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Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of *Sus scrofa* following repeated TASER® exposures

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Abstract

Repeated exposure to electro-muscular incapacitating devices could result in repetitive, sustained muscle contraction, with little or no muscle recovery period. Therefore, rhabdomyolysis and other physiological responses, including acidosis, hyperkalaemia, and altered levels of muscle enzymes in the blood, would be likely to occur. Experiments were performed to investigate effects of repeated exposures of TASER® International's Advanced TASER® X26 on muscle contraction and resultant changes in blood factors in an anaesthetized swine model. A total of 10 animals were used. Six swine were exposed for 5 s, followed by a 5-s period of no exposure, repeatedly for 3 min. (In five of the animals, after a 1-h delay, a second 3-min exposure period was added.) The remaining four animals were used for an additional pilot study.

All four limbs of each animal exhibited contraction even though the electrodes were positioned in areas at some distances from the limbs. The degree of muscle contraction generated during the second exposure period was significantly lower than that in the first exposure series. This finding was consistent with previous studies showing that prolonged activity in skeletal muscle will eventually result in a decline of force production.

There were some similarities in blood sample changes in the current experiments with previous studies of muscular exercise. Thus problems concerning biological effects of repeated TASER exposures may be related, not directly to the ''electric output'' per se, but rather to the resulting contraction of muscles (and related interruption of respiration) and subsequent sequelae. Transient increases in hematocrit, potassium, and sodium were consistent with previous reports in the literature dealing with studies of muscle stimulation or exercise. It is doubtful that these short-term elevations would have any serious health consequences in a healthy individual. Blood pH was significantly decreased for 1 h following exposure, but subsequently returned toward a normal level. Leg muscle contractions and decreases in respiration each appeared to contribute to the acidosis. Lactate was highly elevated, with a slow return (time course greater than 1 h) to baseline.

Other investigators have reported profound metabolic acidosis during restraint-associated cardiac arrest. Since restraint often occurs immediately after TASER exposure, this issue should be considered in further development of deployment concepts. On the basis of the results of the current studies, the repeated use of electro-muscular incapacitating devices in a short period of time is, at least, feasible, with the caveat that some medical monitoring of subjects may be required (to observe factors such as lactate and acidosis).

Keywords: Blood; Electric injuries; Electronic weapons; Muscle; Acidosis; TASER

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1. Introduction

There is a need in both military forces and civilian law-enforcement agencies for non-lethal weapons that can quickly and effectively incapacitate hostile adversaries without causing permanent injury. TASER® (Thomas A. Swift's Electronic Rifle) is the registered trademark of a battery-powered device that generates electric pulses designed to accomplish such a mission. A number of deaths after exposure to such weapons have recently been reported in the popular media, with much interest in these events generated within the forensic science community. (For comprehensive review of TASER effects, see Bleetman et al. [1].) Disagreements have occurred among forensic pathologists [2] regarding the potential lethality of exposure to the TASER.

Several reports of TASER use by law-enforcement personnel have involved repeated shots to a single individual in a short period of time. Effects of the TASER could result either (a) directly from the electrical properties of the applied stimulus or (b) from the resultant muscle contraction. Although such contraction is not synonymous with conventional muscular exercise, there may be some similarities. Rhabdomyolysis (breakdown of muscle tissue to the extent that contents are liberated into the circulation) may be caused by either excessive muscular activity [3] or electrical injury [4]. One may expect any possible electrical injury due to TASER exposure, however, to be quite different from conventional electrical injury (due to, e.g., 60 Hz alternating current) since electrical properties (most importantly, current flow) of the two modalities are dissimilar.

Repeated exposure to electro-muscular incapacitating devices, such as the TASER, could result in repeated, sustained muscle contraction, with little or no muscle recovery period. Therefore, rhabdomyolysis and other physiological responses, including acidosis, hyperkalaemia, and altered levels of muscle enzymes in the blood, would be likely to occur.

The current study was not intended to fully address the suitability of employment of electro-muscular incapacitating devices, but rather to obtain initial data on effectiveness and any immediate health effects. These experiments were performed to investigate effects of repeated exposures of TASER International's Advanced TASER X26 on muscle contraction and resultant changes in blood factors in an anaesthetized swine model. This was, to our knowledge, the first study of such factors after any type of TASER exposure.

2. Material and methods

2.1. Animal model

Ten domestic swine (*Sus scrofa domestica*) with a mean weight of 53.6 kg (range 49.5–58.0 kg) were used for these studies. The chemical and physical characteristics of human and swine blood are very similar, with the exception of lower values for hematocrit, mixed venous oxygen content, and oxygen saturation [5]. Respiratory parameters of swine resemble those of humans [5]. In addition, the pig's responses to muscular exercise are similar to those in humans [6].

Obviously, changes in conscious pigs could be different from those observed in anaesthetized animals. To measure muscle contraction and facilitate repeated blood sampling, however, the anaesthetized preparation was considered to be more appropriate for these experiments.

2.2. Anaesthesia and experimental set-up

All experiments and animal care procedures were approved by the Institutional Animal Care and Use Committee of Air Force Research Laboratory, Brooks City-Base, Texas, USA, and were conducted according to the US National Institutes of Health's ''Guide for the Care and Use of Laboratory Animals'', prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources – National Research Council.

