Effects of Repeated Thoracic Discharges From a Stun Device

by

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Background: Very little objective laboratory data is available describing the physiological effects of stun guns or Electromuscular Incapacitation (EMI) devices, but increasing amounts of morbidity and even deaths are associated with their use. Most of the deaths have been attributed to specific cardiac and metabolic effects. We, therefore, hypothesized that the application of an EMI device in a model animal system would induce clinically significant metabolic acidosis or ventricular fibrillation.

Methods: Ten Yucatan mini-pigs, 6 experimental and 4 sham controls, were anesthetized using ketamine, xylazine, and glycopyrollate. All animals were intubated and ventilated. The experimental group was exposed to two 40 sec discharges from an EMI device (MK63, Aegis Indus.) over the left thorax. Ventilation was withheld during the 40 sec discharges, but ventilator support was given during the 10 sec time between discharges and otherwise during anesthesia. EKGs, troponin I (TnI), blood gases, and lactate levels were obtained pre-exposure, at 5, 15, 30 and 60 min and 24, 48 and 72 hr after EMI discharge. Cardiac biopsies were also obtained.

Results: *No evidence of acute arrhythmia or myocardial infarction was found.* Rhythm strips taken before, during, and after discharge of the MK63 over the thorax did not show acute arrhythmia at any time. CK-MB levels were not significantly affected by the discharge versus controls or experimental baseline values at any time. TnI values increased at the 24 hr time point in negative controls (mean ± SEM 0.023±0.019 ng/ml) and experimentals (0.040±0.031 ng/ml; see Figure 1). The observed increase in experimentals, however, was not statistically significant when compared controls ($p=0.695$, t-test). The mean value at 24 hrs for the experimental group (0.040±0.031 ng/ml) was not significantly increased from the experimental baseline value (0.000±0.000 ng/ml, t-test). A TnI value of 0.040 ng/mL is considered to be the upper limit of normal for this assay. The largest TnI value obtained for a single animal was 0.190 ng/ml at 24 hrs post-EMI discharge. This value returned to baseline at 48 hrs. Myocardial tissue biopsies revealed no evidence of injury or necrosis in any animal.

Clinically significant acidosis was not observed after EMI discharge. Central venous blood pH showed a small initial decrease after EMI discharge at the 5 min time point (see Figure 2). This shift from 7.45±0.03 to 7.39±0.02 was statistically significant ($p<0.05$) although the observed values were still within normal ranges. Central venous blood pH returned to baseline within 60 min. Control animals had a significantly higher pH during the entire 60 min time period. Acidosis was noted only in one control animal (pH=7.32) at the 5 min time interval. All other animals maintained blood pH at or above normal levels for the duration of the experiment.

Central venous pCO_2 was not significantly changed by EMI discharge. A small increase was seen at 5 min (39.2±2.8 mmHg) post-discharge but this was not significantly different ($p>0.05$) from the baseline value of 36.6±2.7 mmHg. The pCO_2 returned to baseline within 60 min. One animal had pCO_2 levels above the normal range (49.6 mmHg) at 5 min post-discharge. Levels for this animal returned to normal within 60 min.

Bicarbonate levels decreased at 5 min post-discharge (24.3±0.6 mmol/L) from baseline values (25.2±1.1 mmol/L), but this was not a significant change ($p>0.05$, t-test). This decrease contrasted with a small observed increase in negative controls. There was no significant difference when comparing controls to experimentals over the initial 60 min period ($p>0.05$,

Tukey's test). All bicarbonate levels were within the normal reference range. One experimental animal had a bicarbonate level of 20.6 mmol/L prior to EMI exposure. The bicarbonate level in this animal rose to normal levels following exposure.

Lactate values showed a small increase at the 5 min time point (4.89±0.7 mmol/L). This increase was not statistically significant ($p>0.05$) from baseline values (3.7±1.3 mmol/L). The experimental group showed significantly higher levels of lactate when compared to controls over the initial 60 min period ($p<0.05$, Tukey's test). Lactate was below baseline values at 24 hrs and was similar to controls. Two experimental animals had elevated baseline lactate levels (8.13 and 7.43 mmol/L). These were peak values for these animals which then decreased to normal levels over the initial 60 min. The other 4 experimental animals had normal starting lactate levels and showed slight increases after EMI exposure which then returned to baseline.

Discussion and Conclusions: The results of the present study are largely at variance with those of Jauchem et al. (Jauchem, J.R. et al. 2005. Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of *Sus scrofa* following repeated TASER exposures, Forensic Sci. Int., doi:10.1016/j.forsciint.2005.10.014) which examined the effects of discharges from a Taser X-26 in swine. In that study, severe acidosis ($pH<7.0$) was seen immediately after EMI exposure and this was accompanied by dramatic increases in pCO_2 (>100 mmHg) and lactate (>15 mmol/L). There are, however, numerous differences between the present study and that study. First, the Taser X-26 used by Jauchem et al. (2005) delivers pulses at a voltage of about 50 kV, a pulse duration of 140 µsec, a frequency of 15-19 Hz, and power of 0.36 J/pulse (www.taser.com). The MK63 device used here also delivers pulses but at a voltage >19 kV, with pulse durations of 100 µsec, at 65 Hz, and 0.08 J/pulse (C. Hathcock, personal communication). Second, in the present study the MK63 device was discharged for 40 sec, one 10 sec rest was allowed, and this was followed by another 40 sec discharge for a total of 80 sec of EMI exposure. Jauchem et al. (2005) administered discharges in 5 sec intervals with 5 sec rests for 3 min for a total of 90 sec of EMI exposure. Third, different anesthetic agents and methods of physiologic support were used. Jauchem et al. (2005) did not mechanically ventilate the intubated animals at all during study whereas the swine in the present study were ventilated while anesthetized except during the 80 sec EMI discharge. As a result, it is unclear what role respiratory depression from the propofol/ Telazol anesthesia and buprenorphine analgesia or the lack of respiratory support may have played in the observed acid-base abnormalities seen by Jauchem et al. (2005).

