THE EFFECT OF TASER ON CARDIAC, RESPIRATORY AND METABOLIC PHYSIOLOGY IN HUMAN SUBJECTS

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OVERVIEW OF STUDY

Study Objective: We studied the effects of a single TASER exposure on markers of physiological stress in humans in a two phase study. Phase one looked at subjects exposed to a TASER at rest, and phase two looked at subjects exposed to a TASER after vigorous exercise.

Methods: This is a prospective controlled two phase trial investigated the effects of a single TASER exposure. As part of their police training, healthy law enforcement officers received a 5-second TASER electrical discharge. Measures followed before and for 60 minutes after a exposure included: minute ventilation (VE), tidal-volume (TV), respiratory rate (RR), end-tidal PCO_2 (PETCO2), oxygen saturation (O2sat), heart rate (HR), blood pressure (SBP/DBP), arterialized blood for pH, pO2, pCO2, and lactate, and venous blood for bicarbonate, and electrolyte. Data were analyzed using a repeated measures ANOVA and paired t-tests.

Results: VE, TV, and RR increased from baseline at 1-min post exposure. Blood pH decreased statistically, but clinically unimportantly, from baseline at 1-min post exposure. Blood lactate increased from baseline through 30 min post exposure. Bicarbonate decreased from baseline through 30 min post exposure. All of these measures returned to baseline levels. HR and SBP were higher before the TASER exposure than any time afterwards. All troponin I values were less than 0.2 μg/L. Ventilation was not interrupted and there was no evidence of either hypoxemia or CO2 retention. Preliminary results from Phase 2 (n=22) indicate no significant differences between control and taser groups after exercise.

Conclusions: A 5 s exposure of a TASER X-26 to healthy law enforcement personnel either at rest or following vigorous exercise does not result in clinically significant changes of markers physiological stress.

INTRODUCTION

Background

There has been growing public demand for effective, less lethal law enforcement weapons, which include blunt impact weapons such bean bag guns or rubber bullets, mace, pepper spray and batons. The TASER is an electrical law enforcement and self-defense device originally developed in the 1970s and manufactured by TASER International (Scottsdale, AZ). Early versions were bulky and often ineffective. Various models of the TASER device have been developed, and their newest version, the X26, differs from the prior model, the M26, mainly in the size and shape of the device.

The National Institute of Justice reports that 11,000 United States law enforcement agencies currently authorize the TASER device which is being carried by over 225,000 officers. Additionally, they report that over 120,000 U.S. citizens also have a TASER device. Although the actual number of uses is unknown, they have reported that the TASER has been used on over 600,000 volunteers during training and in over 425,000 "real-life" police confrontations. The manufacturer asserts that the device helps officers avoid the use of deadly force while lowering the risk of injury to officers.

The TASER X26 is designed to be deployed up to 7 m from the subject. The operator fires the device releasing two 9mm barbs attached to the gun by thin, 7-m copper wires. When the circuit is completed, an electrical pulse of 5 seconds duration is automatically delivered through the wires to incapacitate the subject by causing involuntary tetanic muscular contractions. The officer may deliver continued electricity by pulling the device trigger again.

Although the effect of the TASER is poorly studied, it is generally regarded as safe (1-3) and has been approved by the U.S. Consumer Product Safety Commission for the current

indication for which it is being used. Most of the data supporting the product's approval by the U.S. Consumer Product and Safety Commission was based on theoretical calculations and not on the basis of animal or human studies (4).

Importance

There have been a number of reports of sudden death following TASER administration. Amnesty International reports "152-TASER related deaths" since 2001 and the Arizona Republic reports "167 cases of death following stun gun use" since 1999 (5,6). The majority of deaths in humans who were exposed to a TASER device were associated with illicit drug use, especially phencyclidine, methamphetamine and cocaine (3,7-8). However, there have been several deaths reported in individuals after TASER exposure who were not under the influence of illicit drugs. These cases generally involved a clinical presentation of "excited delirium" and other co-morbid factors that were likely to be related as the cause of the suspect's death (9-11). Most case reports and police reports note that the suspect gets shocked with a TASER and then 5 to 40 minutes later the suspect then goes into cardiac arrest (12). If a lethal dysrhythmia, particularly ventricular fibrillation, was at fault from the electrical discharge, cardiac arrest would be expected to occur at the time of the TASER activation. However, if individuals were under the influence of sympathomimetic drugs like cocaine, methamphetamine or PCP or were having the clinical presentation of excited delirium other important clinically significant physiologic aberrations might hypothetically contribute to these sudden deaths

Goals of the Investigation

As the metabolic and ventilatory effects of an acute TASER exposure are unknown in humans, the aim of this two phase study was to investigate the extent of physiological stress following exposure to the TASER X26 in subjects at rest (phase 1) and after a period of vigorous exercise (phase 2). We monitored cardio-respiratory and blood parameters in police officer volunteers before, during and after a 5 second TASER exposure that was part of their police training. Because of the widespread and increasing use of TASER devices by law enforcement agencies, it is vital to assess whether its use on humans increases the risk physiological stress, ventilatory impairment, cardiac muscle damage or sudden death.

