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TECHNICAL NOTE

PATHOLOGY/BIOLOGY

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Repetitive TASER X26 Discharge Resulted in Adverse Physiologic Events with a Dose–Response Relationship Related to the Duration of Discharge in Anesthetized Swine Model*

ABSTRACT: The objectives of our study were to investigate the dose–response relationship of the TASER X26 discharge duration in an anesthetized swine model. Fourteen swines were anesthetized and then exposed to TASER X26 discharge for 5 sec (n = 5) or for 10 sec (n = 6). The sham control group (n = 3) was anesthetized and studied using the same protocol except TASER X26 discharges during the experiments. Hemodynamic parameters were obtained. Blood pressure and total peripheral resistance decreased significantly after TASER discharge and returned to baseline value at 15 min after 5 sec of TASER discharge but did not return to baseline values during the 30-min observation period after 10 sec of TASER discharge. Repetitive TASER X26 discharge resulted in adverse physiologic events with a dose–response relationship related to the duration of TASER X26 discharge in an anesthetized swine model.

KEYWORDS: forensic science, TASER, conducted energy weapon, swine, hypotension, vasodilation, lactic acidosis

Conducted energy weapons (CEWs), such as the TASER X26, have been adopted by more than 11,000 law enforcement agencies throughout the world (1). TASER X26 (TASER International, Scottsdale, AZ) is a handheld CEW that fires two sharp darts, which are connected to the device by wires that also extend to the target. The device then delivers high-voltage, low-amperage electricity that induces forceful muscular contractions and incapacitation. Although this weapon is regarded as safe, the general safety of TASER remains to be determined (2). To investigate the relationship between TASER discharge and the induction of fatal arrhythmia or cardiac damage, many animal experiments involving TASER discharge used extreme durations of TASER exposure, as compared with those commonly experienced during law enforcement use of the TASER (3–5). The standard duration of TASER X26 discharge is 5 sec, but this can be repeated by the operator (1). Although the majority of instances of TASER exposure in the field last for 5 sec or less, repeated exposures can occur and may potentially be more harmful. Many deaths occurring after TASER exposure were associated with repeated TASER discharge (2,6), but any dose–response relationship related to the duration of TASER discharge remains unclear.

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The objectives of our study were to investigate the physiologic effects of repetitive TASER X26 discharge and the dose–response relationship related to the duration of TASER X26 discharge in an anesthetized swine model.

Materials and Methods

Study Design

This study was a laboratory investigation based on an anesthetized swine model. This project was reviewed and approved by the institutional animal use committee of our institute.

Animal Subjects and Handling

Fourteen commercial swine weighing between 21 and 23.5 kg were used in this study. They were delivered by local farms and were fasted from the night before the procedure. Animals were assigned to either study groups or the sham control group. Study groups were anesthetized and treated with either 5 sec of TASER discharge (n = 5) or 10 sec of TASER discharge (n = 6). The sham control group (n = 3) was also anesthetized and studied using the same protocol as that used for study groups except that they were not exposed to any TASER X26 discharges during the experiments. The average mass of animals in each group was similar.

Study Protocol

All animals of study groups and sham control group were anesthetized with an intramuscular injection of Zoletil 50®

(tiletamine/zolazepam; Virbac, France) at a dose of 5 mg/kg and Rompun® (xylazine; Bayer, Germany) at a dose of 4 mg/kg. Respiratory secretions were inhibited by intramuscular injection of Mobinul® (glycopyrolate; Myungmoon, Republic of Korea) at a dose of 0.01 mg/kg. Animals were then maintained in dorsal recumbence; all four limbs of the animal were restrained to the table. Anesthesia was supplemented by subcutaneous infiltration of 2% lidocaine in the areas of vascular dissection and tracheostomy. After anesthesia, a median longitudinal incision (10 cm long) was made, extending cranially from the suprasternal notch, exposing the trachea for the tracheostomy.

A 5.0-mm endotracheal tube was then introduced. The animals were then placed on self-respiration without ventilation support. The right femoral artery was dissected and catheterized with a 20-gauge, one-lumen catheter (Arrow International Inc., Asheboro, NC) and then connected to a monitor (IntelliVue MP30; Phillips, Andover, MA) for continuous monitoring of blood pressure and heart rate. The right femoral vein was also dissected and catheterized with a 20-gauge, one-lumen catheter (Arrow International Inc.) for blood sampling. Anesthesia was maintained by intravenous injection of Zoletil 50® (tiletamine/zolazepam) at a dose of 2 mg/kg and Rompun® (xylazine) at a dose of 2 mg/kg every 30 min.