For each experiment, a swine was given pre-anaesthetic (atropine 0.05–0.5 mg kg^{-1} body weight, subcutaneously) and analgesic (buprenorphine, 0.02 mg kg^{-1} body weight, intramuscularly) 10–15 min prior to induction of anaesthesia. The animals were anaesthetized with an intramuscular injection of tiletamine HCl and zolazepam HCl (Telazol®) (6 mg kg^{-1}), followed by oral endotracheal intubation, with the tube secured to the maxilla or mandible. An aural intravenous catheter (3/4–1 in 20–22 ga.) was placed and secured with a cyanoacrylate adhesive and tape. Anaesthesia was maintained with 100–125 μg kg^{-1} min^{-1} of propofol (Diprivan®) delivered by a Baxter syringe pump. Compared with other anaesthetic regimens, total intravenous anaesthesia with propofol is less likely to cause cardiovascular [7] or pulmonary [8] dysfunction, and less decrease in arterial or mixed venous oxygen partial pressure [9], in swine. Inhalation anaesthetics such as halothane may not have been appropriate for these studies, since muscle-cell damage (as indicated by increased creatine phosphokinase [CPK]) can result from such use [10]. Depth of anaesthesia was verified by nasal septum pinch, coronary band hoof pressure, and jaw tone. Absence of both reflexes and lack of jaw tone were taken to indicate the animal was at a suitable anaesthesia plane. A jugular venous catheter was placed for subsequent blood sampling. Each pig was delivered to the laboratory anaesthetized, placed on its dorsal surface in a canvas sling. At the conclusion of the day's experiment, each animal was euthanized with pentobarbital sodium (Nembutal®), 100 mg kg^{-1} intravenously, without regaining consciousness.

The muscle contraction test structure included a framework constructed of Unistrut® metal framing system (Unistrut Construction, Wayne, Michigan, USA). A sling (to

contain the swine), pulleys, strain gauges (Model SSM-HA-150, Interface Inc., Scottsdale, Arizona, USA), and 3/8″ by 16″ zinc eye-to-eye turnbuckles (Crown Bolts Co., Cerritos, California, USA) were mounted on the system. Each anaesthetized swine was placed on its dorsal surface in the sling. Twisted polypropylene truck rope (3/8″ diameter, Model 87054, Wellington, Madison, Georgia, USA) was attached to each limb via a neoprene tennis elbow support (Wal-Mart Stores, Inc., Bentonville, Arkansas, USA), while the other end of the rope was attached to a turnbuckle and strain gauge. A second set of ropes was attached to each limb with neoprene-blend adjustable wrist/elbow supports (Model 483746, BD Consumer Healthcare, Franklin Lakes, New Jersey, USA). Each of these latter ropes ran through a 4″ diameter sheave block (Tuf-Tug Products & Accessories, Model SB3000FM, Moraine, Ohio, USA) and were attached to a 2.27-kg (5 lbs) mass. The output of the strain gauges was quantified, displayed, and stored using equipment and software made by DATAQ Instruments, Inc. (Model DI-720-USB data acquisition system and Version 2.67 WinDaq/Pro+ software, Akron, Ohio, USA). Prior to each exposure, the turnbuckles were adjusted to bring the swine's limbs to a standardized anatomical position (stretched maximally), with a baseline force of approximately 44.5 N (10 lbs).

The skin was pierced with standard TASER darts (TASER International, Scottsdale, Arizona, USA). One dart was placed approximately 5 cm to the right of the midline (approximately 13 cm cranially from the xiphoid process); the other was approximately 7 cm left of the umbilicus (resulting in approximately 30 cm separation between darts diagonally).

2.3. TASER exposures, physiological measurements, and blood sampling

Six swine were exposed to the output of the TASER International Advanced TASER X26 for 5 s, followed by a 5-s period of no exposure, repeatedly for 3 min. In five of the animals, after a 1-h delay, a second 3-min exposure period (again, of 5 s on, 5 s off) was added.

The force of the muscle contraction was measured and recorded (as described above). Heart rate, respiration rate, and pulse-oximeter oxygen saturation (SpO_2) were monitored continuously using a pulse oximeter (VetOx® G2 Digital, Heska Corporation, Fort Collins, Colorado, USA), with the probe placed on the ear.

According to Adrogué et al. [11], hypercapnia and acidemia at the level of the tissues are better detected in central venous blood than arterial blood. In addition, venous blood pH (a measure of the concentration of hydrogen ions in solution) and carbon dioxide partial pressure (pCO_2) are usually similar to arterial values [12]. Thus jugular venous blood samples were drawn for measuring pCO_2 and pH. In addition, oxygen partial pressure (pO_2), lactate, glucose, hematocrit, sodium, potassium, and calcium were measured before and after TASER exposures. (At physiological pH, lactic acid will almost completely dissociate to lactate and hydrogen ions. For this reason, the terms lactate and lactic acid are regularly used synonymously [13].)

Venous blood samples (3 cc each) were taken from the jugular vein within 1 min before and 1 min after each TASER exposure, and at other time points, for measurement of whole blood factors listed above. An additional 9 cc of blood was drawn and allowed to clot at room temperature for at least 30 min. Within 90 min of collection, the samples were centrifuged, and serum was refrigerated until assay. Serum troponin, CPK, and lactate dehydrogenase (LDH) (including isoenzyme forms) were used to provide qualitative estimates of skeletal (or cardiac) muscle damage that might occur as a result of TASER exposure. Although levels of these factors are often analyzed in clinical settings after many hrs or even days, increases may be detected earlier in some situations (e.g., CPK exhibits a peak 2 h post-exercise in rats [14].) Although peak levels of CPK after electrical injury to muscles are expected to occur after 24 h, Brumback et al. [15] hypothesized that CPK values within a few hrs after an injury can indicate the severity of any muscle fiber destruction. Increased levels of cardiac troponins T and I (each of which can reflect cardiac damage) can be detected in pigs within 30 min of coronary ischemia [16].