METHODOLOGY

Selection of Participants

Phase 1

This was a prospective study evaluating healthy police volunteers drawn from the pool of San Diego County (CA) Sheriff's officers who had already volunteered to have a TASER exposure as part of their tactical training. Inclusion criteria included subjects who were between 18 and 60 years of age. Prior to conducting the study, each subject was screened by the physician investigators to insure that he or she was free of acute illness or pregnancy that would prevent completion of the study; all women underwent a urine pregnancy testing. In addition, subjects weighing less than 45.5 kg or having a body mass index (BMI) less than 18 kg/m2 were excluded from the study. As there had been no previous human trials on the physiologic effects of the TASER on humans at the time this trial was going through the IRB, and the fact that most TASER activations used in the field were on larger individuals, a lower limit of weight and BMI was specified by our institutions' IRB committees. Initial cardiovascular screening of subjects was conducted using the Physical Activity Readiness Questionnaire (PAR-Q) (www.csep.ca/pdfs/par-q.pdf.). If the subject answered in the affirmative to any of the questions on the PAR-Q, they were excluded from the study. Although there were no occurrences, any subject with a reported history of recent illicit drug use within the last six months or a positive point-of-care urine drug screen for illicit drugs (Biosite urine drug assay San Diego, CA) would have been excluded from the study. In addition, subjects with a baseline pulse exceeding a rate of 120 bpm or a systolic or diastolic blood pressure greater than 150 or 90 mm Hg, respectively, or an abnormal 12-lead ECG were excluded from participation.

Phase 2

The subject pool and screening criteria were the same as phase 1 except that the inclusion criteria were modified to include subjects who were between 18 and 45 years of age. Exclusion criteria were modified to exclude subjects with a baseline systolic or diastolic blood pressure greater than 160 or 100 mm Hg, respectively.

Human Subjects Committee Approval

The study was approved by the University of California, San Diego and the San Diego State University institutional review boards for both Phase 1 and 2, and all subjects provided written informed consent before participating in the study.

Experimental Procedures

Phases 1 and 2

Each subject was exposed to a 5 second TASER electrical discharge. In Phase 1, darts from a standard TASER X-26 were shot into the subject's back by training personnel at a range of 2-3 m with the target laser centered on the subject's back between the shoulder blades. This was done with the subject in a standing position supported under each axilla by assistants to avoid falling when the TASER was activated. In phase 2, the subject performed an incremental cycling protocol to near-maximal effort, using standard exercise physiology protocols. The goal was to reach a heart rate of 85% of predicted HR_{max}. During the first part of the Phase 2 trial, the positioning of the subject was as in Phase 1 with a probe deployment to the back while in a standing position; however, partway through Phase 2, an adverse event of a thoracic spine compression fracture in one of the subjects resulted in a modification in the subject positioning

during the actual TASER deployment. The subject was positioned lying on his or her side on a mat with the TASER attached using alligator clips to the left upper anterior chest and the right belt line.

Methods of Measurement

Phase 1

Vital signs, including blood pressure (SBP/DBP), heart rate (HR), and pulse oximetry (O2Sat), were recorded prior to intervention and repeated at 5, 10, 15, 20, 25, 30, 40, 50, and 60 minutes post TASER activation. Ventilatory measures, including minute ventilation ($\dot{V}E$), tidal volume (TV), respiratory rate (RR) and end-tidal PCO_2 ($P_{ET}CO_2$), were obtained using a wireless portable metabolic measurement system (Oxycon Mobile,VIASYS Healthcare, Yorba Linda, CA). These ventilatory parameters were measured prior to, 1 min following the TASER activation as well as at 10, 30 and 60 minutes.

A 12-lead ECG was performed at baseline prior the TASER activation and repeated at 60 minutes post activation. These ECGs were evaluated in a blinded manner for ischemia as well as for interval changes.

Venous blood samples were drawn for electrolyte measures that included calcium (Ca^{2+}), sodium (Na^{+}), potassium (K^{+}) and bicarbonate (HCO_3^{-}) concentrations. These studies were drawn prior to intervention and repeated at 1, 10, 30 and 60 min post TASER activation. Subjects had an intravenous catheter placed in standard sterile fashion for ease of repeated blood draws. Arterialized capillary blood was drawn from a finger stick before and at 1, 10, 30 and 60 minutes post TASER activation for determination of pH, PO_2, PCO_2, and lactate concentration (i-STAT Portable Analyzer, Abbott Laboratories, Abbott Park, IL). The hand was placed in a

warm water bath (~41 ºC) for approximately 3 minutes, and blood was drawn using standard capillary sampling techniques. A final venous blood sample was drawn 6 hours post TASER activation for evaluation of troponin I utilizing the Advia Centaur Immunoassay System (Bayer Diagnotics, Terrytown, NJ).

Phase 2

Vital signs, including blood pressure (SBP/DBP), heart rate (HR), and pulse oximetry (O2Sat), were recorded prior to intervention and repeated at 5, 15, 30, 45 and 60 minutes post TASER activation. Ventilatory measures, including minute ventilation ($\dot{V}E$), tidal volume (TV), respiratory rate (RR) and end-tidal PCO_2 ($P_{ET}CO_2$), were obtained using a wireless portable metabolic measurement system (Oxycon Mobile,VIASYS Healthcare, Yorba Linda, CA). These ventilatory parameters were measured prior to, 5 min following the TASER activation as well as at 30 and 60 minutes.

A 12-lead ECG was performed at baseline prior the TASER activation and repeated at 60 minutes post activation. These ECGs were evaluated in a blinded manner for ischemia as well as for interval changes.

