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Cells to Society: Lactate and Neuromuscular Incapacitation Devices

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Abstract— **Devices employed in electrical policing rely on fundamental responses of nerve and muscle to supraphysiologic current to quell disruptive behavior. Whilst widely deployed the use of neuromuscular incapacitation (NMI) remains controversial, in part, due to gaps in understanding of the underlying mechanisms related to injury. NMI device manufacturers are thus constrained by empirical evidence for safety and effectiveness for a specific device design. Here we examine published data for several NMI devices considering *ex vivo* signal characteristics and *in vivo* responses in relation to effectiveness and injury. The sensitivity of lactate production to NMI device signal frequency (Hz) and macro-dosimetry is explored as a primary device design factor in NMI modes of injury. The non-lethal approach to policing regardless of technology will result in fatalities due to compromised health and substance abuse status unknowable at the time of NMI application. Thus, research to establish a science-based understanding of NMI injury mechanisms, particularly for lactate production and limitations of deployment, are essential for social acceptance and improved NMI device design.**

I. Introduction

The Colt 45, introduced in 1873 by the Colt Manufacturing Company, was also called the Colt Peacemaker [1] and was intended to promote social order. The popular 6-round revolver in many respects still represents the primary means of ballistic policing world-wide despite associated violence, lethality and social decry. Non- lethal approaches to policing to promote social order and peacekeeping are worthy humanitarian goals but present biological and social complexities not present in the lethal genre [2]. The growing use of NMI devices in policing over the last decade [3] suggest the potential for a future with fewer guns and violence, however, scientific understanding and social acceptance of the technology and deployment are controversial and lack consensus [4]. The uncertainties represent considerable challenges to NMI device makers.

Here we examine published data representing selected studies of NMI devices to identify lessons learned and pathways for both scientific and social acceptance. The NMI industry from one perspective can be viewed as an electronic version of the "wild, wild west" as it is neither regulated nor promulgated by any state or federal agency (e.g., FDA, Bureau of Tobacco and Firearms, Consumer Product Safety Commission). Alternatively, as we suggest here, the NMI genre is the subject of increasingly sophisticated scientific study at cellular, systemic and whole-body scales converging on an understanding of underlying mechanisms for functionality and thresholds for injury. Improvements in device design and mode of application to subjects should expand NMI options matched to subject status and risk for injury and death.

A key area of concern identified initially in [5] continues to involve the production and sequelae of lactic acid production (e.g., global lactic acidosis, compensatory hyperventilation, cardiac arrhythmias) in relation to NMI device specification and application to high risk groups for injury and death [6]- [8]. NMI application during a single incident is often prolonged lasting from 15 seconds up to recorded cases of 450 seconds [9]. Additionally, interest in prolonged applications of up to 60 seconds with modified waveforms require careful study with respect to lactic acid and associated changes in blood chemistry [10].

Published data for animal and human studies using un-modified NMI devices of differing designs under comparable conditions are few to-date. Accordingly, device comparisons have not been examined for fundamental differences in electronic signal output and dose-dependent *in vivo* response. Data employing signal generators are included in this work to assess a mechanistic basis for injury and effectiveness. While the comparisons presented here are limited the data clearly suggest that additional research and design improvement of NMI devices are needed to ensure effectiveness and public safety.

II. DATA

Data for the Taser X-26 (Taser International [11]) and the MK 63 (Aegis Industries, Inc. [12]) are utilized in this work. Three publications, conducted by the same laboratory, represent comparable discharge conditions for both devices [13]-[15]. Additionally, three publications [16], [17], [10] with data for lactate concentration in relation to the Taser X-26 and a modified waveform generated X-26 output [18], [19] are included for comparison and discussion. The studies represent animal data as described in respective references. Implications of the animal data are discussed in relation to human studies employing the X-26. Human data for the Mk63 have not been published. Additional discussions related to *in silico* model studies and to broad social analysis are included. The data adapted from the cited

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references and illustrated graphically in this work are shown in Table 1. Figures from the respective publications were digitized allowing data point extraction. Standard errors of grouped data reported in the publications were not utilized in this analysis except where noted.

