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FINAL REPORT

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**THE IMPACT OF OLEORESIN CAPSICUM SPRAY ON
RESPIRATORY FUNCTION IN HUMAN SUBJECTS IN THE
SITTING AND PRONE MAXIMAL RESTRAINT POSITIONS**

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FINAL REPORT

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I. EXECUTIVE SUMMARY

Introduction. Oleoresin Capsicum (OC) spray has gained wide acceptance in law enforcement as a swift and effective force method to subdue violent, dangerous suspects in the field. Derived from the extract of the capsicum pepper plant, OC spray causes inflammation and edema over areas of contact (primarily the face, eyes, nose and mouth), resulting in pain and discomfort such that many victims lose their capacity to resist.

With widespread use, however, OC spray has been alleged to have been associated with a number of “in-custody” deaths in the media. Because symptoms of cough, gagging and shortness of breath are common with OC exposure, concern has focused on the respiratory effects of OC spray as playing a potential role in these deaths.

Moreover, individuals subdued with OC spray in the field often require physical restraint, including the prone maximal restraint or hobble position. Some have argued OC in combination with restraint can lead to significant respiratory compromise and risk for asphyxiation and death.

While capsaicin, the active ingredient of OC, has been studied extensively in the medical literature for its ability to induce cough, there have been few studies on the physiologic effects, particularly relating to respiratory function, of OC on humans. In addition, there have been no prior studies on the effects of OC in combination with positional restraint. We sought to investigate the effects of OC on respiratory function by itself and in combination with the prone maximal restraint position.

The goal of our study was to assess the safety of a commercially available OC spray in use by law enforcement agencies nationwide. Specifically, we examined both OC spray and positional restraint in human subjects to determine if OC exposure by itself or in combination with positional restraint resulted in any significant respiratory compromise as measured by pulmonary function testing and assessment of oxygenation and ventilation.

Methodology. We conducted a randomized, cross-over, controlled trial on volunteer human subjects recruited from the local law enforcement training academy. Prior to participation, data were collected on subject weight, height, age, gender, history of lung disease, smoking, and medication use. Subjects performed 4 different experimental trials over 2 separate days in a pulmonary function testing laboratory in random order:

- a. Placebo spray exposure followed by sitting position;
- b. Placebo spray exposure followed by restraint position;
- c. OC spray exposure followed by sitting position;
- d. OC spray exposure followed by restraint position.

Prior to exposure, baseline spirometry measurements were obtained by pulmonary function testing. Measurements included forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1). These measurements assess the amount and the rate at which an individual can move air in and out of their lungs. In addition, baseline oxygen saturation (by transcutaneous oximetry), end-tidal carbon dioxide (CO₂) levels (by expired gas analysis), heart rate (by electrocardiographic monitoring), and blood pressure measurements (by automated sphygmomanometer) were obtained.

Subjects then placed their head in a 5' x 3' x 3' exposure box that allowed their face to be exposed to OC or placebo spray (see EXHIBIT A). A one-second OC or placebo spray was delivered into the box from the end opposite the subject (approximately 5 feet away). Subjects remained in the box for 5 seconds after the spray was delivered. During this time, subjects underwent impedance monitoring to assess whether inhalation of the OC or placebo spray occurred.

After this exposure period, subjects were placed in either the sitting or prone maximal restraint position (by means of handcuffs and a restraining cuff currently in use by local law enforcement agencies). Subjects remained in these positions for 10 minutes. Repeat spirometric measurements (FVC and FEV1) were performed at 1.5 and 10 minutes. Oxygen saturation, end-tidal CO₂ levels, and pulse rate were recorded at 1, 5, and 9 minutes. Blood pressures were recorded at 3, 6, and 9 minutes. An arterial blood sample was drawn at 8 minutes to assess arterial oxygen (pO₂), CO₂ (pCO₂), and acid-base status (pH levels).

After the 10 minute period, the subject had a 1 hour rest and washout period to allow for resolution of any residual effects from either exposure or position. After this rest period, the subject performed a second experimental trial. The subject only performed 2 trials on each experimental day. Though the sequence of trials was randomized, no subject was randomized to receive 2 exposures to OC in a single day (see EXHIBIT B for study trial protocol).

