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Physiological responses in reindeer to the application of a conducted electrical weapon

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Abstract: Conducted electrical weapons (CEWs) have potential as effective alternatives to chemical restraint for short-term nonroutine capture and handling of wildlife. To assess immediate and delayed physiologic effects of exposure to a CEW, we assigned 15 captive reindeer (*Rangifer tarandus tarandus*) to 1 of 3 treatment groups: immobilized with carfentanil and xylazine (CX), 10-second exposure to a CEW, or exposure to the CEW while immobilized with CX (CEW+CX). Blood samples were collected pre-treatment, immediately post-intervention, 10 minutes, 20 minutes, 4 hours, and 24 hours post-intervention. Physiologic effects were evaluated by analysis of blood, clinical observation for signs of physiologic compromise, and vital signs. Parameters that changed significantly ($P < 0.05$) post-exposure (lactate, glucose, rectal temperature, blood oxygen, cardiac troponin I, cortisol, and catecholamines) were not significantly different from baseline values within 24 hours. Cortisol, glucose, and peak rectal temperature were lower in CEW-exposed individuals, while lactate, oxygen, and catecholamines were higher than for the CX-exposed individuals. The catecholamine response observed in the CEW-only group paralleled the response in the CEW+CX group. No long-term health effects were detected from either restraint method. Use of a CEW does not appear to increase the risk of capture myopathy.

Key words: conducted electrical weapon, physiological response, *Rangifer tarandus tarandus*, reindeer, TASER®

Effective wildlife management includes efficient and safe methods for short-term restraint to reduce stress on animals while facilitating actions such as sample collection, restraint, handling for the purpose of relocation, or release from snares or entanglements. Chemical restraint is frequently used but may not be appropriate in all situations, such as when there is a risk to the public from misdirected projectiles, restrictions on use of projectors considered firearms, or from exposure to controlled substances (e.g., opioids) from darts or drug residue in meat salvaged for consumption. Additionally, many of the drugs used for chemical restraint are controlled substances, and trained personnel that can legally administer them are not always available. Darting and chemical restraint also have inherent risks to wildlife including injury and capture mortality (Kock et al. 1987). Conducted electrical weapons (CEWs; Figure 1) are being used as a nonlethal alternative for short-term (<1 min) restraint or hazing (Lewis et al. 2011).

Interest is high among wildlife agencies in the efficacy of this tool. Preliminary research was undertaken by Alaska Department of Fish and Game Division of Wildlife Conservation at the Kenai Moose Research Center (Sterling, Alaska, USA) to evaluate CEW effects on moose (*Alces alces*; K. B. Beckmen, Alaska Department of Fish and Game, unpublished data), and those results were considered when designing this study as well as for developing a standard operating procedure for CEW use by the Alaska Department of Fish and Game (2010). Since then, Alaska Department of Fish and Game Division of Wildlife Conservation personnel have used CEWs for human safety while temporarily restraining wildlife, such as moose, to remove them from entanglements (Lewis et al. 2011). For example, as an alternative to killing a cow moose, a CEW has been used to protect personnel from an aggressively protective cow in order to rescue her calf that had gotten trapped in an open pit (Lewis et al. 2011). Additionally, a CEW has been used for hazing and aversive conditioning in >400

Figure 1. TASER X2 device (TASER International, Scottsdale, Arizona, USA). Axon/TASER does not endorse the use of their products for wildlife usage.

bears (*Ursus* spp.) between 2010 and 2017 at Port Armstrong, a remote fish hatchery on Baranof Island near Sitka, Alaska, USA and at other locations around Alaska. Application of CEWs to bears led to greater human avoidance behavior and decreased returns to the human-occupied areas of the fish hatchery compared to the use of other standard hazing methods (P. Mooney, Alaska Department of Fish and Game, unpublished data).

Conducted electrical weapons have widespread use by law enforcement agencies for the restraint of humans. In humans, CEWs have been found to reduce the rate and severity of injuries in both suspects and officers (Smith et al. 2007, Bozeman et al. 2009). However, there are still safety concerns, including their contribution to arrest-related deaths (Bozeman et al. 2009, Jauchem 2015). Many of the concerns, especially potentially fatal cardiac effects (such as ventricular fibrillation) and acidosis (from increased lactate), although not reported in humans, are based on animal model studies. These animal studies have been mostly done in pigs (*Sus* sp.; Walter et al. 2008, Jauchem et al. 2009, Jauchem 2015), where prolonged or repeated discharges and location of the probes (such as transcardiac positioning or direct chest exposures) may be factors. In humans, physiological changes (including measures of stress, acidosis, and cardiovascular disruption) from exposure to CEWs have been no greater than those seen with other law enforcement nonlethal restraint methods (Ho et al. 2008, Dawes et al. 2009, Ho et al. 2009*a*, Ho et al. 2009*b*, Ho et al. 2010).