TABLE I
DATA SUMMARY

Device	Hz (cycles per second)	Number of Subjects (experimental/control)	Discharge Conditions	Reference
Taser X-26 Unmodified	19	6/5	80 sec total discharge: 40 sec on,10 sec off, 40 sec on	Dennis et al., 2007
Taser X-26 Unmodified	19	10	90 sec total discharge: 5 sec on, 5 sec off, for 3 minutes; 4 variants over 4 minute intervals	Jauchem et al., 2006
Taser X-26 Unmodified	19	10	15 sec total discharge: 5 sec on, 5 sec off, over a 30 sec interval	Jauchem et al., 2008
Taser X-26 Waveform Generator	19	10	30 sec or 60 sec continuous exposure	Jauchem et al., 2009
Mk63 Unmodified	65	22/4	continuous discharge for 10, 20, 40 sec groups; 80 sec discharge consisted of two 40 sec discharges with a 10 sec interval	Valentino et al., 2008
MK 63 Unmodified	65	24/6	80 sec total discharge: 40 sec on,10 sec off, 40 sec on	Valentino et al., 2007

III. FIGURES

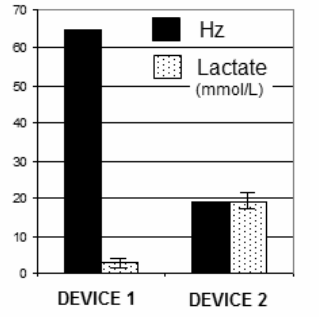


Fig. 1. Frequency shown in Hz (solid bars) and associated central venous blood lactate (stippled bars) for two NMI devices: device 1 (Mk 63, Aegis Industries [13]); device 2 (Taser X-26, Taser International [15]). Error bars are shown for lactate data as reported in the respective publications.

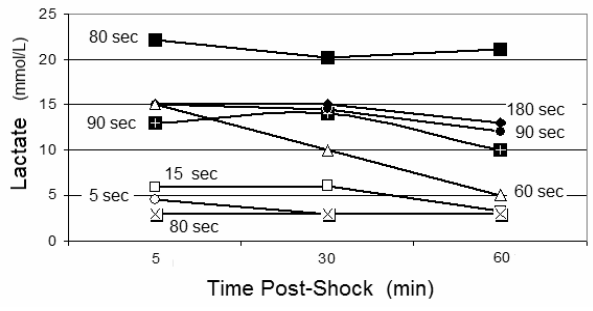


Fig. 2. Blood lactate concentration as a function of post-shock sampling time for specific NMI trials employing the MK63 (open square with x) or the X-26 (all other symbols). The data references are as follows: Solid square, 80 sec, X-26 [15]; triangle, 60 sec, X-26 [16]solid diamond, 180 sec, X-26 [16] solid circle, 90 sec, X-26 [16]; solid square with cross, 60 sec, X-26 [16]; open square, 15 sec, X-26 [17]; open circle, 5 sec, X-26 [16]; open square with x, 80 sec, Mk 63 [14]. Note that the results for the MK63 (65 Hz) show no increase in lactate over the 80 sec period in comparison with the X-26 (19 Hz) 80 sec time course studied using comparable protocols. The remaining time series data suggest increasing lactate as a function of time for the X-26. Note that lactate values for data in [16] exceeded 15 mmol/L lactate as discussed [16].

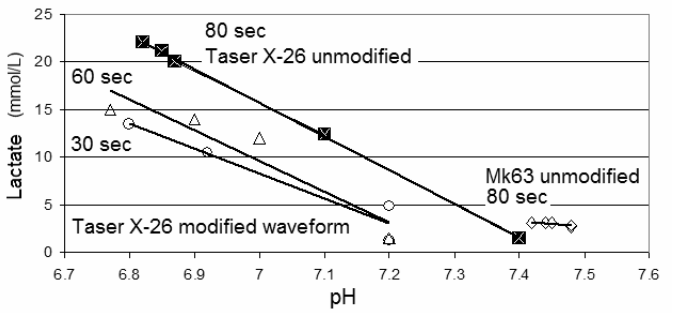
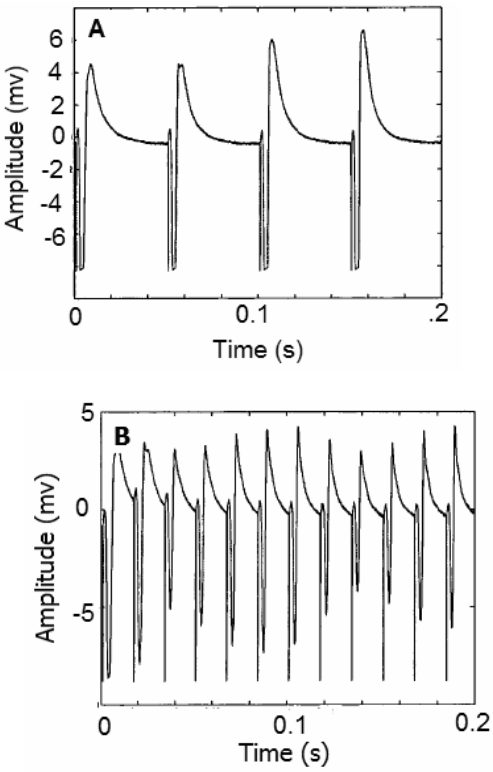


Fig 3. Blood pH versus lactate post-shock following published time courses immediately following shock to 60 minutes. Note the narrow range for both pH and lactate recorded for the MK63 [13]. The data for the unmodified Taser X-26 [15] and Taser X-26 modified waveform [18], [10] show a dose-dependent response for lactate with device discharge. Two animals died after exposure from acute onset ventricular fibrillation [15].



