

The author(s) shown below used Federal funds provided by the U.S. Department of Justice and prepared the following final report:

**Document Title: Respiratory and Cardiovascular Response
During Electronic Control Device (ECD)
Exposure in Law Enforcement Trainees**

**Author: Kirsten M. VanMeenen, Ph.D., Marc H. Lavietes,
M.D., Neil S. Cherniack, M.D., Michael T. Bergen,
M.S., Ronald Teichman, M.D., Richard J.
Servatius, Ph.D.**

Document No.: 236952

Date Received: December 2011

Award Number: 2005-IJ-CX-K065

This report has not been published by the U.S. Department of Justice. To provide better customer service, NCJRS has made this Federally-funded grant final report available electronically in addition to traditional paper copies.

**Opinions or points of view expressed are those
of the author(s) and do not necessarily reflect
the official position or policies of the U.S.
Department of Justice.**

Respiratory and Cardiovascular Response During Electronic Control Device (ECD) Exposure in Law Enforcement Trainees

Kirsten M. VanMeenen, PhD^{1,2}, Marc H. Lavietes, MD^{1,3}, Neil S. Cherniack, MD^{1,3}, Michael T. Bergen, MS^{1,2}, Ronald Teichman, MD⁴, Richard J. Servatius, PhD^{1,2}

¹Stress & Motivated Behavior Institute, ²Department of Neurology and Neurosciences, and ³Department of Medicine, University of Medicine and Dentistry, New Jersey Medical School, Newark, NJ USA, ⁴War-Related Illness and Injury Study Center, VA New Jersey Health Care System, East Orange, NJ

Keywords: respiration, heart rate, TASER X26[®]

This research was supported by the National Institute of Justice (NIJ) to RJS (grant #2005-IJ-CX-K065).

Corresponding author:

Kirsten M. VanMeenen, Ph.D.

University of Medicine and Dentistry of New Jersey, New Jersey Medical School

Department of Neurology and Neurosciences

Medical Science Building

185 South Orange Avenue H-506

Newark, NJ 07101 USA

Phone: (973) 972-5208

Author email:

Kirsten M. VanMeenen, Ph.D.

vanmeekm@umdnj.edu

Marc H. Lavietes, MD

lavietmh@umdnj.edu

Neil S. Cherniack, MD

Deceased

Michael T. Bergen, MS

Michael.bergen@umdnj.edu

Ronald Teichman, MD

Ronald.Teichman@va.gov

Richard J. Servatius, Ph.D.

servatrj@umdnj.edu

This project was supported by Award No. 2005-IJ-CX-K065 awarded by the National Institute of Justice, Office of Justice Programs, US Department of Justice. The opinions, findings, and conclusions or recommendations expressed in this publication/program/exhibition are those of the author(s) and do not necessarily reflect the views of the Department of Justice.

Abstract

Objective: Law enforcement represents a large population of workers who may be exposed to electronic control devices (ECDs). Little is known about the potential effect of exposure to these devices on respiration or cardiovascular response during application.

Methods: Participants ($N=23$) were trainees exposed to ECDs as a component of training.

Trainees were asked to voluntarily inhale during exposure. **Results:** The exposure period resulted in the cessation of normal breathing patterns in all participants and in particular a decrease in inspiratory activity. No significant changes in heart rate during

ECD exposure were found. **Conclusions:** This is the first study to examine breathing patterns during-ECD exposure with the resolution to detect changes. In contrast to

reports suggesting respiration is unaffected by ECDs, present evidence suggests that voluntary inspiration is severely compromised. There is no evidence of cardiac disruption during-ECD exposure.

INTRODUCTION

Electronic control devices (ECDs) are widely used both nationally and internationally in law enforcement as an alternative to lethal force. Many law enforcement trainees volunteer to be exposed to ECDs during the course of their training. Voluntary exposure takes place in the face of questions regarding the extent of the physiological impact of ECD exposure.

Little is known about the impact of ECD exposure on respiration. The animal literature is not particularly informative in this regard. Sedation, intubation, and ventilation of swine preclude reasonable assessment of the impact of electrical stimulation on respiration. Three current reports in humans concluded that ECD exposure does not significantly affect respiration. ECD exposure lasts seconds, typically 5-seconds, but devices now are extending to 30-seconds. Vilke and colleagues (Vilke et al., 2007) reported respiration in terms of rate, minute ventilation and tidal volume sampled over several minutes. Thus, the observation of increased ventilation after ECD exposure was insensitive to the period of exposure. Ho and colleagues (Ho et al., 2007) evaluated tidal volume, end-tidal O₂ and end-tidal CO₂ on a breath-by-breath basis prior to exposure, during exposure and after ECD exposures in trainees. Two types of exposures were examined: a single continuous 15-second exposure and a routine of three 5-second exposures separated by 1-second for a total duration of 17-seconds. For both types of exposure tidal volume did not differ between the period preceding exposure and during exposure. Volumes of expired O₂ and CO₂ also did not differ. Ventilation rates increased slightly for both types of exposure. Dawes and colleagues (Dawes et al., 2010a) also examined respiration rate, tidal volume, and minute volume using a breath-by-breathe analyzer for ECD exposures that were either two or three simultaneous 5-

second exposures. The breath-by-breath analyzer that was implemented has the same limitations as the aforementioned study (Ho et al., 2007) with respect to resolution and synchronization with ECD exposure. That said, no statistically different differences in respiratory measures were found. Thus, the available published data suggest that ECD exposures have minimal impact on respiration.

Nonetheless, recent observations suggest that some respiratory mechanisms may be momentarily impaired by the electrical discharge. Anecdotal reports from law enforcement officers during an earlier study from our laboratory suggest that respiration, particularly the ability to inspire, is severely affected in some trainees. A self-report measure of breathing ability in 90 law enforcement trainees confirmed that 20% of those who attempted to inspire during exposure reported that they were unable to do so. Further, this percentage of self-reported inability to breathe remained constant when an additional 90 officers were expressly aware that questions concerning ability to breathe were of interest. These data raise questions regarding an individuals' ability to normally inspire during a 5-second ECD exposure.

To date, investigations on the health impact of ECD exposure have focused primarily on the potential for adverse cardiovascular outcomes (Dawes et al., 2010a; Dawes et al., 2010b; Dennis et al., 2007; Ho et al., 2006; Ho et al., 2007; Ho et al., 2008; Levine et al., 2007; Sloane et al., 2008; VanMeenen et al., 2010; Vilke et al., 2007; Vilke et al., 2008; Walter et al., 2008; Wu et al., 2007). Although direct evaluation of the cardiac activity during ECD exposure is technically challenging (i.e., obtaining direct measures of electrical activity of the heart (e.g., electrocardiogram) during exposure is made impractical given the electrical energy of the discharge of ECDs), the available evidence suggests that the ECDs have only transient effects on normal healthy hearts. A number

of studies have assessed 12-lead electrocardiograms (ECGs) for morphology and found little impact from baseline to immediately post exposure (Vilke et al., 2008) and 24-hours post exposure (Ho et al., 2006), although there is suggestive evidence that exposure to ECD in those with pre-existing 12-lead abnormalities worsen (VanMeenen et al., 2010). Currently, the cardiovascular impact of the ECD during its application has been confined to a few studies using echocardiography (Dawes et al., 2010a; Dawes et al., 2010b; Ho et al., 2008). Advances in pulse oximetry offered the promise of obtaining heart rate from a measure unaffected by the ECD itself. The current study is one of the first to capture cardiovascular response during ECD exposure and the first to use this particular method.