The American Veterinary Medical Association does not currently recommend the use of electro-immobilization, such as with a CEW, for routine capture or restraint of animals (AVMA 2008, AVMA 2010). The concern with their use is the potential for distress and physical discomfort to an animal. Their use has been considered potentially appropriate in emergency or life-threatening situations when dealing with an aggressive or dangerous animal. It is not the intent of this paper to recommend the use of CEWs for routine capture. The goals of this study are to better understand the effects of CEWs on animals to evaluate their potential use in management situations when other methods (such as chemical immobilization) are not available or practical.

Because of the concern for capture myopathy (or exertional rhabdomyolysis) in wildlife, it is critical to ascertain that use of a CEW does not add more stress than is already associated with capture and handling. Capture myopathy is a syndrome that leads to fatality and is seen in situations after prolonged or severe stress or exertion in animals. It is characterized by muscle (skeletal and cardiac) necrosis and renal failure. Acidosis (low blood pH, often due to increased lactic acid) and hyperthermia may exacerbate the condition (Williams and Thorne 1996).

The goal of this study was to assess the immediate and delayed physiologic effects of exposure to a CEW and compare them to the effects of chemical restraint using carfentanil and xylazine (CX), the standard drug combination for wildlife capture and handling in the state of Alaska. Readily available captive reindeer (*Rangifer tarandus tarandus*) were used as a model for barren-ground caribou (*R. tarandus granti*) in this study. We hypothesized that the physiological effects of a CEW would not be significantly different than chemical restraint, and thus, would not pose a greater risk of capture myopathy.

Methods

In June 2014, 15 adult female reindeer housed at the Robert G. White Large Animal Research Station (University of Alaska Fairbanks Institute of Arctic Biology, Fairbanks, Alaska, USA) and habituated to physical restraint and blood collection were assigned to 1 of 3 treatment groups (n = 5 for each treatment):

immobilized (hand injection IM) with CX (0.03 mg/kg carfentanil and 0.3 mg/kg xylazine), a 10-second exposure to a CEW, or a 10-second exposure to the CEW while immobilized with CX (CEW+CX).

For CEW interventions, a TASER X3W® wildlife-specific handheld conducted electrical weapon (TASER International, Scottsdale, Arizona, USA) was used (large animal setting). The device has an open circuit arcing voltage of 50,000 V to achieve an arc to overcome resistance to air, fur, and hide and complete a circuit between the 2 wired probes through which the charge is delivered. When a circuit is complete, the voltage drops to approximately 1,200 V and the device delivers pulsed electricity consisting of 85–130 microcoulombs of charge per pulse at a pulse rate of 29 pulses per second (0.0035 amperes).

Pre-treatment (T0) blood samples were collected from each individual while halter tied. Animals were then exercised for 5 minutes by running them within the corridors between pens to simulate a pre-darting excitement, then brought into a chute for treatment. Subsequent blood samples were collected at the following time points: immediately (TC), 10 minutes (T1), 20 minutes (T2), 4 hours (T3), and 24 hours (T4) post-intervention. For the immobilized individuals, drugs were injected, and anesthesia was induced (2–4 min) and maintained. After 10 minutes post-injection, naltrexone (100 mg per mg of carfentanil given) and atipamazole (1 mg per 10 mg of xylazine given) were administered intravenously to antagonize the effects. Recovery to standing occurred in 2–4 minutes. The TC for the individuals exposed to the CEW was immediately after the device was turned off; for the CX group, it was 10 minutes after the drugs were administered (just before antagonists were given). Behavior and vital signs were monitored throughout exposure and through recovery. Rectal temperature was measured with a clinical digital rectal thermometer.

Venous blood (from the jugular vein) was collected in lithium heparin (6 ml Vacutainer, Becton, Dickinson and Co, Franklin Lakes, New Jersey, USA) and serum separator evacuated tubes (8.5 ml Vacutainer, Becton, Dickinson and Co, Franklin Lakes, New Jersey, USA). Immediately after sampling, a subset of whole blood was tested using point of care devices. EPOC (Heska, Loveland, Colorado, USA) was used to determine pH, blood carbon dioxide (pCO2), blood oxygen (pO2), sodium (Na+), potassium (K+), chloride (Cl-), ionized calcium (Ca++), creatinine, glucose, and hematocrit at T0, T1, T2, and T3. Lactate Pro Analyzer (Arkray Global Business, Kyoto, Japan) was used to measure blood lactate at all 6 time points.