TASER Discharge

An unmodified TASER X26 was used. The discharges were delivered by law enforcement personnel. The darts were placed along a transcardiac vector. The superior dart was placed 13 cm superior to the xiphoid process and 5 cm to the right of the midsternal line (3). The inferior dart was placed 7 cm to the left of the umbilicus (3). Darts were manually inserted perpendicular to the skin to their maximum depth (12 mm), such that the dart tip was located in subcutaneous tissue and the current path between the darts was transcardiac. The charge state of the TASER X26 was monitored before and after each discharge.

Measurements

The following parameters were monitored continuously during the experiment: systolic blood pressure, diastolic blood pressure, heart rate, pulse oximeter oxygen saturation (with the probe placed on an ear), and three-lead electrocardiography. Cardiac output (the volume of blood pumped by the heart per 1 min) was measured noninvasively using a USCOM™ (Ultrasonic Cardiac Output Monitor; USCOM Inc., Sydney, Australia). Cardiac output measurements were performed by one well-trained physician with more than 3 years of experience with USCOM™. The position and direction of the USCOM™ probe were adjusted until the USCOM™ screen displayed the maximum Doppler blood velocity wave of the aorta and the speaker displayed sharp and strong acoustic signals. The cardiac index was calculated by dividing the cardiac output by the weight of the animal to evaluate heart performance to the size of the animals. The total peripheral resistance was calculated by dividing the mean arterial pressure by the cardiac output. Hemodynamic parameters and blood samples were collected at four time points (predischarge, immediately postdischarge, and 15 and 30 min postdischarge). Immediately after being drawn, each blood sample was analyzed to determine the partial pressure of oxygen in arterial blood (PaO_2), the partial pressure of carbon dioxide in arterial blood ($PaCO_2$), arterial pH, arterial HCO_3^-, and venous lactate levels. Animals were humanely killed upon completion of the study by injection of KCL (3 M) under anesthesia.

Statistics

Parametric data are presented as means ± standard deviation. Nonparametric data are presented as medians (maximum and minimum). If variables passed the test of normality, the differences in predischarge measurements between the control and study groups were analyzed using one-way analysis of variance (ANOVA). Repeated-measures ANOVA was used to detect differences between groups. When the repeated-measures ANOVA results indicated significance, a paired *t*-test was performed to compare measurements obtained predischarge, immediately after discharge, and 15 and 30 min postdischarge. A p-value <0.05 was considered statistically significant. If the variables did not pass the test of normality, the difference in predischarge measurements between control and study groups was analyzed using the Kruskal–Wallis test. A p-value <0.05 was considered statistically significant. The Friedman test was used to detect differences between groups. When the Friedman test results indicated significance at $p < 0.05$, the Wilcoxon signed-rank test was used to compare measurements obtained predischarge, immediately after discharge, and 15 and 30 min postdischarge. Because of the use of multiple comparisons, a p-value <0.0125 obtained by using a Bonferroni adjustment was considered statistically significant. All statistical analyses were performed using SPSS version 12.0 (Chicago, IL).

Results

Blood Pressure

The differences in predischarge systolic blood pressure and diastolic blood pressure values observed between the sham control group and the study groups were not significant (one-way ANOVA, $p > 0.05$). Systolic blood pressure and diastolic blood pressure did not display any significant change in the sham control group throughout the experimental period (repeated-measures ANOVA, $p > 0.05$). Systolic blood pressure and diastolic blood pressure decreased significantly after 5 sec of TASER discharge as compared with baseline values (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and gradually returned to baseline values by 15 min. After 10 sec of TASER discharge, systolic blood pressure and diastolic blood pressure also decreased significantly as compared with baseline values (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and did not return to baseline throughout the 30-min observation period (Tables 1 and 2).