The present study examines the potential impact of exposure to ECD upon respiratory and cardiovascular responses. Using pulse oximetry from which heart rate was calculated we were able to examine cardiovascular response during ECD exposure. Both inspiratory flow and expiratory flow were measured continuously before, during, and after ECD exposure. To increase the likelihood that an inspiration occurred during the relatively brief period of exposure, all participants were asked to sniff (volitional inhalation) during the 5-second exposure period. The sniff maneuver is often used to examine diaphragmatic muscle strength in various populations (Koulouris et al., 1989b; Koulouris et al., 1989a; Stefanutti et al., 2000) and was used to ensure that trainees exhibited breathing activity (particularly inhalation) during the brief period of ECD exposure.

MATERIALS AND METHODS

Recruitment

Law enforcement agencies across the country conducting ECD training were recruited through direct mailings with an IRB approved letter. From these correspondence two training facilities (one in Virginia and one in New Mexico) agreed to participate. Only trainees officially enrolled in the class, and who had agreed to receive ECD exposure as a component of their training, were further recruited for the present study. We did not exclude participants for any reason. Although all participants were eligible for compensation (\$50) for their participation, one of the two departments disallowed compensation. Therefore, 10 (43%) participants received monetary compensation. The research described in this paper was approved by the Institutional Review Board (IRB) at the University of Medicine and Dentistry, New Jersey Medical School.

Summary

Participants completed demographic and health history questionnaires after we obtained informed consent. In addition, as a means of examining volitional inhalation, participants were asked to sniff through the nostrils prior to ECD exposure, during ECD exposure, and shortly after ECD exposure. Participants stood on a mat with two spotters to prevent injuries due to falls. During the exposure, participants were held upright by spotters. Participants were either equipped with alligator clips on their clothing or were shot to their back from the instructor. The location of the darts or clips on the body was documented. Respiration was measured in two systems, through changes in flow and temperature, continuously for 20-seconds before the exposure, through the exposure and

for 20-seconds post exposure. Pulse oximetry was continuously measured during this same period of time. Self-report measures were taken immediately post exposure.

Demographics

Demographics and participant characteristics can be found in Table I. Health history data for participants are shown in Table II. Of 25 people who participated in data collection at these two sites, two were excluded due to system failure resulting in ECD exposures less than 2 seconds.

Exposure

Both sites used the X26[®]. All of these trainees included in the analyses received the full 5-second discharge. The method of transmission varied: 17 (74%) participants received the discharge via darts and 6 (26%) received the discharge via clips applied to clothing. Although the location on the body for exposure varied, all exposures took place on the back of the body. Previous exposure to ECD was reported by 3 participants (13%).

Measures

Respiratory Flow

Subjects were fitted with a mask (RT040M, Fisher & Paykel Healthcare) that covered the mouth and nose. A 32" hose with an inner diameter of 3/4" was connected to the port. Flow was measured with a pneumotach (model 3813, Hans Rudolph, Kansas City, MO) connected in-line with the hose and mounted on a tripod. Data were collected using a 12-bit analog to digital converter (PCMCIA6024E, National Instruments, Austin, TX)

at a sampling rate of 1000Hz to a notebook computer using a custom LabVIEW (National Instruments, Austin, TX) program for display and storage. The pneumotach waveform traces were 15Hz low-pass filtered to removed 18Hz noise, a characteristic of the X26[®] that was evident during the exposure period. The threshold for the calculation of flow was set at +/- 50 mL/s, the minimum sensitivity of the system. Respiratory flow (L/S) was recorded before, during, and after ECD exposure. See Figure 1 and Figure 2 for examples of respiratory flow for individual subjects. A thermocouple was placed inside the tube at the port of the mask and measured the temperature inside the hose.

Inspection of the data indicated that breathing patterns were severely disrupted not allowing for traditional calculation of tidal volume during ECD exposure. In order to examine observed differences in volumes of the expiratory and inspiratory traces, volumes were separately obtained for all movements in the inspiratory and expiratory direction during the 5-second exposure. To facilitate comparison, sums of inspiratory and expiratory volumes were calculated for a similar 5-second epoch immediately preceding ECD exposure and the 5-second epoch after ECD exposure.

Tidal volume was also calculated from the last definable breath prior to ECD onset and the first definable breath post-ECD onset. Further, from the last full breath (FB) the inspiratory volume (FB_I) and the expiratory volume (FB_E) were calculated.

Pulse Oximetry

An optical pulse plethysmography (PPG) sensor (Xpod Nonin Medical, Inc Plymouth, MN) was clipped to the subject's right ear lobe. The PPG waveform obtained from the sensor was processed using a custom peak detector Splus programming language (TIBCO Somerville, MA) for the identification of inter-pulse interval (IPI). Of

the 23 participants who had respiratory data, 15 also had heart rate data. IPI data was not able to be obtained on eight participants due to motion artifact.

Our goal was to compare heart rate prior to ECD exposure to heart rates during and after ECD exposure. ECD exposure produced a significant artifact in the flow signal such that a dramatic positive flow was induced soon after ECD onset and a negative flow corresponding to ECD cessation. This was observed in all subjects. These two artifacts represented regions of exclusion of the calculations heart rate. However, the durations of these regions of exclusion differed for each subject. The periods of interest for each subject were determined by the amount of artifact-free time available during ECD exposure, that duration was then measured in the pre-ECD and post-ECD period. See Figure 3 for a representative example of blood flow and corresponding heart rate. IPIs were converted into heart rate (bpm) for each of these three time windows (Graham, 1978). Oxygen saturation was derived from the measure of pulse oximetry however, due to the narrow window of time of interest in the current report and the slow time course of the signal these data will not be reported.

Body Mass Index (BMI) and Body Fat Percentage

Body fat percentage was obtained just prior to exposure using a body fat monitor (Model HBF-306C) from Omron Healthcare, Inc. (Bannockburn, IL) and height and weight were used to calculate BMI. Classifications were then made based upon standards from the World Health Organization (2001).

