these in-custody deaths is entirely excited delirium and therefore that the TASER® is safe? Not necessarily.

Individuals with excited delirium may demonstrate a profound metabolic acidosis. In one study, arterial pH ranged from 6.25 - 6.81 [30]. Presumably, the profound acidosis derives at least in part from the violent struggle and lactate production. One overlooked aspect of the AFRL NTAR study was the effect of the TASER® on acid-base status [3,12]. Immediately after the initial 18 TASER® strikes, whole blood pH fell from approximately 7.4 to approximately 7.0, and by one hour post-exposure, blood pH had only risen to 7.2. Lactate levels peaked at 15 mmol/L 30 minutes after TASER® strike, up from less than 3 mmol/L pre-deployment. The pre-deployment pCO₂ was less than 50 mm Hg, and increased to nearly 100 mm Hg immediately post discharge. Unfortunately, there is no information to state whether this whole blood was derived from arterial or venous sampling.

It has been argued that these findings are similar to those observed after 30 seconds of strenuous

exercise, something "most humans can perform without dropping dead UNLESS there are other factors at work such as an overdose of a stimulant, congenital disease, etc, which are all factors associated with or without the use of a TASER device" [33]. However, that argument is a little disingenuous. First, although venous pCO₂ did increase to 106 mm Hg in the Kowalchuk et al study, as did muscle pCO₂, arterial pCO₂ actually declined from 41 mm Hg pre-exercise to 37 mm Hg post exercise [34]. Peak lactates in the Kowalchuk et al. study were 14 mEq/L (arterial) and 18 mEq/L (venous). Only peak muscle lactate reached 47 mEq/L. Moreover, excited delirium patients are not like "most humans" and as such, further acidemia might result in significant medical compromise. The fact that suspects may have an on-going medical process does not necessarily imply that the TASER® is incapable of causing further harm. Consider the analogy of cocaine chest pain. Although the chest pain may be due to cocaine, beta-adrenergic blockade may still result in harm to the patient.

Finally, death in excited delirium appears to be due to respiratory arrest. As such, anything that interferes with respiration might hasten or precipitate the lethal process, as well as further compound the acidemia by adding a respiratory component to the profound metabolic acidosis. TASER International, in its discussion on duration of field applications, comments that TASER® discharge across the chest may cause "sufficient muscle contractions to impair normal breathing patterns" [1]. Although TASER International concludes that this is not a significant concern for a short duration (1 cycle - 5 second) discharge, it may be "a more relevant concern for extended duration applications."

Does this mean therefore that TASER® use is contraindicated in the setting of excited delirium? Again, not necessarily. As TASER® deployment may shorten the duration of physical struggle, and its metabolic consequences, it has been speculated that it might actually reduce risk of subsequent death. [12]

Conclusions

As can be seen, there remain more questions than answers. Available studies, although limited, would appear to suggest that the TASER® does not precipitate ventricular dysrhythmias. The question remains whether the experimental end-point under investigation is even correct or appropriate. Studies should be undertaken to identify subgroups of individuals at higher risk for in-custody death after TASER® deployment. As part of the risk assessment process, an analysis of TASER® discharge outcomes specifically against individuals with suspected excited delirium should be undertaken, so as to determine the actual risk of death when deployed against this subgroup.

Until more studies can be performed specifically addressing these questions, the following recommendations appear reasonable:

1. First and foremost, law enforcement and EMS personnel should be trained in the recognition and management of excited delirium.
2. Use of multiple TASER® discharges, while not always avoidable, should be minimized

wherever possible.

3. Use of TASER® against smaller individuals should be undertaken judiciously.

In the end, the continued use of TASER® remains one of public perception and risk-benefit analysis. Law enforcement and the general public must understand that the term non-lethal, as defined by the US Marine Corps and used by TASER International, does not imply lack of ability to kill, but rather an intent that the weapon system "incapacitate personnel or material, while minimizing fatalities, permanent injury to personnel, and undesired damage to property and the environment" [3]. The TASER® system should be viewed more accurately as less-lethal, rather than non-lethal or less-than-lethal.

As with any mechanical device, an element of risk will always exist with use. Placed in perspective, the likelihood of death after being shot by a police officer is approximately 50% [35]. If one were to argue that all 120 in-custody deaths are due solely to the TASER® (an argument for which there is essentially no support), then the risk of death after TASER® discharge remains only 0.24%, supporting the use of TASER® as an option of lesser risk.

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Table 1: Summary of the Upper Limit of a 1-Sided 95% CI, for a Given Sample Size and Zero Observed Events.**Sample size 0 events**

20	8.4%
40	4.4%
60	3.0%
100	2.95%
150	1.98%
200	1.49%
300	0.99%
500	0.6%
1000	0.3%
1500	0.2%
3000	0.1%