Venous blood samples were drawn for electrolyte measures that included calcium (Ca^{2+}), sodium (Na^{+}), potassium (K^{+}) and bicarbonate (HCO_3^{-}) concentrations. These studies were drawn at baseline, immediately after the subject reached 85% of predicted HR_{max} (bpm) one the exercise ergometer, and repeated at 1, 10, 30 and 60 min post TASER activation. Subjects had an intravenous catheter placed in standard sterile fashion for ease of repeated blood draws. Arterialized capillary blood was drawn using the technique as described in phase 1 from a finger

stick at baseline, immediately after exercise, and at 1, 10, 30 and 60 minutes post TASER activation for determination of pH, PO_2, PCO_2, and lactate concentration.

Each subject served as his or her own control by returning to our laboratory on a different date. In the control portion, there was no TASER activation, but rather, the subject would stand for the time period that was previously determined as the interval from exercise to TASER activation in the intervention portion of Phase 2. Arterialized capillary blood was drawn using the technique as described above from a finger stick at baseline, immediately after exercise, and at 1, 10, 30 and 60 minutes post simulated timing of the TASER activation for determination of pH, PO_2, PCO_2, and lactate concentration. Exercise was performed using the ergometer utilizing the same protocol as in the intervention phase.

The subject had baseline vital signs (blood pressure, heart rate, and blood oxygen saturation from pulse oximetry) and end-tidal CO2 recorded at the same time intervals as in the intervention portion of the Phase 2 study.

Outcome Measures

Phases 1 and 2

Outcome measures are as follows: Hypoxemia, as expressed by pulse oximetry <95%. Hypoventilation as evidenced by end tidal CO2 >40 mm Hg, and pCO2 > 40 mm Hg on arterialized capillary blood sampling. Changes in pH as evaluated by arterialized capillary blood sampling. Cardiac myocardial damage by assessing troponin I levels at six hours post TASER activation (Phase 1 only) as well as by evaluating 12-lead ECG at one hour post activation. Other outcome measures consist of vital signs, ventilatory function, and venous and capillary

blood indicators as mentioned above. The change in each measure was evaluated separately to assess any relevant change in the measure.

Primary Data Analysis

Phases 1 and 2

Power analysis indicated that 24 subjects with complete data would be needed to detect a pH change of 0.15 (7.40 to 7.25), assuming 80% power, an alpha of 0.05, and SD of 0.30. A study population of 30 subjects would adequately account for missing values for specific measures. All measures were reported as means and standard deviations (SD). A one-way repeated measures analysis of variance (ANOVA) was used to detect differences in respiratory, ventilatory and blood measurements. In Phase 1, when the repeated measures ANOVA results indicated significance at $p<0.05$, differences in means, pairwise comparisons using a paired t-test between the baseline and the four or nine subsequent measures (1, 10, 30 and 60 minutes post activation or 5, 10, 15, 20, 25, 30, 40, 50 and 60 minutes, depending on outcome measure), including only subjects with data for all time measures. In Phase 2, when the repeated measures ANOVA results indicated significance at $p<0.05$, differences in means, pairwise comparisons using a paired t-test between the baseline and the four or five subsequent measures (1, 10, 30 and 60 minutes post activation or 5, 15, 30, 45 and 60 minutes, depending on outcome measure), including only subjects with data for all time measures. Changes from baseline and subsequent measures are reported as mean differences and associated 95% confidence intervals (CI) with associated p-values. Because of multiple comparisons, a Bonferroni adjustment was used to define statistical significance ($p<0.006$ for vital comparisons and $p<0.013$ for all other outcome measures). However, because limited data have been presented regarding the physiological

effects of a Taser activation on healthy adults, $p<0.05$ were considered to be differences of interest. Clinical significance was determined based on current medical practice. All analyses were performed using SPSS for Windows version 14.0 (SPSS Inc., Chicago, IL).

DETAILED FINDINGS

Phase 1

Characteristics of Study Subjects

A total of 42 Sheriff's officers volunteered to participate in the study. 32 completed the study that included 27 men and 5 women. Ten subjects screened out prior to consent and enrollment and did not participate (6 due to elevated baseline systolic blood pressures, 1 with an abnormal baseline ECG, and 3 for taking medications for hypertension or cardiac disease). Complete cardio-respiratory measurements and blood samples were obtained from all 32 participants for each collection period. Subject characteristics are reported in Table 1a.

Main Results

Vital Signs

Repeated measures ANOVA results indicated statistically significant differences in vital sign means between measures for SBP ($p<0.001$), but no significant differences for HR or DPB (Figure 1). SBP decreased linearly prior to TASER activation (139 mmHg at baseline) to normal (123 mmHg at 60 minutes) (difference of 16, CI=12.7, 20.3, $p<0.001$). There were no significant differences between baseline (97%) and any subsequent measure for O2 sat and no measure was below 97% (data not shown). The change in SBP was not clinically significant.

Effects on Respiratory and Ventilatory function

Table 2a reports the effects of TASER exposure on respiratory and ventilatory measures (VE, TV, RR, PETCO2, pO2 and pCO2). Repeated measures ANOVA results identified significant differences in means between readings for all measures ($p<0.001$ for VE, TV and RR; $p=0.009$ for PETCO2) (Figure 2), but not for pO2 or pCO2 ($p>0.05$). VE, TV, RR all had an

initial significant increase from baseline to 1 minute after TASER activation (12.8 L/min, CI=8.5-17.1, p<0.001 for VE; 0.5 L/breath, CI=0.3-0.7, p<0.001 for TV; 3.8 breaths/min, CI=1.6-5.9, p<0.001 for RR). All measures returned to and remained at baseline readings at 10, 30 and 60 minute comparisons. The 30 minute PETCO2 measure was different than baseline when not adjusting for multiple comparisons (decrease -1.1 mm Hg, CI=-2.1, -0.2, p=0.025), but it was no longer significant after adjustment (p>0.013). PETCO2 readings were not different at 1, 10 or 60 minutes compared to baseline. There was no evidence of hypoxemia or hypoventilation.