Volitional Breathing and Health History

To understand the drive to breathe, trainees completed a short Breathing Survey immediately after ECD exposure. The survey asked whether they tried to breathe during the ECD exposure (yes/no/unsure) and if so, whether they were successful (yes/no/unsure). Trainees also reported whether or not they held their breath during the ECD exposure (yes/no/unsure). Lastly, trainees were asked if they tried to sniff during ECD exposure (yes/no/unsure), and if so, whether they were successful (yes/no/unsure). During the consent process trainees were shown the Breathing Survey and were reminded prior to exposure that their ability to recall breathing and their attempt to sniff during exposure was of interest to the investigators.

Participants completed a Health History form which asked specifically about cardiovascular or pulmonary problems and a list of any medications that they were currently taking (See Table II).

Exposure Location

The location of the darts or clips on the body was also documented. Exposure types were categorized into two groups: 1) Participants whose exposure locations were both above the bottom of the spine and 2) Participants whose exposure locations spanned the bottom of the spine. See Figure 4 for a representative example of each of these types of exposures.

RESULTS

Volitional Breathing

As can be seen in Table III, most trainees could recall either in the positive or negative their ability to breathe during ECD exposure. Only two of the 23 participants reported that they did not attempt to breathe during the 5-second exposure (and one of these also reported that they held their breath). Both records showed an absence of inhalation with minor volumes of exhalation during ECD exposure. Inasmuch as the present study is focused on the ability to breathe, and these two subjects self-reported that they either did not try to breathe or actively held their breath, we excluded their data from further analysis. Although three trainees reported that they were unsure of whether they tried to breathe, these were included in the analysis insofar as exposure to ECD may alter the ability to recall events during exposure.

A relatively high proportion of the trainees (44%) complied with our request to try to sniff during ECD exposure. Not one reported success at being able to sniff during ECD exposure. The absence of waveforms characteristic of sniffing in all those trainees confirmed that sniffing did not occur. However, wave forms characteristic of sniffing were apparent in two flow records which were also confirmed in temperature records (See Figure 5). For these two subjects, one was unsure of whether they tried to sniff and one tried to sniff but was unsure of their success. Clearly, volitional breathing was difficult during the 5-second ECD exposure.

Inspiratory and Expiratory Volumes

The volumes for inspiration and expiration at the three times points were analyzed with a two-way repeated measures analysis of variance (ANOVA). Total volumes for

inspiration and expiration did not differ, $F(1,19) = 1.61$ $p > .10$. The main effect of time, $F(2,38) = 43.03$, was qualified by the Respiration x Time interaction, $F(2,38) = 15.42$, all $ps < .001$). Post hoc analyses were accomplished with Dunn's Multiple Comparisons test. Inspiratory and expiratory volumes did not differ during the pre-ECD period $t_D = 1.8$, $p > .05$ (See Figure 6). For both inspiration ($t_D = 8.3$) and expiration ($t_D = 3.3$) there was a significant decrease in volumes during ECD exposure, with a concomitant increase in the post-ECD period to volumes greater than pre-ECD ($t_D = 15.8$ and $t_D = 7.9$, respectively). Moreover, the inspiratory volume during ECD was lower than the expiratory volume ($t_D = 3.8$), which reversed during the post-ECD period ($t_D = 4.1$).

Individual differences in inspiratory and expiratory volume over these periods of time were also examined. Figure 7 shows that for the vast majority of subjects there was a dramatic decrease in inspiratory volume from pre-ECD to during ECD exposure and a greater increase from during ECD to post-ECD. To facilitate comparisons between individuals given the wide range of volumes as a function of time, inspiratory and expiratory activity during ECD and after ECD exposure was compared relative to the FB_I and FB_E , respectively (See Figure 8). As can be seen, relatively few trainees exhibited inspiratory volumes comparable to their pre-ECD values; not one exhibited levels of inhalation exceeding pre-ECD levels. Only four trainees exhibited inhalation greater than 25% of the pre-ECD levels. Inspection of their data indicated that 3 of the 4 received ECD through darts with one of the darts penetrating below the spinal level (e.g., buttocks). Only one other trainee received ECD in this manner, that trainees' inhalation levels resembled the others with two darts (or clips) at spinal levels. Exhalation was much less restricted; all but four of the trainees have expiratory volumes which exceeded 25% with three participants exhibiting exhalation exceeding pre-ECD levels. The

location of ECD did not distinguish levels of exhalation. Note that inhalation levels were not in balance with exhalation levels during ECD exposure. In the aftermath of ECD exposure, for all but two trainees, inhalation exceeded pre-ECD levels. One of these trainees increased from 4% to 7% whereas the other increased from 0.2% to 37% of pre-ECD inhalation, this latter trainee had exhalation values with exceeded pre-ECD exhalation. Three additional trainees exhibited expiration levels lower than their pre-ECD levels and also had expiration levels substantially lower during ECD exposure (all < 40%). Overall, the relationship between relative inhalation and exhalation after ECD exposure is roughly linear ($r^2 = 0.58$). Although a marked recovery was evident in inhalation and exhalation immediately upon ECD cessation, one trainee exhibited a similar degree of respiratory disruption during the 5 second period after ECD cessation.

To understand whether participant characteristics (age, height, weight, BMI or body fat values) or exposure locations modified or explained the volume findings, these characteristics were included in stepwise fashion to the model. None of the characteristics provided explanatory power.

Heart Rate

Heart rate over the three time points was analyzed with a repeated measures analysis of variance (ANOVA). To understand whether the participant characteristic of BMI might interact with heart rate, low BMI (< 25) and high BMI (> 25) groupings were included as a between-subjects factor in the model. There was a significant main effect of Time, $F(2,26) = 20.57, p < .001$. No Time x BMI interaction was found ($p > .10$). Post hoc analyses were accomplished with Bonferroni procedures. There was a significant increase in heart rate from both the pre-ECD period and the during-ECD period to the

post-ECD period ($p < .0005$ for each). However, no significant change in heart rate was found between the pre-ECD period and the during-ECD period ($p > .10$). Mean heart rate values and standard deviations for the pre, during, and post-ECD periods were 110 bpm (15.9), 100 bpm (18.5), and 121 bpm (13.0), respectively.

DISCUSSION

The primary goal of the current study was to examine breathing in law enforcement trainees during ECD exposure. A difficulty in understanding breathing during ECD exposure is that the period of exposure is extremely brief compared to the normal rate of breathing; one would expect little respiratory activity during the 5-second exposure period. To ensure that at least one breath was taken during ECD exposure, trainees were encouraged and prompted to breathe. Self-report responses of breathing show that 78% of trainees attempted to breathe during ECD exposure. Self-report was verified by flow measured by pneumotach with complimentary temperature changes derived from a thermistor. Although the trainees were actively trying to breathe, most trainees in the present study showed a cessation of breathing (i.e., absence of any orderly tidal breathing). As can be seen in Figures 6 and 7, the inspiratory flow approached zero during the ECD exposure. Expiratory flow also severely decreased. Sound recordings indicated that many of those trainees with significant expiratory flow were also vocalizing. Both inspiratory and expiratory flow recovered immediately upon the cessation of the ECD; these post-ECD volumes significantly exceeded corresponding flows measured immediately prior to ECD exposure. This respiratory disruption does not appear to be related to the volume of air in the lungs at the time of exposure. The flow

records of those who were either unsure of whether they breathed or simply did not try were otherwise indistinguishable from those that reported trying.