Samples were centrifuged for 10 minutes at 1500 G (Clay Adams Triac centrifuge, Becton, Dickinson and Co, Franklin Lakes, NJ, USA). Serum and plasma were flash frozen in liquid nitrogen, then stored at –80°C until analyzed. The T0 and T3 serum were sent to the University of Alaska Fairbanks Animal Resources Center (Fairbanks, Alaska, USA) for chemistry analysis on an Element DC Veterinary Chemistry Analyzer (Heska, Loveland, Colorado, USA) using test slides for albumin, alkaline phosphatase (ALP), alanine transferase (ALT), blood urea nitrogen (BUN), total calcium, creatine kinase (CK), creatinine, cholesterol, gamma-glutamyl transferase (GGT), globulin, glucose, phosphorus, total bilirubin, and total protein. Additional serum samples (from T0, T1, T2, T3, and T4) were sent to Texas A&M Veterinary Medical Diagnostic Laboratory (College Station, Texas, USA) and tested for cardiac troponin I (cTnI) using a cardiac troponin I high sensitivity assay on an ADVIA Centaur CP Ultra-TnI (Siemans Healthcare, Tarrytown, New York, USA) and serum cortisol using a solid phase, competitive chemiluminescent enzyme immunoassay. Plasma (from T0, TC, and T3) was analyzed at Ani Lytics Incorporated (Gaithersburg, Maryland, USA) for catecholamines (epinephrine and norepinephrine) using a radio-immunoassay (RIA) with an extraction/acylation prior to assay. The reagents were obtained from ALPCO (Salem, New Hampshire, USA).

Data analysis

Mean and SD were calculated for all parameters. Additionally, the change from baseline (results from T3 minus results from T0) was calculated for blood chemistry results. The T3 laboratory chemistry analysis results for 1 individual (in the CEW+CX group) were outside of the expected physiologic range for most parameters. Comparing the results