2.4. Analysis of blood samples

Levels of whole blood factors were measured with a Model GEM Premier 3000 blood gas/electrolyte analyzer (Instrumentation Laboratory, Lexington, Massachusetts, USA). Levels of myoglobin, CPK, and LDH in serum samples were evaluated by AniLytics Incorporated (Gaithersburg, Maryland, USA). Electrochemiluminescent immunoassays and an ORIGEN analyzer (IGEN International, Inc.; Gaithersburg, Maryland, USA) were used by AniLytics Incorporated to analyze cardiac troponins T and I. Streptavidin-coated beads, incubated with a mixture of sample, biotinylated anti-troponin, and ruthenium labeled anti-troponin antibodies, form the basis of these sensitive assays, suitable for detection of the troponins from different animal species.

2.5. Statistical analyses

Data for (a) eight of the nine whole blood measures (glucose, pH, hematocrit, pCO_2, pO_2, sodium, potassium, and calcium) and (b) the serum measures of myoglobin, CPK, and LDH, were separately analyzed by two one-way repeated-measures analyses of variance (ANOVAs), in which measurement time was the repeated factor. Analyses only included those subjects receiving one or two 3-min exposure sequences (or "sessions"). In the first of the two ANOVAs, measurement times were pre-Session 1, immediate post-Session 1, 30-min post-Session 1, and 60-min post-Session 1. In the second ANOVA, measurement times included were pre-Session 1, immediate post-Session 2,

30-min post-Session 2, and 60-min post-Session 2. (Thus, in each analysis, the underlying intent was to contrast whole blood values following exposure sessions with those prior to both of the two sessions.) In cases where the assumption of compound symmetry was not met, the adjusted degrees of freedom used are those recommended by Greenhouse and Geisser [17]. Post hoc comparisons were made using the Dunnett approach (two-tailed) with the pre-Session 1 time point as the control.

Data for three other physiological measures (heart rate, respiration rate, and SpO_2) were analyzed in a manner similar to that for the whole and serum blood dependent variables. Values for each of the three measures were collected every 30 s, but adjacent values were collapsed across time in order to yield a single value per subject for each of the following time points: pre-Session 1, during the Session 1 exposure, immediate post-Session 1, 30-min post-Session 1, 60-min post-Session 1, during the Session 2 exposure, immediate post-Session 2, 30-min post-Session 2, and 60-min post-Session 2. These mean values were analyzed by two one-way repeated-measures ANOVAs, one per session with measurement time as the repeated factor, in a manner analogous to that employed for the whole blood and serum measures.

Because results for the whole-blood lactate and serum troponin I showed zero variance at some time points, those data were analyzed by a Friedman ANOVA and post hoc comparisons were conducted employing nonparametric analog of the Dunnett approach.

In all analyses the rejection level for statistical significance was set at $\alpha = 0.05$.

3. Results

3.1. Muscle contraction force

In most cases, after several repeated 5-s exposures, the positioning of the straps on the four limbs did not remain consistent. Thus, only the muscle-contraction measurements during the initial portion of the 3-min exposure periods were considered to be an accurate reflection of limb movement. For the five subjects receiving two 3-min exposure periods, the mean level of maximal limb flexion was 30.5 kg (67.3 lbs) for the first of the two exposures (measured by averaging maximal flexion values during the first second of exposure across all four limbs); the mean level of maximal limb flexion for the second session of TASER exposures was 21.6 kg (47.5 lbs). This decrease in limb flexion was statistically significant as measured by a paired t-test, $t(4) = 5.35$, $p = 0.006$.

Fig. 1 shows an example of limb contraction tracings obtained from one animal, when exposed to the X26. Each limb exhibited an initial series of clonic (alternating tensing and relaxing of the muscles) contractions, followed by subsequent periodic contractions at slightly lower levels of force. This pattern reflects the discharge of the TASER X26, with a change in pulse rate and power after approximately 2 s of discharge.

3.2. Whole blood sample changes

Although subjects were under the influence of propofol anaesthesia, all pre-exposure physiological parameter values