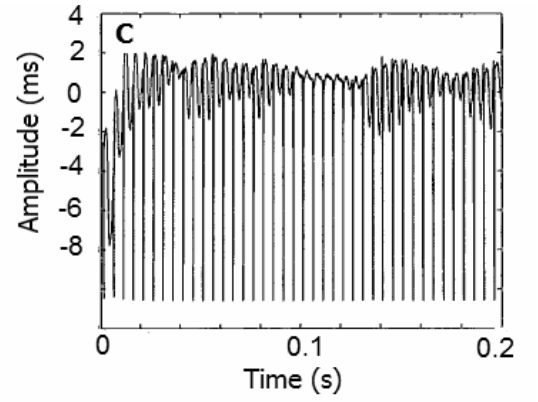


Fig. 4. Combined muscle action potential (CMAP) as a function of stimulation frequency adapted from [19]. Shown are electromyogram (EMG) recordings at stimulation frequencies of: 20 Hz (A), 60 Hz (B), and 200 Hz (C). The responses are inverted to account for electrode polarity and represent a portion of a 5 second record. Stimuli of specific frequency were generated using a square wave pulse generator (Grass S88) arising from stimulation of the rectus femoris muscle [19] 5 milliseconds after stimulation.

IV. DISCUSSION

Ex vivo waveforms have been described in the literature for the Taser X-26 [20] and the Mk63 [21]. Briefly, the Taser X-26 delivers DC pulses with a voltage of approximately 50kV, pulse duration of 140 microseconds, power of 0.36 J per pulse and a frequency of 19 Hz. The Mk63 delivers DC pulses with a voltage of approximately 19kV, pulse duration of 15 microseconds, power of 0.08 J per pulse and a frequency of 65 Hz. Both devices exhibit clear and strong muscle contractions, often in all four quadrants [14], [16]. The Taser X-26 discharge is primarily delivered subcutaneously with barbed darts of variable separation [22]. Drive stun [23], or direct contact of the fixed electrode configuration of the Taser gun without firing darts is widely reported [9]. The MK63 is a baton configuration in which the electrodes are fixed and blunt to avoid subcutaneous discharge [24].

The differences between the Taser X-26 and the MK63 produce clearly differentiated *in vivo* responses based on the data presented here. The Taser X-26 consistently elicits robust lactate production in a dose-dependent manner as shown in Figures 2 and 3 in contrast to the Mk63. Figure 2 shows clearly that for an 80 second discharge delivered under nearly identical conditions for both devices the Taser X-26 produces high lactate levels immediately post-discharge and throughout the 60 minute sampling period. However, the MK63 shows a small increase over control values that do not increase with exposure time. Taser X-26 discharges that range from 5 to 180 seconds (Figure 2) are consistent in the magnitude of response with time post-discharge as are data for brief discharges of 5 seconds on humans [25]-[27]. Figure 3 links lactate production to blood pH showing again that the Taser X-26 elicits an *in vivo* response clearly differentiated from the MK63 pattern in which pH and lactate are confined to narrow ranges judged to be clinically insignificant over periods as long as 80 seconds [13], [14]. Discharges from the X-26 reported in [15] resulted in two deaths immediately following the 80 second discharge from acute onset ventricular fibrillation.

The Taser X-26 data exhibit linear dose-dependent responses for a 60 minute time course. Data of [19] employing a modified X-26 waveform confirmed the characteristic *in vivo* response of high lactate and lowered blood pH of the X-26 overall waveform for stimulations of 30 and 60 seconds. The observed values for lactate and blood pH are consistent with previous data for un-modified X-26 discharge that also suggest the potential for lactate acidosis. The waveform modification consisted of removal of the leading arc phase intended to penetrate clothing in preparation for improved continuous-exposure electronic control devices [10]. Note that in cases of drive stun with the Taser the arc phase will be directly discharged to the skin, in fashion similar to that of the MK63. Thus, within the limitations of the data examined, the Taser X-26, discharged subcutaneously or directly on the skin appears to result in rapid and persistent increases in lactate and decreases in blood pH potentially resulting in NMI induced lactic acidosis (pH < 7.0) [15]. Further comparisons and study of humans are required for the MK63 to confirm the low lactate *in vivo* response.