In summary, **no evidence** of acute arrhythmia, myocardial damage, severe acidosis, or electrolyte/biochemical abnormalities was seen in the present study. In this swine model, prolonged discharges from the MK63 device produced no significant or harmful physiologic changes. Since the previous animal study of the Taser X-26 showed some dramatic physiological changes (Jauchem, 2005), the present findings may be due to differences in the waveforms generated by the EMI devices studied, differences in the electrode spacing for the MK63 as compared to the X-26, or other differences in the model systems. Further studies are needed to distinguish among these possibilities, to evaluate the short- and long-term physiological effects of these devices, and to elucidate the mechanism by which these devices trigger EMI so that their biological and health effects can be more fully understood.

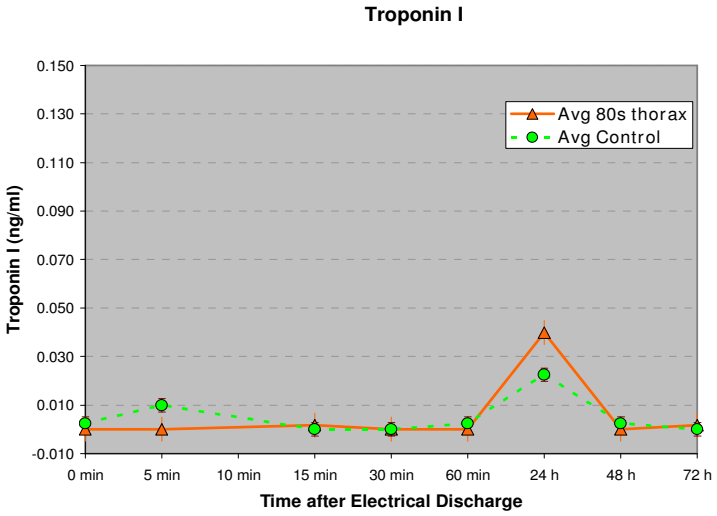

Figure 1. Troponin-I values during the 72 hr time course after EMI discharge. The mean TnI value reached the upper limit of normal (0.040 ng/ml) 24 hr post-EMI discharge. Two animals (n=6) in the experimental group showed TnI levels above the upper limit of normal (0.190 ng/ml and 0.050 ng/ml) at the 24 hr time point. For both animals, TnI returned to baseline (0.000 ng/ml) subsequently. The time-related pattern of TnI elevation in these animals was not like that seen in acute myocardial infarction where prolonged elevation of TnI is seen for days with a gradual return to baseline (Feng, Y. J., et al. Comparison of cardiac troponin I, creatine kinase-MB, and myoglobin for detection of acute ischemic myocardial injury in a swine model. Am.J.Clin.Pathol. 110 (1998): 70-77; Zipes et al. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 7th ed. Saunders: Philadelphia, 2005 1158-61). TnI also increased in one control animal at 24 hr (0.080 ng/ml) but values similarly returned to baseline subsequently. The pattern and magnitude of TnI increases seen here was very different from that seen in humans or swine with myocardial injury. It is therefore unlikely that the elevated TnI values seen here signify myocardial injury as seen in myocardial infarction or swine models of myocardial ischemia.

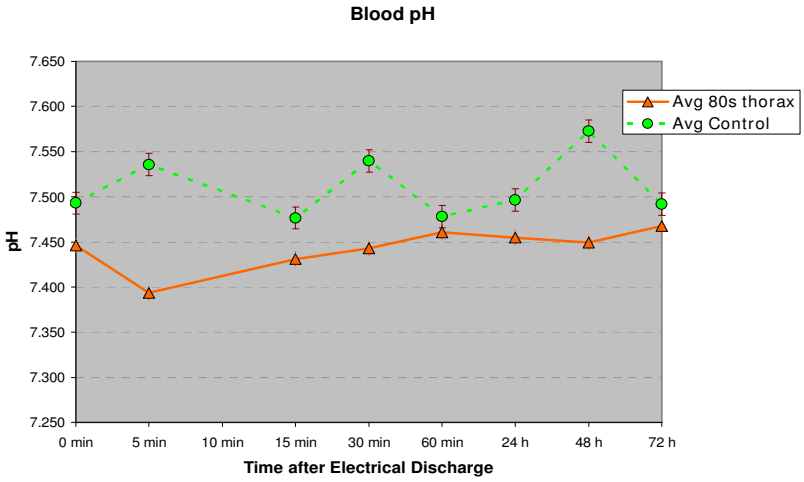

Figure 2. Central venous pH over time for control and experimental animals. It has been postulated that "in-custody deaths" may result from cardiac instability due to EMI-induced lactic acidosis (UNITED STATES OF AMERICA Excessive and lethal force? Amnesty International's concerns about deaths and ill-treatment involving police use of tasers, http://web.amnesty.org/library/index/engamr511392004; Stratton, S.J. et al. Factors associated with sudden death of individuals requiring restraint for excited delirium. Am.J.Emerg.Med. 19 (2001): 187-91). No evidence of severe acidosis was seen in the present study, nor was clinically significant acidosis seen in any of the animals. Similarly, the changes in pCO_2, lactate, and bicarbonate showed only small, insignificant changes correlating with the observed changes in blood pH.