Normally, inspiratory and expiratory flows are in balance. As can be seen in Figure 8, inspiratory and expiratory flows were not related during ECD exposure; inspiratory and expiratory flow returned to a largely linear relationship immediately post-ECD exposure. Inspection of the flow activity during ECD exposure identified four subjects with inspiratory flow greater than 20% of their inspiratory flow prior to ECD exposure. The greater inspiratory activity of these trainees may be related to the location of ECD transmission to the body (See Figure 4). Although the transmission of ECD was predominantly from both electrodes above the bottom of the spine, for 4 trainees transmission spanned the upper and lower torso. Of these 4 trainees, three exhibited inspiratory volumes greater than 25% of their respective pre-ECD volumes. Additionally, one trainee whose electrodes were both in the upper torso exhibited inspiratory volumes that exceeded 25% over their pre-ECD volume. Dart or clip locations are not controlled in the occupational setting; this observation would need more systemic study to understand its relevance.

Further, trainees were additionally asked to inhale by sniffing during ECD exposure. Sniffing can be involuntary or voluntary and is characterized by a short, abrupt rise in inspiratory flow. The behavior is readily understood and easily executed by otherwise healthy individuals; sniff behavior was recorded in all trainees prior to ECD exposure. Only 44% of trainees recalled trying to sniff during exposure, 22% did not try, with the remaining 34% unsure of whether they tried. These data suggest that the ability to recall whether one sniffed or tried to sniff is compromised during ECD exposure. Of only two trainees exhibiting sniff behavior during ECD exposure, one was unsure whether they

were successful and the other was unsure if they tried to sniff at all. Thus, the majority of those reported trying to sniff were unable to do so. These data strongly suggest that voluntary breathing, particularly inspiration, is severely compromised during ECD exposure.

Interruption of breathing may occur at several levels, from brain stem and cerebellum, to the spinal cord, to the muscles of respiration. In nearly all cases, disruption ends with the cessation of the current. Determining the level of disruption in humans will be a daunting task. For practical purposes, these periods resembled periods of severe hypopnea, if not apnea. Of course, 5 seconds of apnea are not likely to be of clinical consequence to anyone other than a severely respiratory - impaired patient. Newer models of ECDs, however, are designed to deliver up to 30 seconds of exposure. An enforced breath hold of 30 seconds, however, could be of great clinical consequence to patients with moderate respiratory impairment. Therefore the issue of respiration during periods of ECD exposure requires further investigation.

The changes in respiration noted herein are in stark contrast with the only other published reports measuring respiration during ECD exposure. For Ho et al. (Ho et al., 2007) ECD exposure did not appreciably affect respiration as measured by tidal volume, respiratory rate, and blood gases. Moreover, respiration did not appreciable change in the post-ECD period. For Dawes et al. (Dawes et al., 2010a) respiration measured by respiratory rate, tidal volume, and minute volume were not significantly affected. There are numerous differences between these two studies: length of exposure, number of exposures, postural position during ECD exposure, and means and location of ECD transmission to the body.

The second goal of the current study was to examine cardiovascular responses in law enforcement during ECD exposure. With the number of adverse events occurring proximal to ECD exposure, concern has been raised on the direct impact of ECD exposure on the cardiac cycle. However, the period during ECD has been largely unquantified, in part because the electrical energy of the device impedes direct collection of cardiovascular response data using traditional methods. This is one of the first studies to capture cardiovascular response during an ECD application. Although portions of the pulse oximetry signal displayed a significant artifact (approximately 2 seconds lost from the onset of ECD and 2 seconds lost with the cessation of ECD), there is no evidence of cardiac disruption. Calculated heart rates varied well within the physiological ranges, with no evidence of missed beats. These data confirm previous work showing ECD exposure does not appear to interfere with normal cardiac cycles in otherwise healthy law enforcement officers (Dawes et al., 2010a; Dawes et al., 2010b; Ho et al., 2008).

Limitations

Parameters of ECD exposure (location, duration, number of exposures, time of day, and body position at time of exposure) were all under the purview of the trainers. The data collected was not intended to be used as expected values for field exposure. Data were collected on a relatively small number of participants.

CONCLUSION

The results show an absence of inspiratory movement during ECD exposure. Normal breathing resumed after the cessation of the ECD exposure. This is the first study to examine respiratory effects of ECD exposure in trainees who specifically attempted to

inhale during the exposure. The results also confirm previous work showing ECD exposure does not appear to interfere with normal cardiac cycles in otherwise healthy law enforcement officers.

ACKNOWLEDGMENTS

We would like to thank the law enforcement communities that allowed us to recruit participants at their facilities as well as the trainees who participated in this study. We would also like to thank Kunal Doshi for his assistance.

Reference List

Obesity: preventing and managing the global epidemic. Report of a WHO consultation (2001). *World Health Organization Technical Report Series, 894*, i-xii.

Dawes, D. M., Ho, J. D., Reardon, R. F., Sweeney, J. D., & Miner, J. R. (2010a). The physiologic effects of multiple simultaneous electronic control device discharges. *The Western Journal of Emergency Medicine, 11*, 49-56.

Dawes, D. M., Ho, J. D., Reardon, R. F., & Miner, J. R. (2010b). Echocardiographic evaluation of TASER X26 probe deployment into the chests of human volunteers. *The American Journal of Emergency Medicine, 28*, 49-55.

Dennis, A. J., Valentino, D. J., Walter, R. J., Nagy, K. K., Winners, J., Bokhari, F. et al. (2007). Acute effects of TASER X26 discharges in a swine model. *J.Trauma*, *63*, 581-590.

Graham, F. K. (1978). Constraints on measuring heart rate and period sequentially through real and cardiac time. *Psychophysiology*, *15*, 492-495.

Ho, J. D., Dawes, D. M., Bultman, L. L., Thacker, J. L., Skinner, L. D., Bahr, J. M. et al. (2007). Respiratory effect of prolonged electrical weapon application on human volunteers. *Acad.Emerg.Med.*, *14*, 197-201.

Ho, J. D., Dawes, D. M., Reardon, R. F., Lapine, A. L., Dolan, B. J., Lundin, E. J. et al. (2008). Echocardiographic Evaluation of a TASER-X26 Application in the Ideal Human Cardiac Axis. *Acad.Emerg.Med.*, 838-844.