Heart Rate

The difference in predischarge heart rate observed between the sham control group and the study groups was not significant. Heart rate did not change significantly in the sham control group or in the group exposed to 5 sec of TASER discharge (repeated-measures ANOVA, $p > 0.05$). Heart rate increased significantly after 10 sec of TASER discharge as compared with baseline values (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and returned to baseline within 15 min (Table 3).

Lactic Acid

The difference in predischarge lactate levels observed between the sham control group and the study groups was not significant (one-way ANOVA, $p > 0.05$). Lactate levels in the sham control

TABLE 1—*Changes of systolic blood pressure (mean ± SD, mmHg) in each group.*

Group	Predischarge	Immediately after Discharge	15 min after Discharge	30 min after Discharge	*F*	*p*
Control	110.7 ± 6.0*	109.7 ± 3.8	115.7 ± 4.9	112.0 ± 3.0	3.94	0.126
5-sec exposure	110.8 ± 8.3*	83.8 ± 3.8†	106.2 ± 5.3	110.2 ± 3.7	45.45	0.001
10-sec exposure	114.5 ± 10.1*	81.0 ± 11.6‡	89.2 ± 10.4‡	100.5 ± 7.3‡	31.45	<0.001

*$p > 0.05$, one-way ANOVA.
†,‡Significantly lower than predischarge value.
F- and *p*-values by Greenhouse–Geisser method.

TABLE 2—*Changes of diastolic blood pressure (mean ± SD, mmHg) in each group.*

Group	Predischarge	Immediately after Discharge	15 min after Discharge	30 min after Discharge	*F*	*p*
Control	74.0 ± 14.4*	70.7 ± 8.0	72.7 ± 6.0	76.7 ± 14.2	0.85	0.465
5-sec exposure	74.0 ± 4.2*	53.0 ± 15.1†	76.6 ± 11.7	73.6 ± 8.3	17.30	0.006
10-sec exposure	72.0 ± 15.7*	49.0 ± 14.8‡	55.2 ± 11.7‡	61.0 ± 8.9‡	11.40	0.003

*$p > 0.05$, one-way ANOVA.
†,‡Significantly lower than predischarge value.
F- and *p*-values by Greenhouse–Geisser method.

TABLE 3—*Changes of heart rate (mean ± SD, beats/min) in each group.*

Group	Predischarge	Immediately after Discharge	15 min after Discharge	30 min after Discharge	*F*	*p*
Control	89.00 ± 19.16*	93.00 ± 13.23	92.67 ± 14.47	91.67 ± 17.39	0.653	0.566
5-sec exposure	93.60 ± 7.13*	98.00 ± 16.08	95.20 ± 8.59	93.00 ± 11.14	1.03	0.389
10-sec exposure	80.33 ± 9.99*	105.00 ± 15.28†	87.67 ± 4.63	87.17 ± 6.68	6.768	0.035

*$p > 0.05$, one-way ANOVA.
†Significantly higher than predischarge value.
F- and *p*-values by Greenhouse–Geisser method.

TABLE 4—*Changes of serum lactate (mean ± SD, mM) in each group.*

Group	Predischarge	Immediately after Discharge	15 min after Discharge	30 min after Discharge	*F*	*p*
Control	1.96 ± 0.50*	1.95 ± 0.74	1.92 ± 0.71	1.91 ± 0.63	0.10	0.863
5-sec exposure	2.01 ± 0.62*	3.35 ± 0.68†	3.40 ± 0.67†	3.47 ± 0.79†	36.75	<0.001
10-sec exposure	2.98 ± 0.98*	5.78 ± 1.39‡	5.98 ± 1.34‡	5.53 ± 1.61‡	61.28	<0.001

*$p > 0.05$, one-way ANOVA.
†,‡Significantly higher than predischarge value.
F- and *p*-values by Greenhouse–Geisser method.

group did not change significantly throughout the experimental period (repeated-measures ANOVA and paired *t*-test, $p > 0.05$). Lactate levels increased significantly after 5 sec of TASER discharge and remained elevated throughout the 30-min monitoring period (repeated-measures ANOVA and paired *t*-test, $p < 0.05$). Lactate levels increased significantly after 10 sec of TASER discharge and remained elevated throughout the 30-min monitoring period compared with baseline values (repeated-measures ANOVA and paired *t*-test, $p < 0.05$). The lactate levels in the group exposed to 10 sec of TASER discharge were significantly higher than in the group exposed to 5 sec of TASER discharge immediately postdischarge as well as 15 and 30 min postdischarge (one-way ANOVA, *t*-test, $p < 0.05$) (Table 4).