Raw spirometric data were converted to percent of predicted values (% predicted FVC and % predicted FEV1) to normalize for body size and ethnicity. Analysis of variance (ANOVA) for repeated measures was performed on all data with exposure (OC

or placebo) and position (sitting and restraint) to determine if any significant differences existed between the 4 experimental trials. A probability value of less than 0.05 (after Bonferroni correction for independent variables) was considered statistically significant. Clinical significance was determined by any evidence of abnormal pulmonary function (spirometric measurements below 80% of predicted), hypoxemia (oxygen saturation < 95% or pO₂ < 85 mmHg), or hypercapnia (end-tidal CO₂ or pCO₂ > 45 mmHg). Data analysis was performed by means of a computerized statistical software package (Stata 6.0 for Windows, Stata Corporation®).

Results. A total of 34 out of 37 subjects (24 men and 10 women) completed the study, performing 136 trials (4 trials for each subject). Of the 3 subjects who did not complete the study, 2 were excluded due to acute injuries prior to the study that prevented study participation (one subject with an acute rib fracture, and another with an acute forearm fracture). One subject was excluded after he suffered a fainting episode during blood draw phlebotomy. This subject recovered uneventfully. The incident occurred during the subject's first trial, in which he was randomized to the sitting position after placebo exposure. He was never exposed to OC or restrained at any time.

Of the 136 completed trials, 8 were excluded from analysis because the subject did not adequately inhale (as measured by impedance monitoring) during OC exposure. As a result, 128 separate study trials were analyzed for purposes of this study.

In the sitting position, there was no difference or abnormalities in pulmonary function as measured by FVC and FEV₁ at baseline, 1.5 or 10 minutes after exposure between the placebo and OC groups (mean % predicted FVC: 102.8 vs. 103.1% at baseline, 102.0 vs. 102.4% at 1.5 minutes, and 101.8 vs. 102.3% at 10 minutes; mean %

predicted FEV1: 100.1 vs. 100.3% at baseline, 98.9 vs. 98.9% at 1.5 minutes, and 99.2 vs. 99.0% at 10 minutes, respectively) (see EXHIBIT C and D).

There was also no difference in oxygenation or evidence of hypoxemia between the placebo and OC groups in the sitting position (mean oxygen saturation: 99.2 vs. 99.2% at baseline, 99.0 vs. 99.1% at 1 minute, 98.6 vs. 99.0% at 5 minutes, and 99.5 vs. 98.0% at 9 minutes; mean arterial pO₂: 96.8 vs. 99.4 mmHg at 8 minutes, respectively). OC exposure led to slightly lower CO₂ levels when compared with placebo, but no evidence of hypercapnia in either group (mean end-tidal CO₂ levels: 38.0 vs. 38.2 mmHg at baseline, 36.8 vs. 32.4 mmHg at 1 minute, 36.5 vs. 32.9 mmHg at 5 minutes, and 37.0 vs. 35.2 mmHg at 9 minutes; mean arterial pCO₂: 39.4 vs. 36.4 mmHg for placebo and OC groups, respectively) (see EXHIBIT E and F).

In the restraint position, FVC and FEV1 significantly decreased with restraint position, but remained within clinical normal limits. Despite this decline there was no difference found between placebo and OC groups (mean % predicted FVC: 101.9 vs. 103.4% at baseline, 87.5 vs. 87.5% at 1.5 minutes, and 87.9 vs. 87.2% at 10 minutes; mean % predicted FEV1: 99.7 vs. 101.1% at baseline, 83.2 vs. 82.5% at 1.5 minutes, and 83.7 vs. 82.0% at 10 minutes, respectively) (see EXHIBIT C and D).

There was again no difference in oxygenation or evidence of hypoxemia between the placebo and OC groups in the restraint position (mean oxygen saturation: 99.4 vs. 99.3% at baseline, 97.9 vs. 98.1% at 1 minute, 98.0 vs. 98.8% at 5 minutes, and 97.4 vs. 98.4% at 9 minutes; mean arterial pO₂: 90.2 vs. 90.0 mmHg at 8 minutes, respectively). OC exposure led to slightly lower CO₂ levels when compared with placebo, and no evidence of hypercapnia in either group (mean end-tidal CO₂ levels: 37.7 vs. 36.3 mmHg

at baseline, 38.8 vs. 36.7 mmHg at 1 minute, 39.1 vs. 36.6 mmHg at 5 minutes, and 39.5 vs. 37.7 mmHg at 9 minutes; mean arterial pCO₂: 40.9 vs. 39.1 mmHg for placebo and OC groups, respectively) (see EXHIBIT E and F).