The data presented here do not address *in vivo* responses to NMI stimulus related to cardiac complications [28], [29] or for individuals under the influence of alcohol, drugs or exhibiting signs of excited delirium [30]. The relationship between NMI and sudden in-custody death, often associated with excited delirium, is unknown and controversial [31]. However, a consistent feature of excited delirium cases involves violent, erratic behavior and struggle presumably initiating high levels of lactate production and acidosis among other complications such as hyperthermia and hyperventilation [32], [33]. Whilst establishing a cause and effect relationship between NMI deployment and in-custody deaths (e.g., excited delirium) appears to be an insuperable puzzle at present, the use of an NMI device with a minimal *in vivo* lactate impact is indicated. Within the limitations of the data presented the Mk63 shows a highly reduced potential for lactate production compared with the Taser X-26 while showing comparable effectiveness for global muscle tetany in animal studies [13]. Moreover, a fixed electrode configuration and the ability to direct the contact location of the electrodes, as in the case of the Mk63, offers additional flexibility in the use of force with at-risk individuals. Human studies with the Mk63 are required to confirm extrapolation of the animal data.

Related studies of cardiac response to the MK 63 and Taser X-26 discharges are of relevance in further establishing safety profiles for the devices. The Mk63 did not exhibit any significant effect on cardiac function in

studies conducted on swine [28] while the Taser X-26 discharges studied in a similar animal model and under similar conditions [29] invariably produced myocardial capture potentially resulting in fatal ventricular fibrillation as observed for one animal in the study. Studies for the MK63 to date have shown no clinical evidence of injurious changes in blood chemistry, neuromuscular damage or tissue damage [13], [14].

We note that the data presented in this work represent responses over a one hour period post-shock even though data for one or more days are available and invariably show a return to pre-shock values for the parameters studied [13] – [18]. We elected to focus on the one hour post shock period because the majority of deaths associated with 334 documented post NMI fatalities went into cardiac or respiratory arrest at the scene or suffered other complications at the scene that led to a fatality [9]. The period immediately following the NMI event is key to establishing the rapidly changing physiological responses to an NMI discharge.

The distinctly different patterns of *in vivo* lactate production between the Taser X-26 and the MK63 could be due to specific design features and/or discharge patterns as suggested in [13] and [34]. Notably, one consistent feature of each device regardless of electrical output, test conditions including resistive load variations and mode of application is the fundamental frequency or repetition rate with which the NMI signal is broadcast to the tissue. The X-26, as for most NMI devices [35], [36], operates with a 19 Hz frequency while the MK63 operates with a 65 Hz frequency [21].

We suggest that intense, repetitive, mechanical loading of the muscle during contractions can lead to dose-dependent lactate acidosis and that the magnitude of this response is mediated by the time interval between muscle contraction and relaxation over the period of NMI stimulation. Figure 4 (A,B,C) shows combined muscle action potential (CMAP) measured by electromyography (EMG) for muscles stimulated with frequencies of 20 Hz, 60 Hz and 200 Hz with constant voltage (60V), pulse duration (0.1 second) and stimulus of 5 seconds [19]. The panels of Figure 4 show clear differences in the cycle of contraction and relaxation for the three frequencies tested. In the case of 20 Hz stimulation the cycle includes a full relaxation interval prior to re-stimulation representing strong, repeated "kicks" of the leg over the 5 second interval for each stimulation cycle. The 60 Hz pattern demonstrates that the cycle is interrupted in not allowing a return to baseline, causing a full tetanic continuous contraction for the 5 seconds and lacking the repeated kick observed for the 20 Hz stimulation. The 200 Hz pattern is clearly erratic, poorly organized and does not represent sustained or repeated tetanic contractions. The stimulation frequency in these studies is related to CMAP and visually to muscle contraction. Thus, we conclude that the 60 Hz frequency results in a reduced interval of relaxation between activations as well as overall amplitude of the contractions. Reduced mechanical, repetitive loading may be consistent with reduced lactate production in general due to the reduction in contractile activity [37], [38]. The validity of the frequency/lactate link and associated biochemical mechanisms warrant further study to elucidate the mechanisms involved.

V. CONCLUSIONS

The effectiveness and safety of NMI devices depends on the *in vivo* response to the device waveform, duration of application and mode of contact. Differences in NMI device design can produce markedly different outcomes for physiology including lactate production and acidosis, changes in blood metabolites and alteration of cardiac function potentially resulting in death. Designers of NMI devices face challenges for optimal designs due to gaps in an understanding of the basis for injury and underlying mechanisms controlling neuromuscular function, such as the relationship between frequency and lactate production explored in this work. Policing with NMI devices presents an even greater challenge in addressing a highly variable population with unknown health and substance abuse status, particularly those exhibiting signs of excited delirium. Catalysts for device design improvements should integrate *in vivo* and *in silico* studies at the cellular and whole-body levels as described in [39] and [40]. Societal sentiments such as those expressed by minority groups [41] and civil rights advocates [9] should be adequately addressed. A "cells to society" approach offers a scientifically defensible and efficient means to develop an NMI repertoire of devices and use policies that are effective, safe across known at-risk groups and widely accepted in society.

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