Cardiac Index

The difference in predischarge cardiac index observed between the sham control group and the study groups was not significant (one-way ANOVA, $p > 0.05$). Cardiac index did not change significantly in the sham control group throughout the experimental period (repeated-measures ANOVA, $p > 0.05$). Cardiac index increased significantly after 5 sec of TASER discharge as compared with baseline values (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and returned to baseline values within 15 min. Cardiac index also increased significantly after 10 sec of TASER discharge as compared with baseline values (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and did not return to baseline values during the 30-min observation period (Table 5).

Total Peripheral Resistance

The difference in predischarge total peripheral resistance observed between the sham control group and the study groups was not significant (one-way ANOVA, $p > 0.05$). Total peripheral resistance did not show any significant change in the sham control group throughout the experimental period (repeated-measures ANOVA, $p > 0.05$). Total peripheral resistance decreased significantly after 5 sec of TASER discharge (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and returned to baseline

values within 15 min. Total peripheral resistance also decreased significantly after 10 sec of TASER discharge (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and did not return to baseline throughout the 30-min observation period (Table 6).

$PaCO_2$

The difference in predischarge $PaCO_2$ observed between the sham control group and the study groups was not significant. $PaCO_2$ did not change significantly in the sham control group or the group exposured to 5 sec of TASER discharge (repeated-measures ANOVA, $p > 0.05$). $PaCO_2$ increased significantly after 10 sec of TASER discharge (repeated-measures ANOVA and paired *t*-test, $p < 0.05$) and returned to baseline at 15 min (data not shown).

Others

PaO_2, pH, and bicarbonate levels did not show any significant change in the sham control group or the study groups throughout the experimental period.

Discussion

Although most experimental studies examining the cardiovascular effect of the TASER have focused upon the potential risk of electrical discharge in precipitating a malignant ventricular arrhythmia, a dose–response relationship related to the duration of TASER X26 exposure remains to be elucidated. A dose–response trend was observed in human volunteer subjects, with heart rates increasing by 9.7, 11.1, and 12.3 beats/min for the 1-, 3-, and 5-sec TASER exposure times (7). Esquivel et al. (8) showed that a steady decrease in mean arterial blood pressure was observed after repeated exposure of Stinger S-400 CEW in the swine model, but they concluded that this finding was an effect of the anesthesia and not the exposure. The results published for the acute effects of TASER discharge on blood pressure are conflicting. A previous study showed that 5-sec exposures of human subjects to a TASER X26 led to increases in systolic blood pressure (from 138.6 mmHg to 145.8 mmHg, $p = 0.05$) (7). Vike et al. (9) showed that systolic blood pressure decreased linearly before TASER activation (139 mmHg at baseline) as compared to normal values in human subjects (123 mmHg at 60 min) (difference of 16 mmHg; 95% CI 12.7 to 20.3 mmHg; $p < 0.001$). In the swine model, systolic blood pressure was decreased after 80 sec exposure to TASER discharge (4). These conflicting results might be related to sympathetic tone. Human subjects exposed to TASER in the studies above displayed increases in baseline systolic blood pressure; these may have resulted from heightened sympathetic tone in anticipation of the TASER discharge. In contrast, the animal models were anesthetized. These manipulations may have altered the sympathetic tone of animals and may have affected the response to TASER.

In the present study, the animals became hypotensive immediately after TASER discharge; this result is in agreement with a previous study (4,8). This hypotensive response was accompanied by a decrease in total peripheral resistance and an increase in cardiac index. These findings suggested that the decrease in blood pressure might be caused by vasodilation. An increase in cardiac index might represent a compensatory response to vasodilation. Furthermore, the duration of hypotension was in proportion to the duration of TASER X26 discharge, which showed a dose–response relationship related to the duration of TASER X26 discharge. Because vasodilation developed immediately after TASER discharge, the TASER might affect smooth muscle in the peripheral vessels of swine. However, the exact mechanism of vasodilation was unclear. TASER is intended primarily to affect the motor neuron and to thereby cause the activation of skeletal contractions in humans. Notably, an increase in lactate levels was also observed after TASER discharge, with a dose–response relationship related to the duration of TASER X26 discharge. The increase in lactate level in the group exposed to 10 sec of TASER discharge was higher than in the group exposed to 5 sec of TASER discharge, and this changes in lactate level were consistent with previous report (10).