In both the sitting and restraint groups, OC exposure led to small, but statistically significant increases in blood pressure as determined by mean arterial pressure (MAP equal to one-third of systolic pressure + two-thirds of diastolic pressure). This difference persisted up to 9 minutes after exposure (for sitting position: mean MAP 101.0 vs. 103.4 mmHg at baseline, 103.8 vs. 116.8 mmHg at 3 minutes, 100.7 vs. 112.1 at 6 minutes, and 98.1 vs. 107.8 mmHg at 9 minutes; for restraint position: 102.2 vs. 102.7 mmHg at baseline, 102.0 vs. 110.7 at 3 minutes, 99.1 vs. 108.2 mmHg at 6 minutes, and 100.1 vs. 110.0 mmHg at 9 minutes for the placebo and OC groups, respectively) (see EXHIBIT H).

Discussion. In this study, we found no evidence that OC spray inhalation and exposure in human subjects resulted in any respiratory compromise, as measured by pulmonary function testing, oxygen and CO₂ levels, in the sitting position. Moreover, there was no evidence of hypoxemia or hypercapnia as a result of OC exposure. In fact, OC appeared to slightly increase ventilation as evidenced by lower CO₂ levels for this group.

In the restraint position, we found declines in pulmonary function typical for prone maximal restraint body position, but no evidence of clinically significant spirometric abnormalities or differences between the OC and placebo groups. Furthermore, there was no evidence of hypoxemia or hypercapnia as a result of OC

exposure and, similar to the results in the sitting position, OC seemed to increase ventilation as evidenced by small decreases in CO₂ levels for this group.

Based on these results, we conclude that OC exposure and inhalation do not result in a significant risk for respiratory compromise or asphyxiation. Moreover, even when combined with positional restraint, OC inhalation does not result in an increased risk of ventilatory failure or asphyxiation.

OC exposure did, however, result in an increase in blood pressure. The etiology of this increase remains unclear, but may be in part due to the discomfort and pain associated with OC. The clinical ramifications of this elevation in blood pressure remain unknown.

This study has a number of limitations. First, as this study was conducted in a pulmonary function laboratory, not all conditions that occur in the field setting where OC and restraint are employed, could be reproduced. Factors that commonly occur in the field setting, such as physical struggle, physiologic and psychological stress, agitation, drug intoxication, trauma and exertion, were not studied in our investigation. Second, though we believe our protocol adequately replicated a single OC exposure in the field, we did not study prolonged sprays or repeated exposures. Third, all subjects were recruited from cadets at the police academy and were generally healthy young subjects. Fourth, this study did not investigate long-term effects due to OC, nor did we investigate the potential for complications from chronic, occupational exposure to OC.

In conclusion, in our study on human subjects, OC exposure and inhalation did not result in abnormal ventilatory or respiratory function in either the sitting or prone maximal restraint positions. We found no evidence to support the contention that acute

OC exposure places subjects at significant risk for respiratory compromise or asphyxiation.

II. PROJECT DESCRIPTION

A. INTRODUCTION

Law enforcement personnel have a variety of force options when dealing with dangerous, combative, and violent subjects in the field. On a continuum of increasing levels of force, these options include officer presence, verbal dialogue, physical control and restraint, chemical agents, impact weapons, and lethal force or weapons. While higher levels of force are likely to subdue and control subjects more swiftly, they increasingly place both subject and officer at greater risk for injury.

In this regard, the success of Oleoresin Capsicum (OC) or “pepper” spray, and its ability to temporarily incapacitate subjects, has led to its adoption as a force option by law enforcement agencies nationwide. OC spray has been credited with decreasing injuries among officers and subjects by reducing the need for more dangerous force options.¹

Despite its success, there is growing concern regarding the safety of OC spray, particularly when combined with positional restraint. There have been a number of in-custody deaths in subjects exposed to OC.^{2,3} As the spray induces cough, gagging and shortness of breath, some have alleged that OC inhalation places individuals at risk for potentially fatal respiratory compromise.^{4,5}

Custody deaths, however, are not new and occurred prior to the widespread use of the spray. Cases in which subjects die after violent confrontation with law enforcement often share similar patterns. Determining a specific cause of death is difficult as there is little pathologic evidence at autopsy.^{1,6,7,8,9} Commonly, subjects are in a state of altered

sensorium or “excited delirium” induced by drug abuse. Subjects may have underlying cardiopulmonary disease or an obese body habitus. Violent physical struggle is often followed by physical restraint.¹

Attention has focused on these factors, and in particular the use of physical restraint, to explain these deaths. In fact, many of these deaths have been attributed to positional asphyxia, the theory that subjects placed in a prone maximal restraint position are at risk for fatal respiratory compromise from restricted chest and abdominal movement.