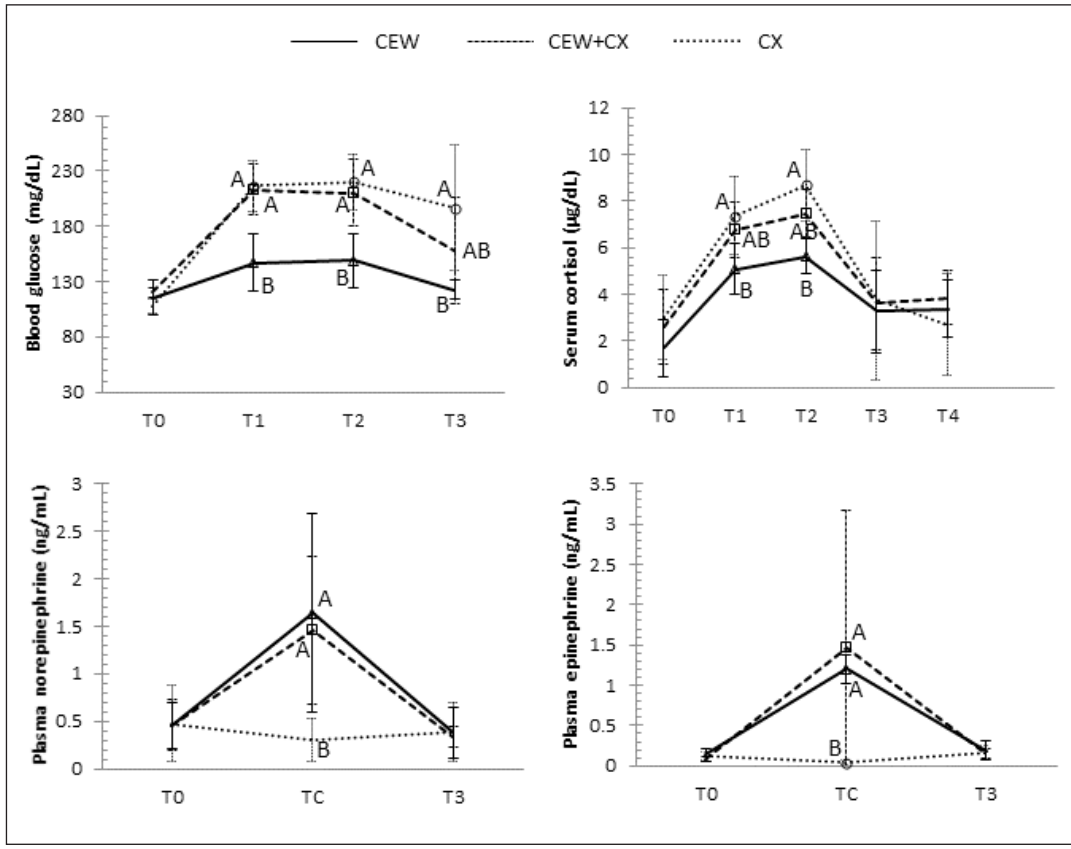

Figure 2. Mean ± SD for parameters to evaluate stress in captive reindeer (*Rangifer tarandus tarandus*) whole blood (glucose), serum (cortisol), and plasma (catecholamines: norepinephrine and epinephrine) collected at baseline (T0), immediately post-exposure (TC), 10 min (T1), 20 min (T2), 4 hours (T3), and 24 hours (T4) post-exposure to a 10-sec exposure to a conducted electrical weapon (CEW), immobilization with carfentanil/xylazine (CX), or to the CEW while immobilized with CX (CEW+CX). Open points (Δ = CEW, ○ = CX, □ = CEW+CX) indicate values that differed significantly ($P < 0.05$) from T0 for that treatment. Points with different capital letters indicate treatments that differed significantly from the other treatments at that time point. Exposure occurred June 2014 in Fairbanks, Alaska, USA.

for glucose analyzed using the point of care device (EPOC) to the glucose results from the standard, benchtop laboratory chemistry analyzer, it appears there was an error with the laboratory results for that sample. There was no difference in the significance of the results whether the individual was retained or omitted from the analysis. When reporting means and ranges, the individual was omitted.

Glucose (serum chemistry and EPOC) and lactate (EPOC and Lactate Pro) results were found to be consistent between the different testing methods (glucose, $r^2 = 0.90$ for all time points; lactate, $r^2 = 0.95$ for lactate values <8 mmol/L and $r^2 = 0.75$ for values >8 mmol/L), so multiple comparison analyses were done using results from the method with the most data points (EPOC for glucose, Lactate Pro for lactate).

Differences among groups were determined using the Kruskal-Wallis nonparametric analysis blocked by treatment group and sampling time. For analyses where the global test was significant, pairwise comparisons were calculated using the Conover-Inman method. For all comparisons, $P < 0.05$ was considered significant. Analyses were done using Stats Direct (version 2.8.0, StatsDirect Ltd., England 2013).

Results

For all comparisons, the results at T0 (baseline) did not vary significantly among treatment groups.

Within each treatment group, results at T3 (4 hours post-intervention) were within an SD of the results at T0 for serum chemistry parameters

with the exception of glucose and CK. For all treatment groups, CK was increased at T3 compared to T0 (P = 0.0016); however, there was no significant difference among treatment groups at either time point. The range that CK increased was wide for all groups (339–9725 U/L for CX, 365–2543 U/L for CEW, 418–8144 U/L for CEW+CX).

Blood glucose (Figure 2) increased at T1 and T2 for all groups (and was still significantly elevated at T3 for the CX group). The increase in glucose was greater in the CX-treated group than the CEW group at T1 (P = 0.0017), T2 (P = 0.0019), and T3 (P = 0.003), although results were not significantly higher than for the CEW+CX group. Glucose in the CEW+CX group was higher than the CEW group at T1 (P = 0.0038) and T2 (P = 0.0048). For 1 individual in the CX group, blood glucose was measured at 24 hours post-exposure and had returned to baseline value (data not shown).

Cortisol (Figure 2) increased significantly from T0 within all treatment groups, peaking at T2 and decreasing to baseline by T3. The CX individuals had higher cortisol values at both T1 (P = 0.494) and T2 (P = 0.0375) than for the CEW individuals. The CEW+CX group did not vary significantly from either the CX or the CEW-treated groups.

Both epinephrine and norepinephrine peaked immediately after exposure to treatment for the CEW and CEW+CX groups (Figure 2). The values at TC were significantly higher for CEW and CEW+CX than for CX. Catecholamine levels decreased to baseline levels by T3. There was no significant difference between CEW and CEW+CX treatments at any time point. Within treatment groups, epinephrine decreased at TC compared to T0 or T3 for the CX group (P = 0.0123); however, there was no significant difference across time points for norepinephrine.