Effects on Blood Parameters

The effects of TASER exposure blood parameters are reported in Table 3. For arterialized capillary blood measures, there were significant differences for pH (p=0.021), bicarbonate (p<0.001) and lactate concentration (p<0.001) in the repeated measures ANOVA analysis (Figure 3). There was an initial decrease in pH at 1 minute (-0.02, CI=-0.04, -0.01, p=0.001), but levels retuned to normal at 10, 30 and 60 minutes. Bicarbonate levels were lower at 1 and 10 minutes compared to baseline (-1.2 mEq/L, CI=-1.8, -0.7, p<0.001 at 1 minute; -1.0 mEq/L, CI=-1.6, -0.4, p=0.002 at 10 minutes), but returned to baseline levels at 30 and 60 minutes. Lactate concentration levels were higher at 1 minute (1.4 mmol/L, CI=1.1, 1.6, p<0.001) and 10 minutes (1.0 mmol/L, CI=0.7, 1.2, p<0.001) compared to baseline, but returned to baseline levels at 30 and 60 minutes. For venous blood measures, there were no significant differences between measures for Ca2+, Na+ or K+ based on repeated measures ANOVA results. None of the blood measure changes that did occur were clinically significant. Troponin I values for all subjects at six hours were <0.2ng/ml, with a positive assay defined as >0.2ng/ml.

Effects on 12 lead ECG

All 32 subjects had no evidence of ischemia noted on ECG and when blinded and compared, there was no evidence of interval changes from baseline to after TASER exposure.

Phase 2

Preliminary Results (results from the final analysis are forthcoming)

A total of 22 Sheriff's officers volunteered to participate in the study and have been analyzed to date. Both Taser and Control groups changed similarly over each measure. Post exercise measures were: pH=7.31, (Taser) 7.29 (Control); HCO3=20.4 (Taser), 19.6 (Control) and Lactate= 8.6 (Taser), 8.5 (Control). From post exercise measures, pH increased 0.10, 95% CI 0.07,0.13 in the Taser group and 0.10, 95% CI 0.07, 0.13 in Control group. HCO3 increased 4.1, 95% CI 2.9,5.2 in the Taser group and 4.8, 95% CI 3.1,6.5 in the Control group. Lactate decreased 6.8, 95% CI 5.3,8.3 in the Taser group and 6.8, 95% CI 5.3,8.3 in the Control group. There were no clinically significant differences of any measurements between the Taser Group and Control Group.

LIMITATIONS

There are several limitations to our study. Our subjects were generally healthy and free from chronic disease and duration of the TASER activation in our study did not exceed a single five second activation, whereas individuals in the field often receive multiple shocks. Our subjects were also not under the influence of illicit stimulant drugs, or in a state of agitated delirium.

ANALYSIS AND DISCUSSION

The TASER delivers energy through a sequence of dampened sine-wave current pulses each lasting about 11 μs. This energy is reportedly neither pure AC nor pure DC, but probably akin to rapid-fire, low amplitude DC shocks. (2) The power output of the device is 26 W, average 2 mA current and a maximum of 50,000 V, which is reported to be below the threshold of ventricular fibrillation.(1) Studies directly stimulating canine hearts using the TASER failed to induce cardiac arrhythmia.(13,14) There is also an industry-sponsored swine model study lauding the cardiac safety of the newer TASER. (15)

Effect of electrical injury on the cardiac conducting system has been studied in both prospective animal studies and retrospective human studies.(16-21) The pulse duration and amplitude of electricity in these cases is different than that of the TASER, that is, the majority of data available about electrocutions is regarding people and animals subjected to very different doses of electricity, such as from lightning or power lines.

Since the funding initiation of this study and there have been several published studies that have prospectively evaluated the effects of the TASER on humans (22-29). Our study is unique in that it was non-industry funded and looked specifically at physiologic metabolic blood and ventilatory parameters in humans prospectively, both without and following exercise.

We found no changes in electrolytes in the 60 minutes of observation in either with or without exercise. Additionally, all of our six hour troponin I levels were normal in phase one and there were no changes in ECG from baseline compared with the ECGs taken one hour post-exposure.

In phase 1, with subjects at rest, we noted a modest increase in respiratory rate and tidal volume, which resulted in increased minute ventilation immediately after the TASER exposure,

but this increase was transient and returned to baseline by 10 minutes. In phase two, preliminary results indicate no significant difference between the control and taser group in measures before and following exercise. In monitoring breath-by-breath ventilation during the TASER activation, all subjects were noted to continue breathing during the exposure. Arterialized capillary sampling of pO2 and PCO2 demonstrated no evidence of hypoxia or CO2 retention during or after the TASER exposure demonstrating no ventilatory impairment secondary to the TASER exposure. Although statistically significant changes in pH were noted in phase 1, the mean pH remained between 7.42 and 7.45. The changes that were noted in pH were clinically insignificant and of are a degree found in mild to moderate exercise.