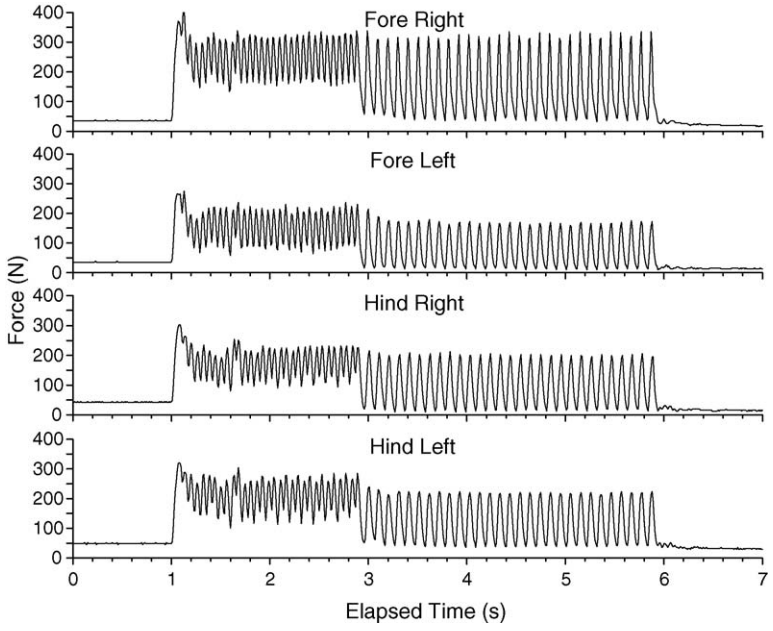

Fig. 1. Example of muscle contraction tracings obtained from one animal, when exposed to the TASER X26. The initial series of contractions, followed by subsequent periodic contractions at slightly lower levels of force, reflects the discharge of the X26, with a change in pulse rate and power after approximately 2 s of discharge.

(with the exception of venous pO_2 and respiratory rate) were similar to the ranges of values previously reported for conscious swine [5].

In this study, all blood-factor data are presented as mean ± standard errors of the mean. Levels of blood lactate and glucose during the experiments are shown in Fig. 2. A large number of the Session 2 TASER exposures resulted in lactate levels greater than the upper limit of measurement of the blood-gas/electrolyte analyzer (15 mmol/L). (The analyzer in these experiments was designed for clinical use, where values rarely exceed 15 mmol/L. The upper range of porcine lactate increase following TASER exposure was unknown before experimentation.) Measurements greater than 15 mmol/L were assigned a value of 15 for purposes of data analysis. Analyses for both lactate and glucose revealed significant effects for measurement time; for lactate: χ^2 ($N = 6$, df = 3) = 12.21, $p = 0.007$ for Session 1 and χ^2 ($N = 4$, df = 3) = 10.55, $p = 0.014$ for Session 2 (samples from only four subjects were analyzed); for glucose: $F(3, 15) = 4.63$, $p = 0.018$ for Session 1. On the basis of post hoc comparisons, lactate was significantly elevated at all post-exposure time points except at 30-min post-Session 2. Comparisons for glucose indicated a significant elevation 30 min following the first exposure, but no other differences.

Since lactate levels remained elevated at all time points after the initial exposure period, a pilot study of observations in four animals was included. One animal (''Subject 1'') was exposed as above, except with the initial ''exposure period'' lasting only one min rather than three min. In addition, single animals received either one 1-min exposure period (''Subject 2'') or one 3-min exposure period (''Subject 3''), with monitoring continued for 2 h afterwards in each case. Lastly, one animal (''Subject 4'') received a single 5-s exposure of the X26, with monitoring for 2 h. Results are presented in Fig. 3. Each animal exhibited increases in lactate following exposure (even with only a single exposure of 5 s). In each case, with the exception of the animal exposed to two sessions, lactate returned toward baseline levels after 2 h.

In Fig. 4, pH and hematocrit are illustrated. Analyses for pH revealed significant effects for measurement time; for pH: $F(1.12, 5.60) = 15.55$, $p = 0.008$ for Session 1 and $F(1.18, 4.74) = 25.42$, $p = 0.004$ for Session 2. The pH was significantly decreased at all post-exposure time points of both sessions (with the exception of 60-min post-Session 1). Distinctly lower values for hematocrit are common in pigs (compared to humans). The pre-exposure samples in the current experiments were within normal ranges as reported by Hannon et al. [5] in studies of conscious pigs. ANOVAs for hematocrit values showed significant effects for measurement time; $F(3, 15) = 10.33$, $p = 0.0006$ for Session 1 and $F(3, 12) = 8.64$, $p = 0.003$ for Session 2. These significant effects reflected increases in hematocrit (relative to pre-exposure)

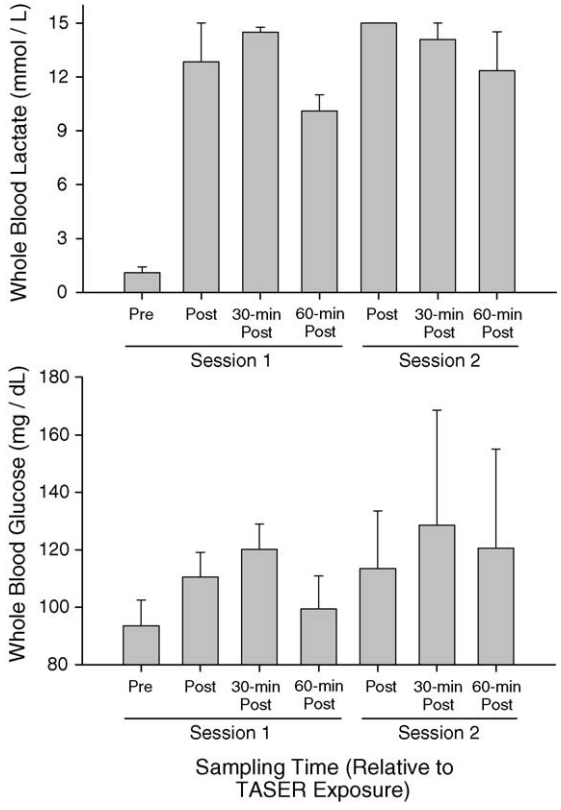

Fig. 2. Jugular venous blood lactate and glucose (mean ± S.E.M.) before, during, and after two sessions (for lactate, $N = 6$ for 1st session; $N = 4$ for 2nd session; for glucose, $N = 6$ for 1st session; $N = 5$ for 2nd session) of 18 TASER exposures each. Some Session 2 exposures resulted in lactate levels greater than the upper limit of measurement of the blood-gas/electrolyte analyzer (15 mmol/L). Measurements greater than 15 mmol/L were assigned a value of 15 for purposes of data analysis.