Significant differences in hematocrit (P = 0.0066), pO2 (P = 0.0016), pCO2 (P = 0.0123), and glucose (P < 0.0001) were found for whole blood test using EPOC. Details of multiple comparisons for significant differences are further detailed below. No significant differences in treatment or time point results for pH, creatinine, Ca++, K+, or Na+ were found.

Hematocrit at T2 (37% ± 3.7) was lower compared to T0 (42% ± 3.5) for the CX-treated group (P = 0.034). For the CEW+CX group, hematocrit at T2 (39% ± 3.2; P = 0.0316) and T3 (39% ± 2.1; P = 0.0394) were lower than at T0 (43% ± 3.6). Although the hematocrit for the CEW group did not vary significantly across time points (P = 0.1318), it was higher than in either CX or CEW+CX at T1 (P < 0.0001 and P = 0.0265), T2 (P = 0.0011 and P = 0.0084), and higher than the hematocrit for CEW+CX at T3 (P = 0.019).

Venous oxygen (Figure 3) was lower in the CX and CEW+CX groups at T1 (P < 0.0001 and P = 0.0018) and T2 (P < 0.0001 and P = 0.0012) compared to the CEW group. At T2, pO2 was also lower in the CX group than the CEW+CX group (P = 0.0485). By T3, pO2 had returned to baseline levels for all treatment groups. Within treatment groups, pO2 did not vary significantly across time points for CEW+CX. However, the pO2 for the CX group was lower at T1 (P = 0.012) and T2 (P = 0014) than at T0. In the CEW group, pO2 was higher at T1 (P = 0.016) and T2 (P = 0.007) than T0.

There was no significant difference in pCO2 (Figure 3) when comparing treatment groups at each time point. However, pCO2 did vary significantly over time within treatment groups. For the CEW group, pCO2 was lower at T1 (P = 0.0057) and T2 (P = 0.0008) than at T0, and for the CEW+CX group, pCO2 was lower at T1 (P = 0.02) and T2 (P = 0.0136) than at T3. Additionally, 1 individual in the CX group had a much higher pCO2 at T1 than any other individual (pCO2 = 56.8 mm/Hg), although as a group the pCO2 values did not vary significantly.

Cardiac troponin I (Figure 3) increased from baseline at T3 in all groups, although this within-group increase was only statistically significant for the CEW and CEW+CX groups (P < 0.0001 for both). Levels of cTnI remained higher at T4 compared to T0 for CEW (P = 0.0085) and CEW+CX (P = 0.0005). When comparing results between treatments at each time point, cTnI for the CEW+CX group at T3 was greater than for the other 2 groups (P = 0.0165 compared to CX and P = 0.0313 compared to CEW). The between-group differences for CEW and CX groups were not significant at any time point. By T4 (24 hours post-exposure), cTnI had decreased toward baseline levels; there were no significant differences between groups at T4.