CONCLUSIONS:

In summary, this work in humans demonstrates no changes in ventilation, acid-base status, electrolyte concentrations (Ca^{2+},Na^{+}, K^{+}), troponin I, or ECG's of a clinically relevant nature. We conclude that a 5 second exposure of a TASER X-26 to healthy subjects at rest or following exercise does not result in clinically significant changes in ventilatory or blood parameters of physiologic stress. This two phase study offers a foundation for the understanding of the effects of a single Taser activation in humans.

REFERENCES

1. TASER International, TASER® promotional literature, (http://www.TASER.com) accessed 9/30/06.
2. Bleetman A, Steyn R, Lee C. Introduction of the TASER into British policing. Implications for UK emergency departments: an overview of electronic weaponry. *Emerg Med J.* 2004;21:136-140.
3. Kornblum RN, Reddy SK. Effects of the TASER in fatalities involving police confrontation. *J Foren Sci.* 1991;36(2):434-448.
4. O'Brien DJ: Electronic weaponry – a question of safety. *Ann Emer Med* 1991;20(5):163-167
5. Amnesty International Website (http://www.amnesty.org/) accessed 3/31/06.
6. Arizona Republic Website (http://www.azcentral.com/) accessed 6/30/06.
7. Koscove EM. The TASER® weapon: a new emergency medicine problem. *Ann Emer Med.* 1985;14(12):109-112.
8. Ordog GJ, Wasserberger J, Schlater T, Balasubramanium S. Electronic gun (TASER®) injuries. *Ann Emer Med.* 1987;16(1):103-108.
9. Allen TB. Discussion of effect of the TASER in fatalities in fatalities involving police confrontation. *J Forensic Sci.* 1991;36:434-438.
10. Kosove EM. The TASER®: research, patients and language (Tom Swift found). *J Emer Med*. 1988;6:343-344.
11. Stratton SJ, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden death of individuals requiring restraint for excited delirium. *Am J Emer Med.* 2001;19:187-19.

12. Presentation: Critical Issues in Policing Series, Police Executive Research Forum, San Diego, California; December, 2005.

13. Panescu D, Webster JG, Stratbucker RA. A nonlinear electrica-thermal model of the skin. *IEEE Trans Biomed Eng.* 1994;41:672.

14. Panescu D, Webster JG, Stratbucker RA. A nonlinear finite element model of the electrode-electrolyte-skin system. *IEEE Trans Biomed Eng.* 1994;41:681.

15. Lakkireddy D, Wallick D, Ryschon K, et al. Effects of cocaine intoxication on the threshold for stun gun induction of ventricular fibrillation. *JACC.* 2006;48(4):805-11.

16. Arya KR, Taori GK, Khanna SS. Electrocardiographic manifestations following electrical injury. *Intl J Cardiol.* 1996;57:100-101.

17. Chandra NC, Siu CO, Munster AM. Clinical predictors of myocardial damage after high voltage electrical injury. *Crit Care Med.* 1990;18(3):293-297.

18. Forrest FC, Saunders PR, McSwinney M, Tooley MA. Cardiac injury and electrocution. *J Roy Soc Med.* 1992; 85:642-643.

19. Jain S, Bandi V. Electrical and lightning injuries. *Crit Care Clin*. 1999;15(2):319-331.

20. Robinson NKM, Chamberlin DA: Electrical injury to the heart may cause long-term damage to conducting tissue: a hypothesis and review of the literature. *Intl J Cardiol.* 1996;53:273-277.

21. VanDenburg S, McKormick GM, Young DB. Investigation of deaths related to electrical injury. *Southern Med J.* 1996;89(9):869-872.

22. Barnes DG, Winslow JE III, Alson RL, et al. Cardiac effects of the TASER conducted energy weapon *Ann Emerg Med*. 2006; 48(4 Supp 1):S102.

23. Ho JD, Miner JR, Lakireddy DR, Bultman LL, Heegaard WG. Cardiovascular and physiologic effects of conducted electrical weapon discharge in resting adults. *Acad Emerg Med*. 2006;13(6):589-95.

24. Vilke GM, Sloane C, Levine S, Neuman T, Castillo E, Chan TC. Twelve-lead electrocardiogram monitoring of subjects before and after voluntary exposure to the Taser X26.Am J Emerg Med. 2008;26(1):1-4.

25. Levine SD, Sloane CM, Chan TC, Dunford JV, Vilke GM. Cardiac monitoring of human subjects exposed to the taser. J Emerg Med. 2007;33(2):113-7.

26. Vilke GM, Sloane CM, Bouton KD, Kolkhorst FW, Levine SD, Neuman TS, Castillo EM, Chan T Physiological Effects of a Conducted Electrical Weapon on Human Subjects. Ann Emerg Med. 2007:.50(5):569-75.

27. Sloane CM, Chan TC, Levine SD, Dunford JV, Neuman T, Vilke GM. Serum Troponin I Measurement of Subjects Exposed to the Taser X-26(R). J Emerg Med. 2008 Feb 23; [Epub ahead of print]

28. Dawes DM, Ho JD, Johnson MA, Lundin E, Janchar TA, Miner JR.15-Second conducted electrical weapon exposure does not cause core temperature elevation in non-environmentally stressed resting adults. Forensic Sci Int. 2008 Apr 7;176(2-3):253-7.