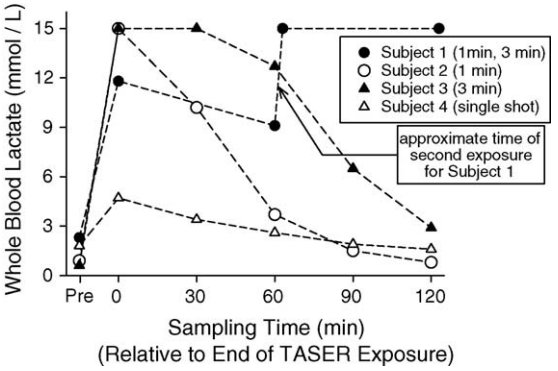

Fig. 3. Pilot study of observations of lactate in four additional animals. Subject 1 was exposed as in main study, except the initial exposure period lasted 1 min rather than 3 min. Subject 2 received one 1-min exposure period and Subject 3 received one 3-min exposure period, each with monitoring continued for 2 h afterwards. Subject 4 received a single 5-s exposure of the X26, with monitoring for 2 h.

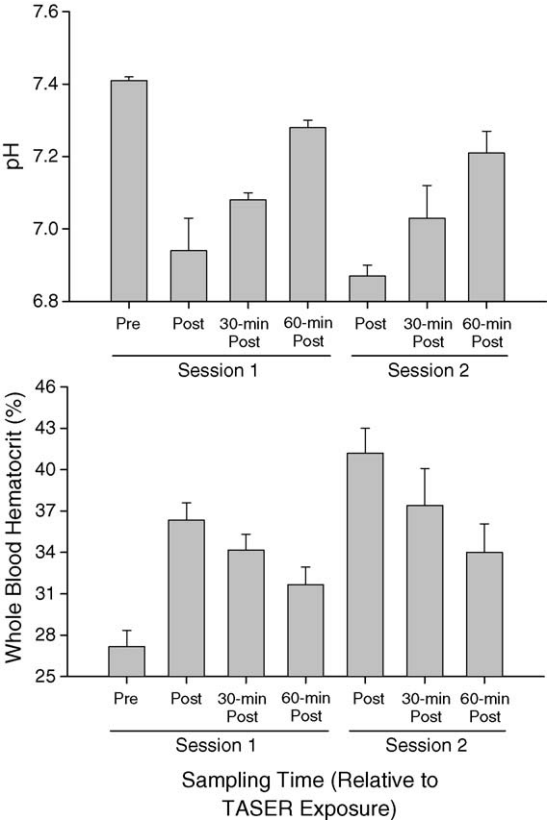

Fig. 4. Jugular venous blood pH and hematocrit (mean ± S.E.M.) before, during, and after two sessions ($N = 6$ for 1st session; $N = 5$ for 2nd session) of 18 TASER exposures each.

both immediately and 30 min after Session 1, and immediately after Session 2, with a return to baseline (pre-exposure) level at later time points within each exposure period.

Blood pCO_2 is shown in Fig. 5. ANOVAs for blood pCO_2 indicated a significant effect of measurement time for both sessions, $F(1.08, 5.42) = 18.93$, $p = 0.006$ for Session 1 and $F(3, 12) = 15.39$, $p = 0.0002$ for Session 2. These effects were the result of a significant elevation immediately after each exposure (relative to the baseline level) with a return toward baseline at 30 and 60 min following exposure. Blood pO_2 (not shown) was extremely variable. Due to this variability, no statistically significant changes were found.

Both potassium (Fig. 6) and sodium exhibited significant increases in levels after exposures; for sodium: $F(1.08, 5.38) = 10.96$, $p = 0.018$ for Session 1 and $F(3, 12) = 21.67$, $p = 0.00004$ for Session 2; for potassium: $F(1.25, 6.24) = 10.36$, $p = 0.015$ for Session 1 and $F(3, 12) = 7.86$, $p = 0.004$ for Session 2. For each of the two measures, values were significantly elevated immediately following each exposure but dropped back to pre-exposure levels within 30 min (with the exception of sodium 30 min post-Session 2). Blood

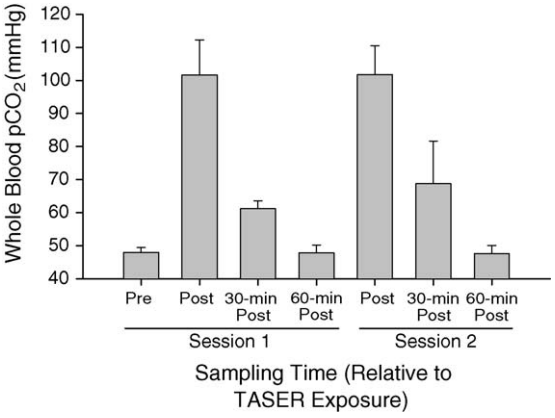

Fig. 5. Jugular venous blood pCO_2 (mean ± S.E.M.) before, during, and after two sessions ($N = 6$ for 1st session; $N = 5$ for 2nd session) of 18 TASER exposures each.

calcium showed a trend of increased levels after exposure, but changes were not statistically significant.