Ho, J. D., Miner, J. R., Lakireddy, D. R., Bultman, L. L., & Heegaard, W. G. (2006). Cardiovascular and physiologic effects of conducted electrical weapon discharge in resting adults. *Acad.Emerg.Med.*, *13*, 589-595.

Koulouris, N., Mulvey, D. A., Laroche, C. M., Sawicka, E. H., Green, M., & Moxham, J. (1989a). The measurement of inspiratory muscle strength by sniff esophageal, nasopharyngeal, and mouth pressures. *American Review of Respiratory Disease*, *139*, 641-646.

Koulouris, N., Vianna, L. G., Mulvey, D. A., Green, M., & Moxham, J. (1989b). Maximal relaxation rates of esophageal, nose, and mouth pressures during a sniff reflect inspiratory muscle fatigue. *American Review of Respiratory Disease*, *139*, 1213-1217.

- Levine, S. D., Sloane, C. M., Chan, T. C., Dunford, J. V., & Vilke, G. M. (2007). Cardiac monitoring of human subjects exposed to the taser. *J.Emerg.Med.*, 33, 113-117.
- Sloane, C. M., Chan, T. C., Levine, S. D., Dunford, J. V., Neuman, T., & Vilke, G. M. (2008). Serum troponin I measurement of subjects exposed to the Taser X-26. *J.Emerg.Med.*, 35, 29-32.
- Stefanutti, D., Benoist, M. R., Scheinmann, P., Chaussain, M., & Fitting, J. W. (2000). Usefulness of sniff nasal pressure in patients with neuromuscular or skeletal disorders. *American Journal of Respiratory & Critical Care Medicine*, 162, t-11.
- VanMeenen, K. M., Cherniack, N. S., Bergen, M. T., Gleason, L. A., Teichman, R., & Servatius, R. J. (2010). Cardiovascular evaluation of electronic control device exposure in law enforcement trainees: a multisite study. *Journal of Occupational & Environmental Medicine*, 52, 197-201.
- Vilke, G. M., Sloane, C., Levine, S., Neuman, T., Castillo, E., & Chan, T. C. (2008). Twelve-lead electrocardiogram monitoring of subjects before and after voluntary exposure to the Taser X26. *Am.J.Emerg.Med.*, 26, 1-4.
- Vilke, G. M., Sloane, C. M., Bouton, K. D., Kolkhorst, F. W., Levine, S. D., Neuman, T. S. et al. (2007). Physiological effects of a conducted electrical weapon on human subjects. *Ann.Emerg.Med.*, 50, 569-575.
- Walter, R. J., Dennis, A. J., Valentino, D. J., Margeta, B., Nagy, K. K., Bokhari, F. et al. (2008). TASER X26 discharges in swine produce potentially fatal ventricular arrhythmias. *Acad.Emerg.Med.*, 15, 66-73.

Wu, J. Y., Sun, H., O'Rourke, A. P., Huebner, S., Rahko, P. S., Will, J. A. et al. (2007).

Taser dart-to-heart distance that causes ventricular fibrillation in pigs. *IEEE*

Trans.Biomed.Eng, 54, 503-508.

TABLE I. Demographics and Participant Characteristics (*N*=23)

	Mean (SD)	Range
Age (years)	27.7 (5.6)	21 - 40
Height (in)	69.9 (3.4)	65-76
Weight (lbs)	187.3 (26.4)	150-250
BMI (kg/m ²)	26.9 (2.7)	22.9 – 32.6
Body Fat (%)	16.5 (5.1)	8.8 – 27.7
	N (%)	
Sex	23 males (100%)	
BMI classification		
Underweight (<18.5)	0 (0%)	
Normal (18.5 – 24.9)	7(30%)	
Overweight (25 – 29.9)	13(57%)	
Obese (≥ 30)	3(13%)	
Body Fat classification		
Low	0 (0%)	
Normal	17 (72%)	
High	5 (24%)	
Very High	1 (4%)	
Race†		
Caucasian	13 (57%)	
African American	1 (4%)	
Hispanic/Latino	8 (35%)	

†n=1 race unavailable

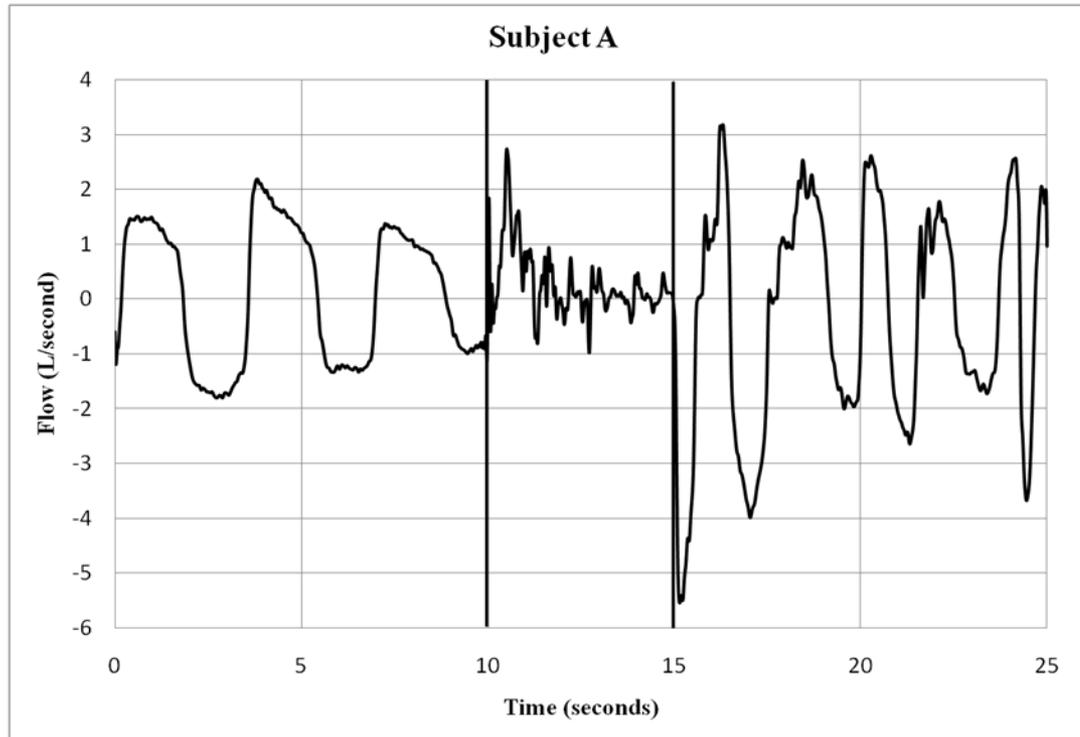
TABLE II. Participant Health History (*N*=23)