29. Ho JD, Dawes DM, Bultman LL, Thacker JL, Skinner LD, Bahr JM, Johnson MA, Miner JR. Respiratory effect of prolonged electrical weapon application on human volunteers. Acad Emerg Med. 2007 Mar;14(3):197-201.

Table 1. Phase 1 subject characteristics (n = 32)

Characteristic	**Mean ± SD**	**Range**
Age (yr)	38.4 ± 7.7	25 – 57
Weight (kg)	89.3 ± 15.0	65.8 – 125.2
Height (m)	1.79 ± 0.08	1.65 – 1.96
Body mass index (weight/height^2)*	27.8 ± 3.3	22.4 – 34.6

* Normal values for BMI = 18.5-24.9

Table 2. Phase 1 effects of TASER exposure on respiratory and ventilatory function (n=32)

	Baseline†	**1-Min**	**10-Min**	**30-Min**	**60-Min**
Measure*	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**
VE (L/min)‡	16.0 (3.7)	28.8 (10.5)	17.9 (4.0)	15.2 (5.3)	14.9 (4.3)
TV (breaths/L)‡	0.9 (0.2)	1.4 (0.7)	0.9 (0.2)	0.9 (0.3)	0.8 (0.3)
RR (breaths/min)‡	19.3 (4.4)	23.1 (5.7)	20.2 (4.6)	18.6 (4.4)	19.6 (4.5)
PETCO2 (mm Hg)‡	33.5 (3.1)	34.5 (4.4)	32.9 (3.3)	32.4 (2.6)	32.7 (2.5)
pO2 (mm Hg)	73.2 (4.8)	75.3 (7.2)	72.7 (6.9)	75.4 (9.8)	74.2 (8.1)
pCO2 (mm Hg)	35.8 (2.8)	35.9 (2.6)	35.0 (3.0)	36.1 (3.3)	36.0 (2.7)

Note: Individual measures missing for VE (n=4), TV (n=4), RR (n=4), PETCO2 (n=4), pCO2 (n=9), pO2 (n=9).

*Normal values: VE (4-7.5 lpm), TV (500 ml), RR (8-15 bpm), PETCO2 (35-45 mmHg), pO2 (80-100 mmHg) pCO2 (35-45 mmHg). VE and TV vary based on gender, size, tobacco use, physical fitness.

†Baseline values were obtained within 5 minutes prior to TASER exposure.

‡Repeated Measures ANOVA <0.05.

Table 3. Phase 1 effects of TASER exposure on blood parameters (n=32)

Measure*	Baseline† Mean (SD)	1-Min Mean (SD)	10-Min Mean (SD)	30-Min Mean (SD)	60-Min Mean (SD)
pH‡	7.45 (0.0)	7.42 (0.0)	7.43 (0.0)	7.43 (0.0)	7.44 (0.0)
Bicarbonate (mEq/L)‡	23.9(2.2)	22.7(2.0)	22.9(1.8)	23.9(1.7)	23.8(1.6)
Lactate (mmol/L)‡	1.4 (0.5)	2.8 (0.7)	2.4 (0.6)	1.5 (0.5)	1.3 (0.5)
Ca2+ (mg/dl)	9.8 (0.4)	9.8 (0.4)	9.8 (0.4)	9.8 (0.4)	9.8 (0.4)
Na+ (mEq/L)	138.3 (3.8)	137.8 (3.9)	138.4 (4.2)	137.8 (4.0)	138.3 (3.9)
K+ (mEq/L)	4.2 (0.6)	4.1 (0.6)	4.2 (0.6)	4.2 (0.6)	4.2 (0.6)

Note: Individual measures missing for pH (n=8), pCO2 (n=9),, pO2 (n=9), bicarbonate (n=9) and lactate measures (n=9).

*Normal values: pH (7.35-7.45), Bicarbonate (20-29 mEq/L), Lactate (0.7-2.1 mmol/L), Ca2+ (8.6-10.3 mg/dl), Na+ (135-147 mEq/L), (K+ (3.5-5.0 mEq/L).

†Baseline values were obtained within 5 minutes prior to TASER exposure.