3.3. Serum sample changes

Serum myoglobin exhibited a trend toward increased levels after exposure; due to variability, however, there were no statistically significant changes.

Although the use of CPK to diagnose myocardial infarction and other cardiovascular problems often involves a longer time course than that of the current experiments, a modest, albeit non-significant, trend toward increased total CPK was seen after exposure. Likewise, there was trend toward increased levels of the CPK-MM fraction following the second exposure session. This increase, however, was not statistically significant.

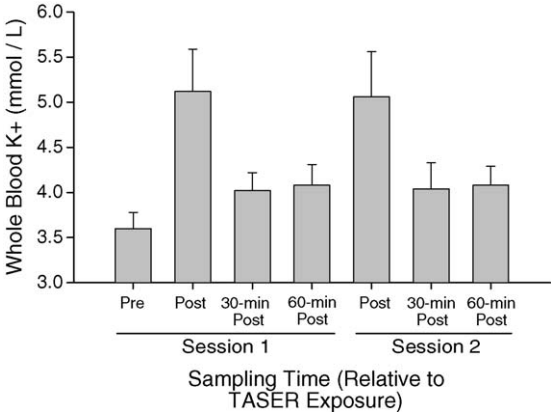

Fig. 6. Jugular venous blood potassium (mean ± S.E.M.) before, during, and after two sessions ($N = 6$ for 1st session; $N = 5$ for 2nd session) of 18 TASER exposures each.

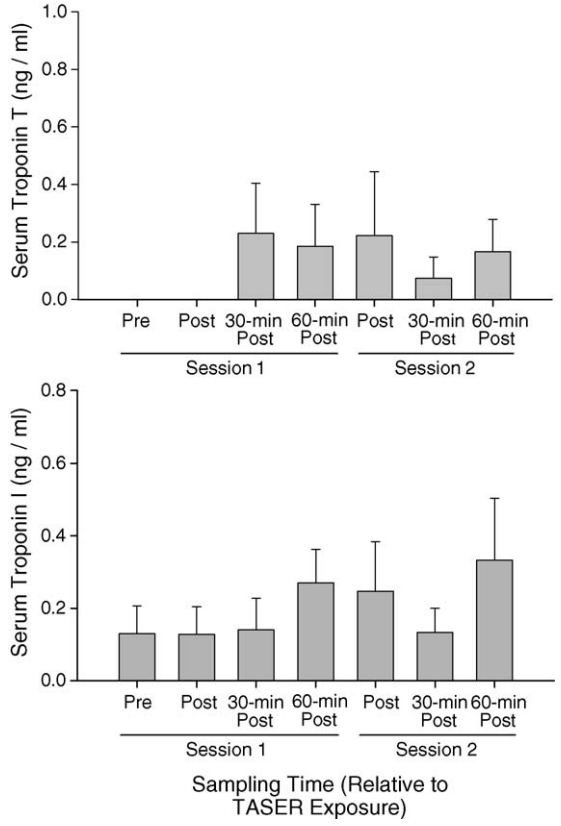

Fig. 7. Serum cardiac troponin T and serum cardiac troponin I (mean ± S.E.M.) before, during, and after two sessions ($N = 6$ for 1st session; $N = 5$ for 2nd session) of 18 TASER exposures each. The detection limit of the assay of swine cardiac troponin T in the present experiments has not been determined. Values listed as zero may simply be below detection limit.

There were no statistically significant changes in total LDH or LDH isoenzymes. There were no statistically significant changes in troponin T or troponin I (Fig. 7).

3.4. Heart rate, respiratory rate, and SpO_2

'As shown in Fig. 8, mean heart rate increased immediately following each exposure session, although this trend was only significant following the Session 1 exposure; $F(4, 20) = 7.67$, $p = 0.0006$. Heart rate returned to pre-exposure levels 30 min following exposure. SpO_2 decreased following each exposure (see Fig. 8); however, this change was only significant following Session 1; $F(4, 20) = 4.06$, $p = 0.01$. Values returned to pre-exposure levels within 30 min. Since the tested range listed for the VetOx G2 Digital pulse oximeter is 70–100%, reliability of measurement below 70% is unknown. Complete cessation of breathing was noted to occur during each 5-s TASER exposure. Mean respiration rate was decreased during and post-exposure, but this shift was not significant.

4. Discussion

4.1. Muscle contraction

In terms of muscular contraction effectiveness (amount of force generated), the current experimental results during the initial 5-s exposures were similar to previous results of exposure to an earlier TASER model (M26) (unpublished data). The degree of muscle contraction generated during Session 2 was significantly lower than in Session 1. This was not surprising, as prolonged activity in skeletal muscle will eventually result in a decline of force production [18].

It has been suggested that voluntary muscular control disappears in an elliptical area between the darts of TASERs during exposure [19]. In the present experiments, however, all four limbs exhibited contraction even though the electrodes were positioned in areas at some distances from the limbs.

There were some similarities in blood sample changes in the current experiments with previous studies of muscular exercise. Thus problems concerning safety of repeated TASER exposures may be related, not directly to the "electric output" per se, but rather to the resulting contraction of muscles (and related interruption of respiration) and subsequent sequelae.