Health History	N (%)	Description
Respiratory problems	n=3 (13%)	Asthma (n=2), pneumonia (n=1)
High blood pressure	n=0 (0%)	
Cardiovascular problems	n=1 (%)	Heart murmur (n=1)
Other disease	n=0 (0%)	
Cardiovascular Medications (e.g., digitalis, anti-hypertensives, lipid lowering)	n=1 (4%)	Lisinopril (n=1)
Respiratory medications (e.g., short-acting beta-2 agonists, leukotriene modifiers)	n=0 (0%)	
Current tobacco use	n=2 (9%)	
Current alcohol use	n=18(78%)	

TABLE III. Self-Report of Volitional Respiration ($N=23$)

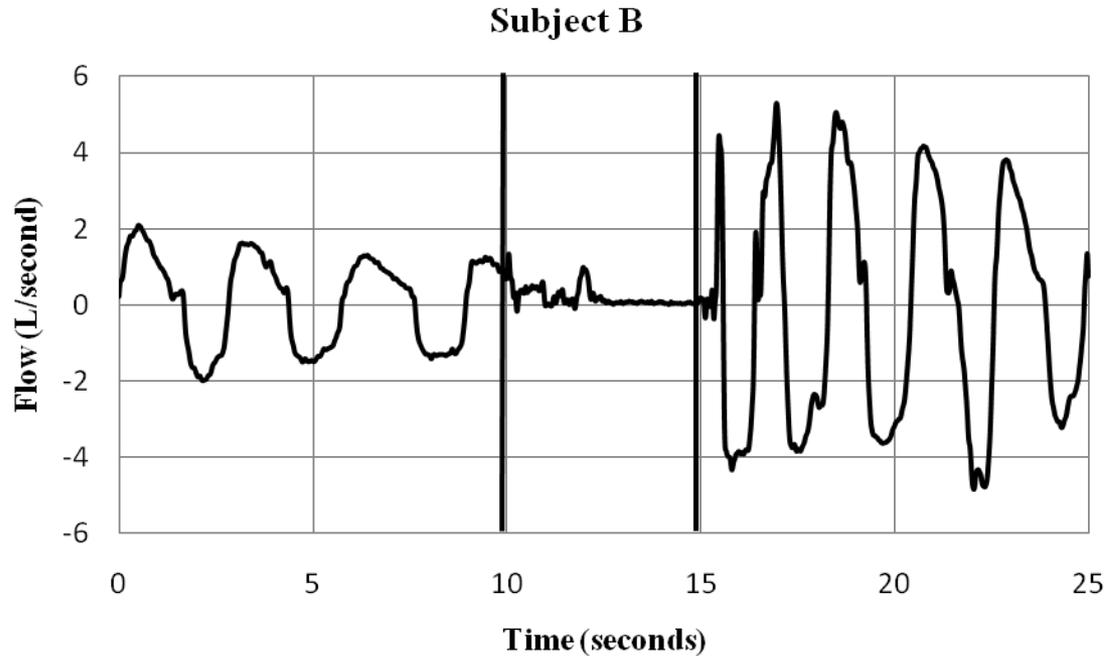
Self-Report of Volitional Respiration	yes	no	unsure
Did you try to breathe?	18 (78%)	2 (9%)	3 (13%)
If yes, were you successful?	5 (28%)	7 (39%)	6 (33%)
Did you try to sniff?	10 (44%)	5(22%)	8 (34%)
If yes, were you successful?	0 (0%)	7 (70%)	3 (30%)
Did you hold your breath?	1 (5%)	15 (65%)	7 (30%)

FIGURE 1. Subject A - Respiratory Flow Pre, During and Post-ECD Exposure



Subject A is an example of a participant who showed both inspiratory and expiratory activity during the ECD exposure period. Only a few of the subjects showed this pattern of respiratory activity during ECD exposure. The vertical lines indicate the onset and offset of ECD exposure.

FIGURE 2. Subject B - Respiratory Flow Pre, During and Post-ECD Exposure



Subject B is an example of a participant who showed a substantial inspiratory volume displacement from the pre-ECD to the exposure period. Most subjects showed this pattern of little or no inspiratory activity during the 5-second exposure. The vertical lines indicate the onset and offset of ECD exposure.

FIGURE 3a. Subject C - Blood Flow Pre, During, and Post-ECD Exposure

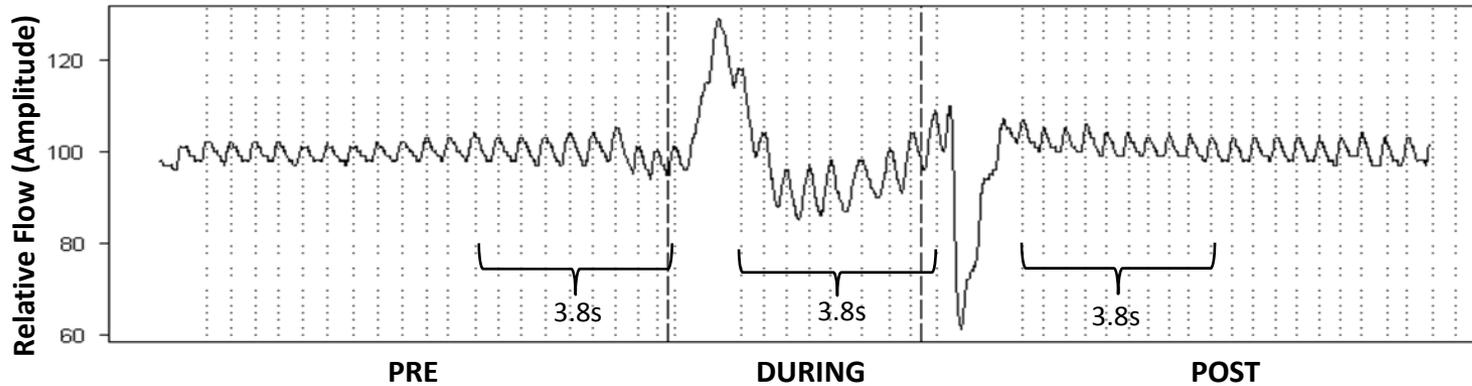
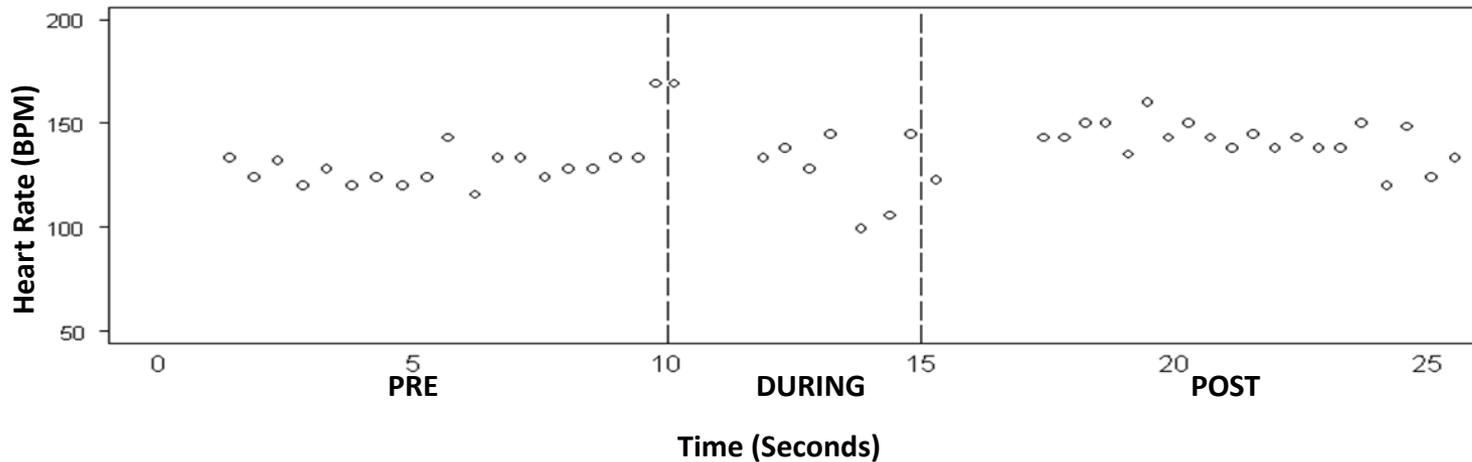