Considering that many of the deaths occurring after TASER exposure were associated with multiple or repeated discharge of the TASER, our study indicated that repetitive TASER X26 discharge led to adverse physiologic effects. Because multiple TASER discharge is not always avoidable, decisions regarding multiple TASER use must include considerations of the risk-to-

TABLE 5—*Changes of cardiac index (mean ± SD, mL/min/kg) in each group.*

Group	Predischarge	Immediately after Discharge	15 min after Discharge	30 min after Discharge	*F*	*p*
Control	77.91 ± 17.73*	85.63 ± 25.14	84.05 ± 19.81	82.45 ± 24.81	1.667	0.323
5-sec exposure	85.14 ± 8.10*	104.19 ± 6.48†	90.49 ± 7.52	88.78 ± 9.71	16.374	0.004
10-sec exposure	95.58 ± 9.53*	117.17 ± 8.42‡	120.69 ± 5.01‡	116.46 ± 11.10‡	16.467	0.001

*$p > 0.05$, one-way ANOVA.
†,‡Significantly higher than predischarge value.
F- and *p*-values by Greenhouse–Geisser method.

TABLE 6—*Changes of total peripheral vascular resistance (mean ± SD, mmHg/L/min) in each group.*

Group	Predischarge	Immediately after Discharge	15 min after Discharge	30 min after Discharge	*F*	*p*
Control	52.78 ± 12.27*	46.69 ± 14.39	49.77 ± 13.04	52.28 ± 15.26	2.449	0.235
5-sec exposure	46.59 ± 4.78*	27.59 ± 4.81†	43.65 ± 6.59	44.15 ± 5.06	41.720	<0.001
10-sec exposure	45.23 ± 8.40*	29.88 ± 11.75‡	37.95 ± 11.89‡	39.34 ± 12.11‡	26.737	<0.001

*$p > 0.05$, one-way ANOVA.
†,‡Significantly lower than predischarge value.
F- and *p*-values by Greenhouse–Geisser method.

benefit ratio. Until more studies can be performed to address the effects of multiple TASER discharges, use of multiple TASER discharge should be minimized whenever possible and TASER should be used only for a limited number of short applications. A study regarding the safe upper limit of TASER discharge is needed.

The present study has some limitations. First, although meaningful statistical analysis could be performed, limited numbers of animals were used, and our study was too small to allow firm conclusions. Second, although TASER is generally used on highly agitated individuals who may be under the influence of heightened sympathetic tone, the animals in our study were anesthetized, and the sympathetic tone of animals might have been altered. This scenario was not relevant to the real situation. Although changes observed in unanesthetized swine model could be different from those observed in anesthetized swine model, the anesthetized swine model was considered to be more appropriate for CEW experiments because it could facilitate measurements of physiologic and biochemical parameters. And we must also consider ethics even if there may be potential advantages of studying unanesthetized swine model (11). Third, the size of swine used in our study (21–23.5 kg) was similar to the size of children. The response of blood pressure to TASER in small swine may be different from that in large swine. Therefore, these small animals may not have been appropriate surrogates for human adults. Fourth, although the accuracy of USCOM™ as compared to a flow probe was assessed in the animal model (12), flow probes are the gold-standard tool used to measure cardiac output in the laboratory. The baseline cardiac index of animals in our study was slightly lower than that in other studies (3,13). This result suggested that USCOM™ may have underestimated the cardiac index of swine in our study. Fifth, the observation period after TASER discharge in our study was just 30 min. Therefore, we were not able to evaluate the chronic effects of repeated TASER discharge. Further studies are needed to overcome these limitations.

Conclusion

This study demonstrated that 10 sec of TASER X26 discharge led to a longer duration of vasodilatory hypotension and a higher increase in lactate level than did 5 sec of TASER X26 discharge in an anesthetized swine model. These results suggest that repetitive TASER X26 discharge resulted in adverse physiologic events with a dose–response relationship related to the duration of TASER X26 discharge in this anesthetized swine model.

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