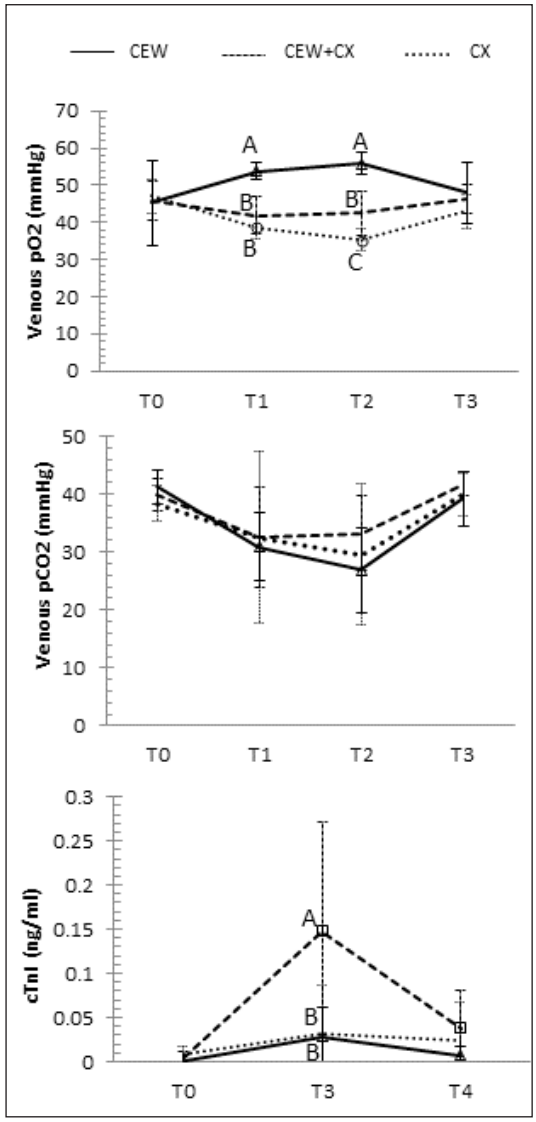

Figure 3. Mean ± SD for captive reindeer (*Rangifer tarandus tarandus*) respiratory (venous partial pressure of oxygen [pO2], carbon dioxide [pCO2]) and cardiac parameters (cardiac troponin I [cTnI]) in blood collected at baseline (T0), 10 min (T1), 20 min (T2), 4 hours (T3), and 24 hours (T4) post-exposure to a 10-sec exposure to a conducted electrical weapon (CEW), immobilization with carfentanil/xylazine (CX), or to the CEW while immobilized with CX (CEW+CX). Open points (Δ = CEW, ○ = CX, □ = CEW+CX) indicate values that differed significantly ($P < 0.05$) from T0 for that treatment. Points with different capital letters indicate treatments that differed significantly from the other treatments at that time point. Exposure occurred June 2014 in Fairbanks, Alaska, USA.

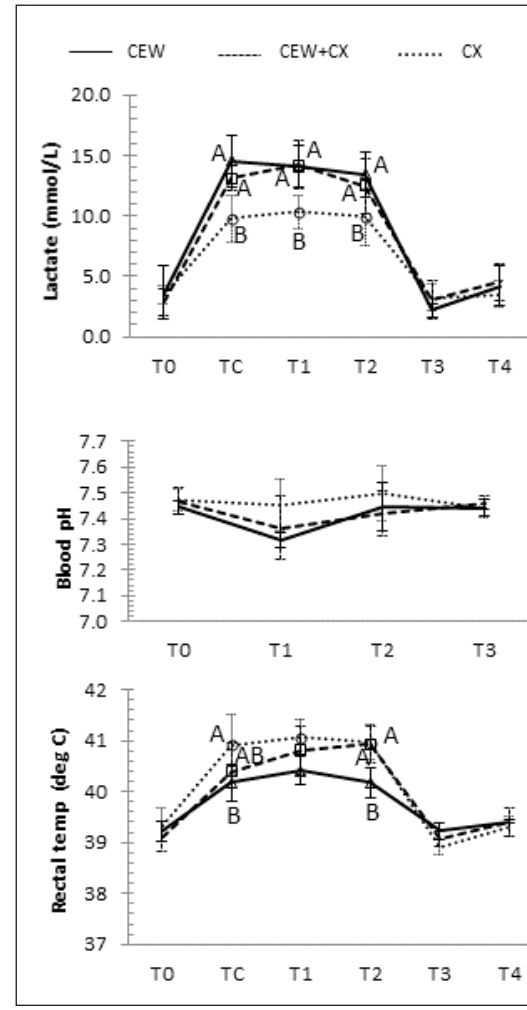

Figure 4. Mean ± SD for captive reindeer (*Rangifer tarandus tarandus*) blood lactate, blood pH, and rectal temperature collected at baseline (T0), immediately post-exposure (TC), 10 min (T1), 20 min (T2), 4 hours (T3), and 24 hours (T4) post-exposure to a 10-sec exposure to a conducted electrical weapon (CEW), immobilization with carfentanil/xylazine (CX), or to the CEW while immobilized with CX (CEW+CX). Open points (Δ = CEW, ○ = CX, □ = CEW+CX) indicate values that differed significantly ($P < 0.05$) from T0 for that treatment. Points with different capital letters indicate treatments that differed significantly from the other treatments at that time point. Exposure occurred June 2014 in Fairbanks, Alaska, USA.

Lactate (Figure 4) increased and was higher at TC, T1, and T2 ($P < 0.0001$) than at T0 for all treatment groups. The increase was similar (and not significantly different at each time point) in both the CEW and CEW+CX groups. Lactate at TC, T1, and T2 for the 2 groups treated with the CEW was higher than the lactate peak for the CX-only group (P ranged from 0.006–0.0028 for

CEW compared to CX, and from 0.0094–0.0153 for the CEW+CX comparisons). For all treatment groups, lactate returned to baseline values by T3. Although there was a slight decrease in pH at T1 for the CEW and CEW+CX groups, the change was not significant ($P = 0.3537$).