4.2. Lactate and pH

The pH dropped from a normal value of 7.4 down to 7.0. According to some investigators, a pH of less than 7.2 indicates "severe acidemia" [20]. Those authors noted that such a blood pH could be of minor consequence if due to a transient or readily reversible condition. One hr after exposure in the current experiments, pH had returned to above 7.2. After a second TASER exposure session, pH showed a similar pattern. Both (a) muscle contractions and (b) changes in respiration (see below) may have contributed to the acidosis.

The degree of lactate increase was similar to that in other studies of pigs performing exhaustive exercise and of humans working for short periods at workloads of 1000 kg/min [6]. In addition, Thomas et al. [21] recently reported that peak venous blood lactate of untrained humans immediately after cycling maximally for one min was 13 mmol/L (identical to that of Session 1 of the current experiments). The rate of disappearance of blood lactate in the present study was similar to that found by Dotan et al. [22] in men during recovery after cycling which resulted in a lactate level of approximately 14 mmol/L. In the current study, in the single animal exposed to the TASER for only six cycles (i.e., 1 min), the slope of lactate recovery was similar to that of another study of humans after low-level exercise [23].

A well-defined relationship between pH and hyperlactatemia has not been defined. In fact, many investigators have reported a lack of significant association between acid–base status and blood lactate concentration [24]. Although there has

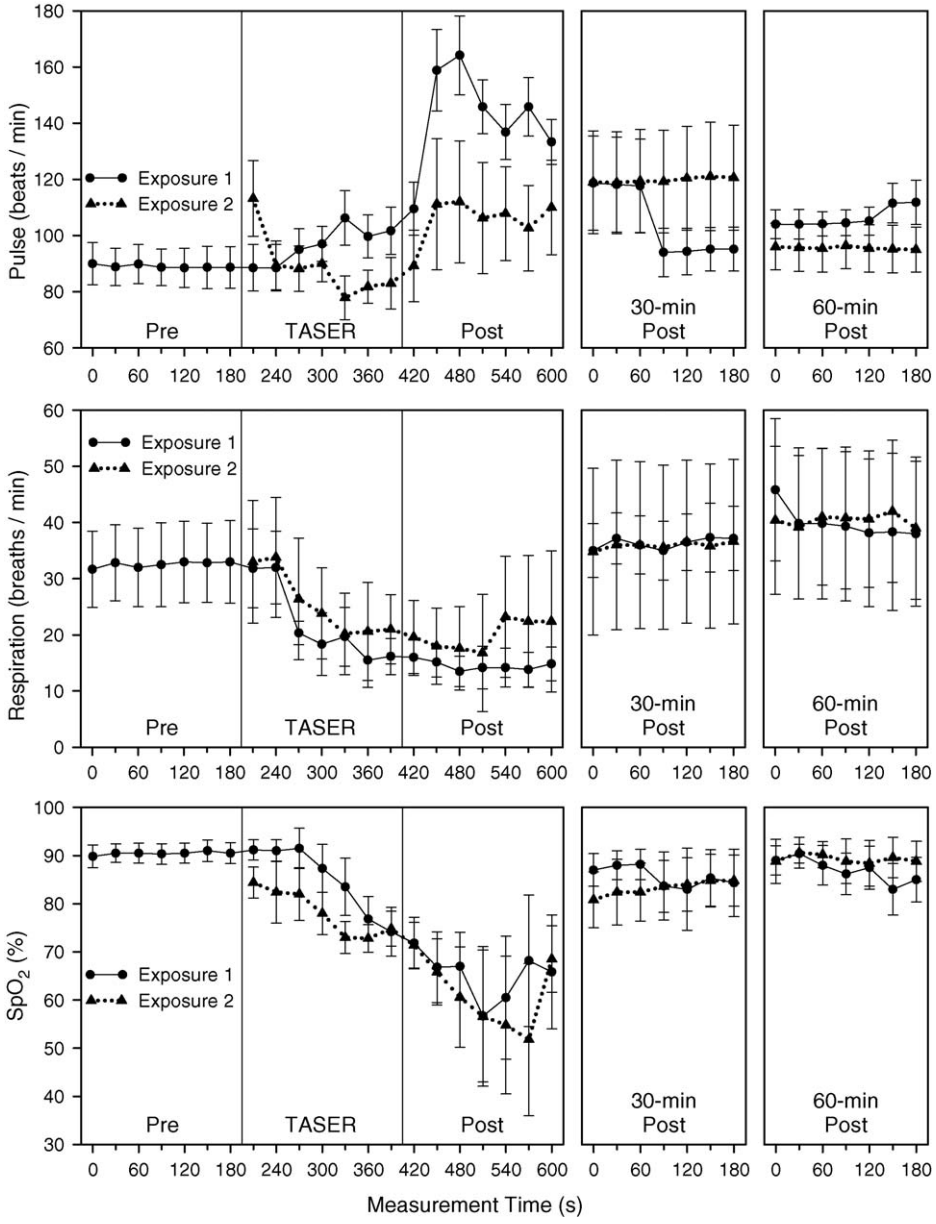

Fig. 8. Heart rate, respiration rate, and pulse-oximeter oxygen saturation (SpO_2) (mean ± S.E.M.) before, during, and after two sessions ($N = 6$ for 1st session; $N = 5$ for 2nd session) of 18 TASER exposures each. Values were collected every 30 s, but adjacent values were collapsed across time in order to yield a single value per subject for each of the "Pre", "TASER", and "Post" periods. Values of SpO_2 below 85% may have been artificially low due to inherent measurement properties of pulse oximetry.

been a traditional view that the lactate anion per se has no known harmful physiological effects [25], some authors have suggested that this opinion should be reconsidered [26]. High concentrations of both high hydrogen ions and lactate can cause declines in myocardial function independently of each other [27]. On the other hand, lactic acidosis can play a protective role against potential harmful effects of elevated potassium on muscle contraction force and excitability [28].