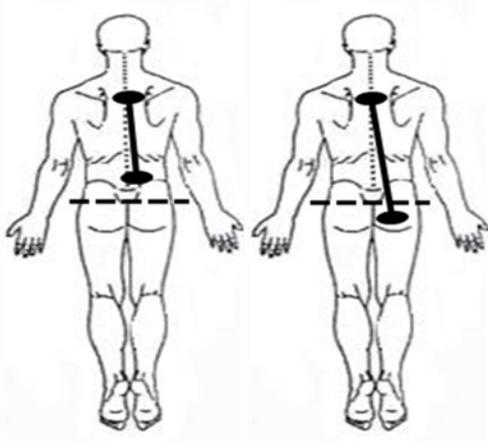
FIGURE 3b. Subject C - Heart Rate Pre, During, and Post-ECD Exposure



Blood flow (Figure 3a): Example of the processed PPG waveform for 10-s prior to ECD exposure, through the 5-s exposure, and for 10-s following exposure. Note that a dramatic flow artifact is evident immediately upon ECD exposure and immediately upon the cessation of exposure; this artifact is evident to a similar degree in all participants.

Heart rate (Figure 3b): Despite the artifact noted above, heart rate was reliably obtained for approximately the last 3-s of ECD exposure. This same time window was applied to the time immediately preceding ECD exposure and the same time epoch immediately following after dissipation of the post-ECD artifact.

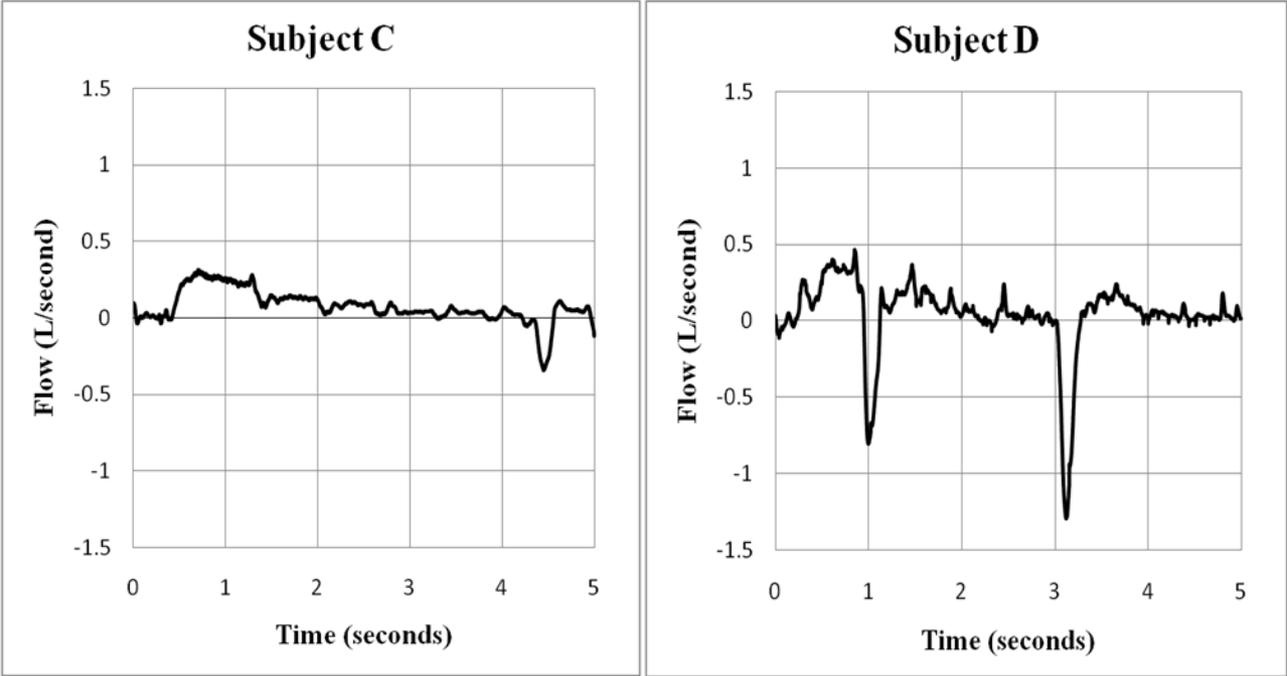
FIGURE 4: Example of ECD Exposure Locations



Example 1

Example 2

FIGURE 5. Evidence of Sniff Behavior During-ECD Exposure



Subjects C and D showed evidence of sniff behavior during the ECD exposure period. Subject C self-reported trying to sniff during exposure, but was unsure of their success. Subject D was unsure in self-report about trying to sniff during exposure.