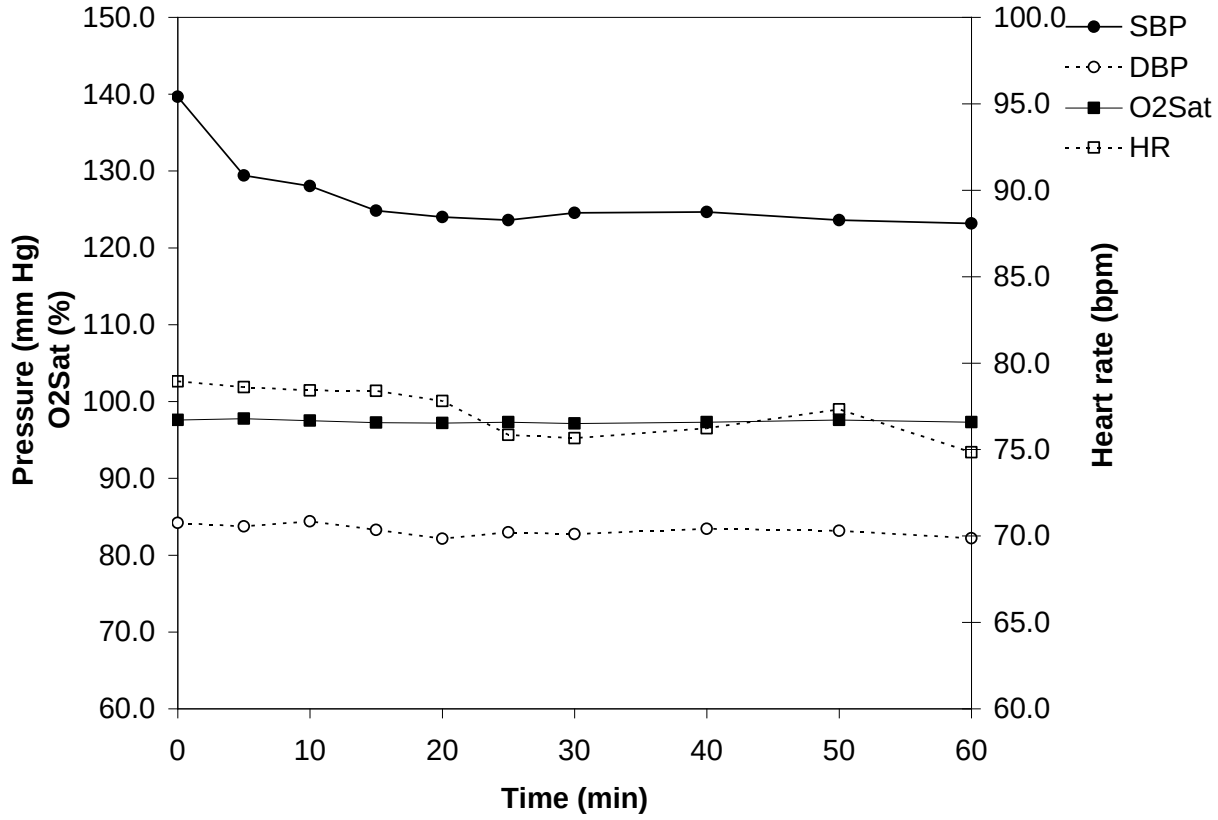
‡Repeated Measures ANOVA <0.05.

Table 4. Phase 2 preliminary results (n=22)

	Baseline	**Post-Exer**	**1-Min**	**10-Min**	**30-Min**	**60-Min**
	Mean (SD)	**Mean Difference (95% CI) from Post Exercise**				
pH						
Taser	7.42 (0.02)	7.31 (0.04)	-0.12 (-0.04,-0.01)	0.04* (0.00,0.07)	0.09* (0.05,0.13)	0.10* (0.07, 0.13)
Control	7.41 (0.02)	7.29 (0.05)	0.02* (0.00,0.04)	0.07* (0.06,0.09)	0.12* (0.9,0.14)	0.11* (0.09,0.14)
HCO3 (mEq·L-1)						
Taser	24.7 (1.1)	20.4 (1.6)	-3.8* (-5.0,-2.5)	-2.2* (-3.6,-0.7)	2.5* (0.9,4.1)	4.1* (2.9,5.2)
Control	24.5 (1.8)	19.6 (2.8)	-1.8* (-2.5,-1.0)	0.0 (-0.9,1.0)	4.1* (2.7,5.5)	4.8* (3.1,6.5)
Lactate (mmol·L-1)						
Taser	1.7 (0.6)	8.6 (2.1)	1.0 (-0.0,2.0)	-1.4* (-2.7,-0.0)	-5.4* (-6.8,-4.1)	-6.8* (-8.3,-5.3)
Control	1.2 (0.5)	8.5 (2.9)	0.1 (-0.8,1.0)	-2.9* (-3.8,-1.9)	-6.2* (-7.8,-4.7)	-7.3* (-9.1,-5.5)

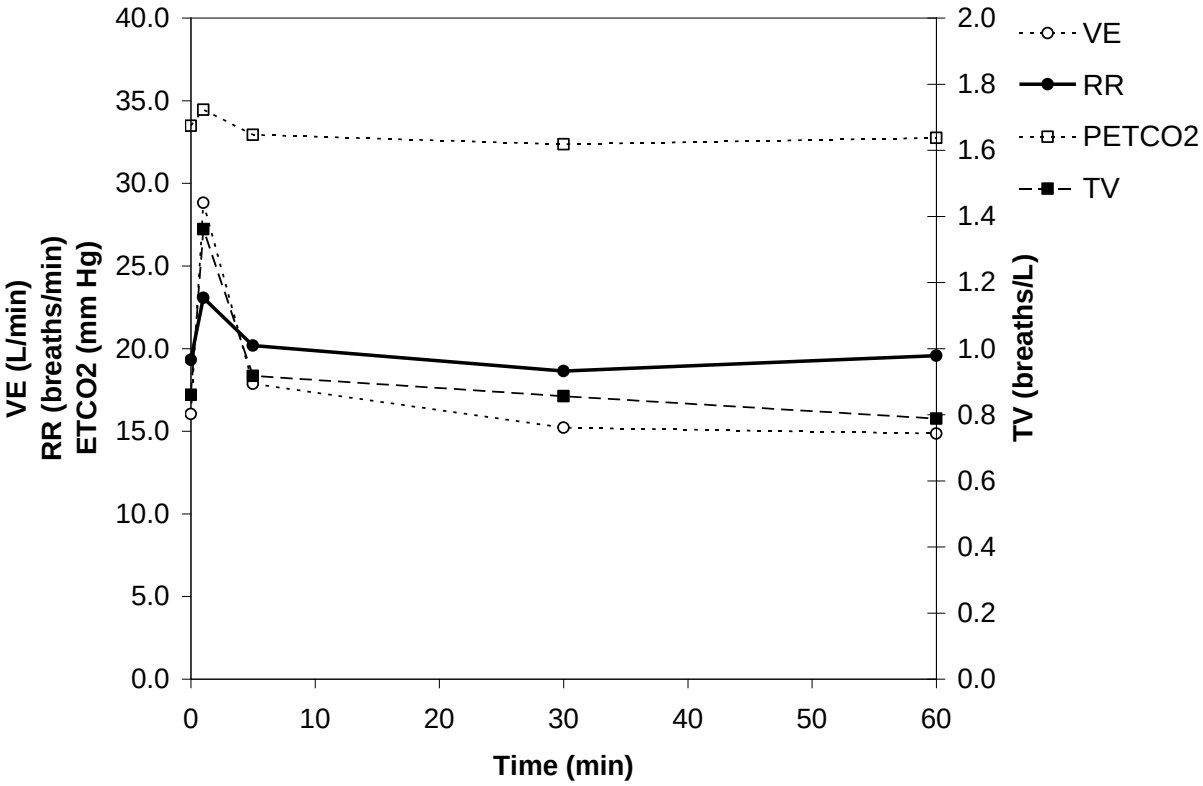
* <0.05 for comparisons from post exercise