Rectal temperature (Figure 4) increased ($P < 0.001$) in all groups after exposure to treatment (TC) and remained elevated through T2. The increase was greatest for the CX treatment groups. Temperatures in the CX group were elevated compared to the CEW group at TC ($P = 0.0481$), T2 ($P = 0.0085$), and approached significance at T1 ($P = 0.0576$). The CEW+CX temperatures were higher from those in the CEW group at T2 only ($P = 0.0101$). The CEW+CX and CX rectal temperatures did not vary significantly at any time point.

Discussion

For all treatment groups, the observed changes in lactate, glucose, rectal temperature, blood oxygen, cardiac troponin I, cortisol, and catecholamines were transient and returned to (or approached) baseline levels within 24 hours. No long-term (>24 hour) or life-threatening health effects were observed for either restraint method. All individuals were still alive 1 year later and had immediately returned to normal breeding function within the herd (J. Blake, University of Alaska Animal Research Center, personal communication).

Creatine kinase is often used as a measure of muscle stress or damage (both skeletal and cardiac). It can reversibly increase with muscle exertion from both handling as well as exercise. To interpret the results and determine if increases are normal physiological responses due to activity or indicative of a more severe myopathy, levels of other enzymes (which also might be affected) and other physiological parameters (like body temperature) are considered. The increase in CK between T0 and T3 was within an expected range due to the physical activity the animals underwent. Similar values have been reported in clover-trapped white-tailed deer (*Odocoileus virginianus*; Boesch et al. 2011), while much higher values for CK are reported in roe deer (*Capreolus capreolus*) captured with drive nets (Montane et al. 2003). The 2 individuals with the highest CK measured at T3, 9632 IU/L and 8032 IU/L (from the CX and CEW+CX groups, respectively), were noted to have struggled while getting in the chute (CX individual) or had a stormy recovery from anesthesia (CEW+CX individual). These additional exertions can account for the observed elevations in CK.

There was no evidence of capture myopathy triggered by any of the treatments in this study. The higher lactate (and lower pH, although it was not statistically significant) seen in the CEW-treated groups are consistent with the effects of muscle contraction due to the electronic stimulus. Similar lactate levels have been reported in clover-trapped white-tailed deer (Boesch et al. 2011), darted moose (Evans et al. 2012), and in humans after intense physical activity (Ho et al. 2009*b*). The greater increase in rectal temperature in the CX-treated individuals is consistent with the impaired thermoregulation caused by the use of opioids (carfentanil in this case) and especially alpha-2 adrenergic agonists (xylazine in this case). The observed increases in temperature and lactate (and decrease in pH) resolved within 24 hours. No evidence of renal dysfunction (assessed by BUN and creatinine) was found. Additionally, all individuals survived and continued to behave normally after the study.

In addition to impaired thermoregulation, the use of chemical immobilizing agents has been associated with impaired cardiopulmonary function, including respiratory depression, hypoxemia, and decreased hematocrit (potentially a result of sequestration of erythrocytes in the spleen or vasodilation due to xylazine). The decrease in venous pO2 and hematocrit observed in the groups treated with CX is consistent with this. Cardiopulmonary effects in these individuals resolved after recovery (by 4 hours post-exposure). The decrease in blood oxygen observed with chemical restraint was not present in the CEW-exposed individuals. The increase in pO2 seen in the CEW only treated group is likely due to an increased respiratory rate observed immediately upon release of the electrical stimulus. The decreased pCO2 found in the CEW-exposed group is consistent with hyperventilation.

Cardiac troponin I is used as a measure of damage to cardiac muscle. The majority of cTnI is found within the cardiac muscle and is

released when the muscle undergoes damage (e.g., myocardial necrosis). In several species, myocardial necrosis (including damage caused by acute coronary syndrome) is characterized by cTnI levels increasing within 4 hours of onset of signs, peaking 24–36 hours later, and decreasing over the course of days to weeks (Cummins and Cummins 1987, Varga et al. 2009, Agewall et al. 2011). However, it has been hypothesized that transient (cTnI levels return to baseline within 24 hours) mild increases may be due to the cytosolic component rather than damage leading to necrosis. Between 3% and 8% of cTnI is unbound in the cytoplasm. Mild transient increases have been reported after exercise in human, equine, and canine endurance runners (Durando et al. 2006, McKenzie et al. 2007, Nostell and Haggstrom 2008, Gresslien and Agewall 2016) and may be related to a reversible ischemic event or a mismatch in the supply and demand for oxygen (White 2011). Although the mechanism is not fully understood, it is believed to be physiological rather than pathological (Gresslien and Agewall 2016).