FIGURE 6: Inspiratory and Expiratory Volume

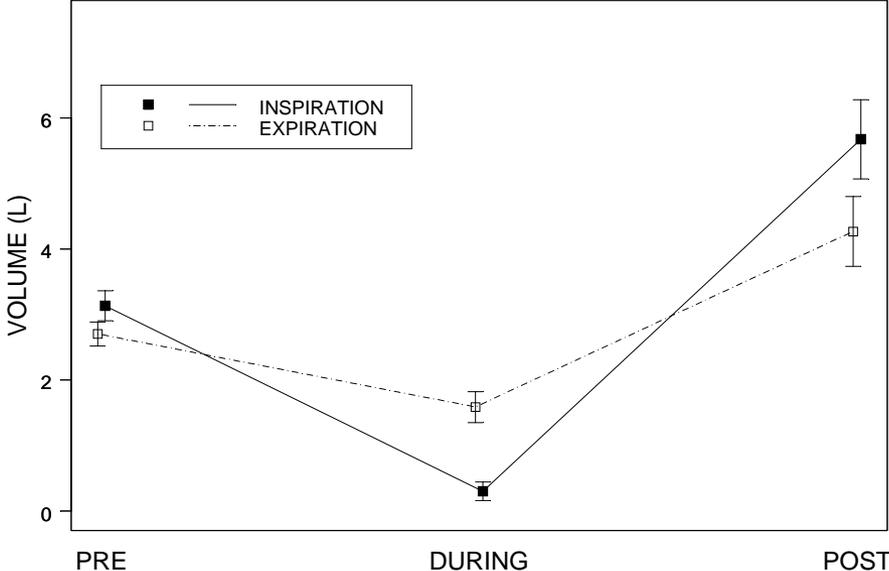


FIGURE 7: Individual Differences in Inspiratory and Expiratory Volume

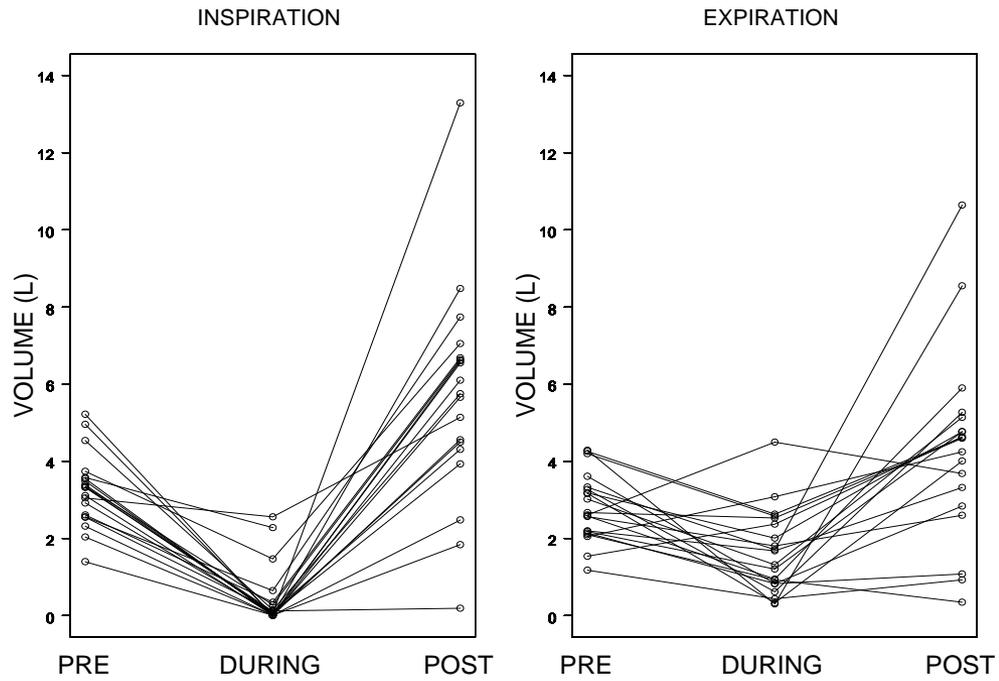
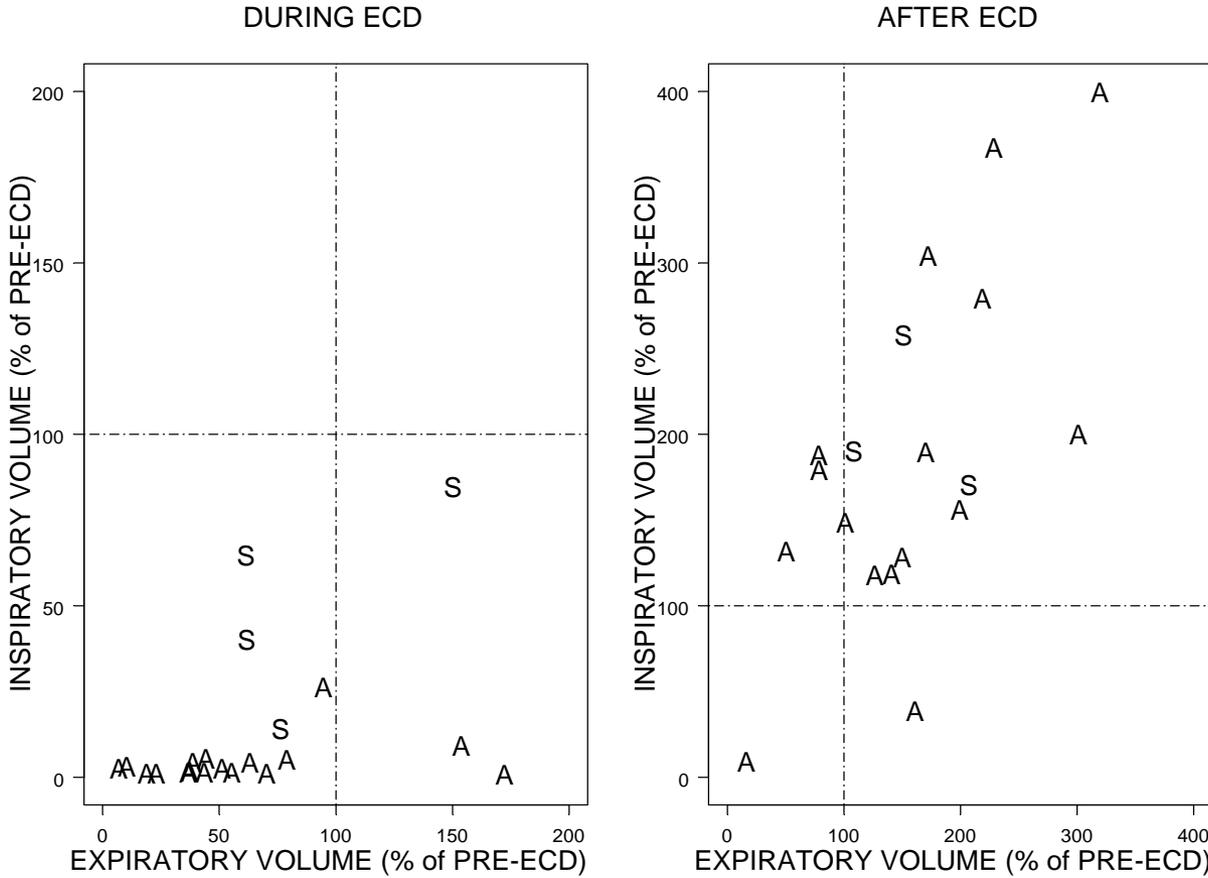


FIGURE 8: Change in Inspiratory and Expiratory Volume During ECD and After ECD



Due to the variability of the percentages the above figures are presented on different scales.