Figure 1. Phase 1 effect of a 5-s TASER exposure on heart rate (HR), O2 Saturation (O2Sat) and systolic (SBP) and diastolic (DBP) blood pressures (n = 32).



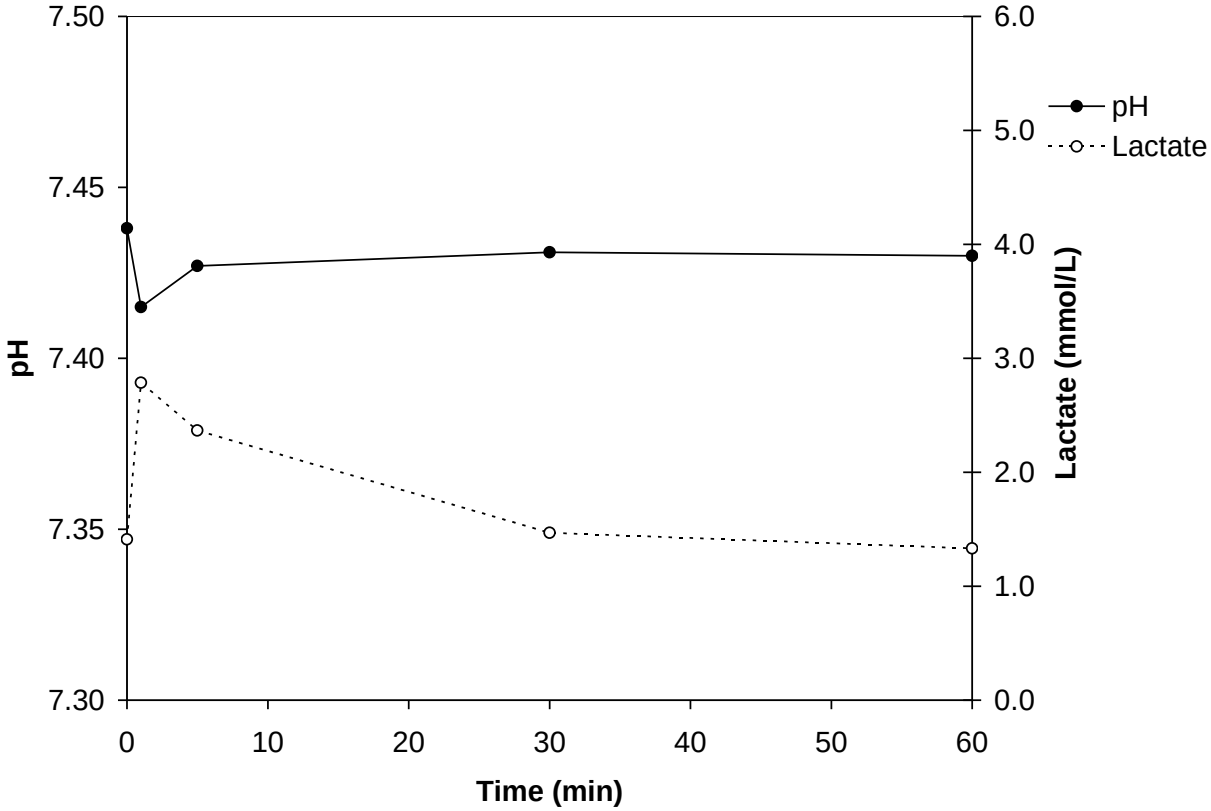
Note: Baseline levels (time=0) were obtained within 5 minutes prior to TASER exposure.

Figure 2. Phase 1 effect of a 5-s TASER exposure on minute ventilation (VE), respiratory rate (RR), tidal volume (TV) and end-tidal PCO2 (n = 32).



Note: Baseline levels (time=0) were obtained within 5 minutes prior to TASER exposure.

Figure 3. Phase 1 effect of a 5-s TASER exposure on blood pH and lactate concentration (n = 32).



Note: Baseline levels (time=0) were obtained within 5 minutes prior to TASER exposure.