Although cTnI was detected and increased levels were found in some individuals (especially for the CEW+CX group), the highest amount detected (0.35 ng/mL) was well below the levels reported by Varga et al. (2009) as being associated with myocardial necrosis (using the same assay) in cattle (*Bos taurus*; 1.04 ng/ml). Additionally, the slight increases in our study animals were transient (returning to baseline within 24 hours), similar to the exercise-related changes described in other species (Durando et al. 2006, McKenzie et al. 2007, Gresslien and Agewall 2016). Thus, the detected cTnI may be cytosolic cTnI rather than an indication of irreversible damage to cardiac muscle. The CEW+CX group may have been affected the most because of the increased demand for oxygen from the contracting muscles in response to the CEW coupled with the decreased heart and breathing rates caused by the drugs (oxygen supply/demand mismatch). The significance of the difference in the CEW group (both for T3 and T4) is likely due to the limitations of the nonparametric statistical methodology used (for which ranks of the measured results are used). The lack of cTnI detected at T0 in any individuals (unlike for the other 2 treatment groups) had a strong influence on the comparison of rankings.

Glucose, catecholamines (such as norepinephrine and its metabolite, epinephrine), and cortisol can be used to assess stress in an individual. Norepinephrine and epinephrine are part of the sympathetic nervous system acute response to stress, while cortisol is associated with the chronic response of the hypothalamic-pituitary-adrenocortical (HPA) axis. Glucose is released as a response to increased levels of cortisol and catecholamines. The immediate catecholamine response observed after exposure to the CEW, even in the group that was also treated with CX, is consistent with a physiological response, although a cognitive one (i.e., due to fear or pain) cannot be ruled out. The mean peak was similar to the catecholamine response in net-gun captured bighorn sheep (*Ovis canadensis*; Martucci et al. 1992). In the CEW-exposed individuals, glucose levels were lower than, and cortisol levels were similar to levels measured in clover-trapped white-tailed deer (Boesch et al. 2011). Cortisol levels were also much lower than for drive-net captured roe deer (Montane et al. 2003) and similar to levels measured in captive reindeer exposed to light restraint or physical activity (Arnemo and Caulkett 2007). Exposure to a CEW does not appear to trigger a greater stress response than current physical restraint methods. Additionally, all measures of stress returned to (or approached) baseline within 4 hours, indicating no prolonged stress response.

Evaluating relative stress with exposure to CX is complicated by the effects of the immobilizing drugs. The alpha-2 adrenergic agonist used, xylazine, blocks production of norepinephrine and epinephrine. This results in respiratory depression, hypotension, and bradycardia (Sinclair 2003). Additionally, xylazine decreases insulin release from the pancreas and increases glucose production from the liver (gluconeogenesis), resulting in hyperglycemia (Fagerholm et al. 2011). Thus, the decreased pO2 and increased glucose seen in the immobilized individuals is likely due to xylazine. The inhibitory effect on norepinephrine is consistent with the lack of a catecholamine response seen in the CX group. Additionally, naltrexone is an opioid receptor

antagonist. It significantly increases cortisol levels due to blocking the central opioid receptors (which are inhibitory), resulting in an activation of the HPA axis. The cortisol and glucose levels observed in the CX group are similar to those reported in medetomidine (a more potent alpha-2 adrenergic agonist) exposed reindeer (Miller et al. 2013), and CX-exposed muskoxen (*Ovibos moschatus*; Harms et al. 2012).

Management implications

Most chemical immobilants are delivered to free-ranging wildlife via remotely projected darts. Darting, especially with heavy weight rapid injection darts, entails risks of injury, such as dart trauma and infections, well beyond mere immobilization. These effects were mitigated in this study by hand injection of the drugs (Cattet et al. 2006). Use of CEWs removes the risk of the severe morbidity and mortality risks directly associated with darting and anesthesia.

The risks of using either CEW or chemical immobilization methods need to be assessed in relation to the circumstances and necessity of restraint. Both CEWs and CX have some physiologic effects that may be considered beneficial over the other (e.g., better oxygenation, less thermoregulatory disruption, decreased risk of bradycardia and bradypnea with use of CEWs; decreased immediate stress response with use of chemical immobilants). Both have strengths and limitations as tools for effective management of wildlife. While CX allows for longer handling time for intervention and can be administered from a longer distance including from a helicopter, it has more rigorous training and legal requirements. The use of a CEW has a very short duration of immobilization (<1 min), and has a limited range (<11 m, dependent on cartridge design). The CEW use, however, is less expensive, more readily available, has decreased health risks for the public as well as personnel, does not expire (cartridges have a 5-year shelf life), and is insensitive to most environmental conditions (except extreme cold below -20°C and heat greater than 50°C).

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