Acidosis can result in a lower threshold for ventricular fibrillation [20]. A full discussion of potential effects of TASER exposure on the heart is beyond the scope of this paper. In the present study, however, no ventricular fibrillation was seen.

Some of the factors referenced in the following sections are, in fact, affected by lactate levels and pH, but are mentioned separately for discussion purposes.

4.3. Respiratory rate, carbon dioxide, and oxygen

Pre-exposure respiratory rate was above the range reported by Hannon et al. [5] in conscious animals. Complete cessation of breathing, which occurred during each 5-s TASER exposure, was probably related to direct effects on muscles involved in respiration.

Since venous pCO_2 is similar to arterial values (except in frank cardiovascular shock [12]), the high blood pCO_2 values in the current experiments would be likely to reflect respiratory acidosis. Animals in the current experiments were not mechanically ventilated. The extremely low values of SpO_2 may have been artificially low due to inherent measurement properties of pulse oximetry. The accuracy of this technique can be diminished when saturation is less than 85% [29] and in situations of low tissue blood perfusion [30]. In addition, Yamaya et al. [31] reported that, in conditions of hypoxia and severe reductions of pH, further decrements in measured O_2 saturation may occur even in the absence of any true changes in pO_2. The tendency of pulse oximetry to underestimate true arterial O_2 saturation should be considered when speculating on the biological significance of the low values in the present study.

4.4. Hematocrit and electrolytes

The increase in hematocrit after exposure was consistent with previous studies of muscle stimulation [32]. The increases in blood sodium, potassium, and glucose during exposures, although statistically significant, were of relatively low magnitudes and returned close to pre-exposure levels within 30 min. It is doubtful that these short-term elevations would have any serious health consequences in a normal individual.

4.5. Myoglobin

Ordog et al. [33] reported mild rhabomyolysis (and myoglobinuria) in 1% of a series of patients who had been shot with a TASER device. Each of these patients, however, exhibited positive levels of phencyclidine (PCP). Thus, one could not determine whether muscle breakdown occurred due to PCP abuse or to muscle contractions related to the TASER exposures. In the current study, there were no significant changes in serum myoglobin.

4.6. CPK, LDH, and troponin

As in previous studies of effects of exercise on pigs [34], there were no significant changes in total CPK after TASER exposures. Although Bolter and Critz [32] reported increases in plasma LDH in dogs within the first 10 min of muscle stimulation, there were no significant changes in pigs after TASER exposures in the current study. This lack of change is consistent with the findings of Doize et al. [34] after exercise in pigs.

Serum levels of both cardiac troponin T and cardiac troponin I remained below 0.35 ng/mL. There were no statistically significant changes. Although some human cardiac troponin assays have been reported to have detection limits as low as 0.01 ng/mL [35], the limits of the swine assays used in the present experiments have not been determined.

To place the present study's values of swine troponin in perspective, one should note that Funahashi [36] and Wu et al. [37] reported mean cardiac troponin T levels of 1.20 and 0.85 ng/mL in pigs with various physiological insults versus control levels of 0.15 and 0.08 ng/mL, respectively. Wu et al. [38] found that cardiac troponin I increased from approximately 0.1 up to 2.0 ng/mL in pigs subjected to hypovolemic shock.

4.7. Heart rate

Pre-exposure heart rate was within ranges reported by Hannon et al. [5] and Hastings et al. [6] for restrained and unrestrained conscious pigs, respectively. Although heart rate increased significantly during the first TASER exposure period, the increase was much less than that seen in other studies of steady-state and exhaustive exercise [6].

4.8. Over-all relevance of results to possible TASER repeated-exposure scenarios

Other investigators have reported profound metabolic acidosis (pH as low as 6.25) during restraint-associated cardiac arrest [39]. Since restraint often occurs immediately after TASER exposure, this issue should be considered in further development of electro-muscular incapacitating-device deployment concepts.

It is important to note that, although the animals in the current study were anaesthetized, pre-exposure values of the physiological and biochemical parameters measured were similar to those observed by other investigators in conscious animals. Thus, in some respects, the results may be applicable to further future studies using a conscious animal model.

5. Conclusion

Experiments were performed to investigate effects of repeated exposures (for 3 min) of TASER International's Advanced TASER X26 on blood factors in anaesthetized swine. Transient increases in hematocrit, potassium, and sodium were consistent with previous reports in the literature dealing with studies of muscle stimulation or exercise. These increases, although statistically significant, were of relatively low magnitudes and returned close to pre-exposure levels within 30 min. It is doubtful that these short-term levels of elevation would have any serious health consequences in a normal individual. Blood pH was significantly decreased for

one hr following exposure, but subsequently returned toward a normal level. Muscle contractions and changes in respiration each appeared to contribute to the acidosis. Lactate was highly elevated, with a slow return toward baseline. Oxygen saturation (measured by pulse oximetry), although significantly decreased immediately after exposure, returned to pre-exposure levels within 30 min.

In conclusion, although 3 min of a TASER repeated-exposure scenario resulted in significant changes in blood chemistry, most levels (with the exception of lactate) returned to pre-exposure ranges within one hr after exposure (a 1-min scenario resulted in similar changes).

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