As opposed to other factors, OC spray use and positional restraint are directly related to policing action, practices and policies. As a result, these force options have received tremendous focus, attention and scrutiny from the public, media and law enforcement community. Moreover, it is just these types of controversial custody deaths that become contentious public issues and strain relations between law enforcement agencies and their communities.¹⁰

Some have gone so far as to label both OC spray and positional restraint as forms of police brutality and excessive force. Litigation has arisen, impacting the financial well-being and morale of law enforcement agencies and their personnel.⁵ Furthermore, these deaths not only impact the subject and their family, but can have significant negative effects on the lives and families of the officers involved.

Yet, scientific data are limited on the effects of OC spray in humans and the data are insufficient to determine if the spray represents any substantial hazard to humans.^{3,5,11} Prior to this study, the effects of OC spray in combination with positional restraint in

human subjects had never been studied, and were largely unknown, particularly with respect to pulmonary and respiratory function.

B. STUDY GOALS, OBJECTIVES AND SIGNIFICANCE

The goal of this study was to assess the safety of OC spray by itself and in combination with physical restraint. Specifically, we examined both OC spray and positional restraint in human subjects to determine if OC spray exposure by itself or in combination with positional restraint resulted in any significant compromise in respiratory or pulmonary function.

The specific main objectives of this study were:

1. To determine if OC spray inhalation in the sitting position results in clinically significant respiratory compromise as measured by pulmonary function testing and assessment of oxygenation and ventilation;
2. To determine if OC spray followed by the prone maximal restraint results in clinically significant respiratory compromise as measured by pulmonary function testing and assessment of oxygenation and ventilation.

In addition, this study examined other issues related to the use of OC spray and positional restraint as follows:

3. Whether OC spray by itself or in combination with positional restraint results in any cardiovascular compromise as measured by pulse rate and blood pressure in human subjects.

4. Whether body size and weight influence the effects of OC spray in regards to respiratory and pulmonary function as measured by pulmonary function testing and assessment of oxygenation and ventilation.
5. Whether pulmonary disease (such as asthma), the use of respiratory inhaler medications, or history of smoking tobacco influence the effects of OC spray in regards to respiratory and pulmonary function as measured by pulmonary function testing and assessment of oxygenation and ventilation.

This study represents a unique collaboration between regional law enforcement agencies and the local academic medical institution to assess and improve the safety of policing practices in our community. Specifically, this study was a joint effort of the San Diego Regional Public Safety Training Institute, as part of the San Diego City Police and San Diego County Sheriff's Departments, and the Department of Emergency Medicine (and its Division of Medical Toxicology) and Department of Internal Medicine (and its Division of Pulmonary and Critical Care Medicine), at the University of California San Diego Medical Center.

C. BACKGROUND

Oleoresin Capsicum Spray

Oleoresin capsicum, the active component of OC spray, is the oily extract of the pepper plant of genus capsicum, consisting of a complex mixture of capsinoids, including capsaicin and a variety of its closely related analogues.¹² These agents act as irritants to the skin, ocular and mucous membranes of the upper aerodigestive tract.

History. Japanese samurai warriors used pepper extracts placed in rice paper bags to throw at the eyes of enemies and cause temporary blindness. Chinese fighters would heat red peppers in hot oil to form an irritant smoke while in battle. In 1973, oleoresin capsicum became available in aerosol spray formulations for use to incapacitate animals and humans on a temporary basis, and was initially used by FBI personnel and US mail carriers. During the late 1980s, OC spray was widely adopted by law enforcement agencies nationwide and also became available for general public use as personnel deterrent devices.^{5,13,14}

Currently, hand-held canister spray models are produced and used in this country. These models produce an aerosol, liquid stream or foam spray, with OC concentrations varying from 1 to 10%, mixed in a carrier solvent such as isopropyl alcohol, denatured ethanol, or propylene glycol. Actual amounts of capsaicinoids are variable.¹⁵ Unlike other classic forms of tear gas and mace such as CN (2-chloro-acetophenone) and CS (o-chlorobenzyl-idenemalononitrile), pepper sprays are felt to be more immediately effective, safer and less toxic.^{3,15}

OC Spray Effects. Biochemically, capsaicinoids stimulate chemoreceptors in primary afferent nerve endings, resulting in immediate pain and burning sensation over exposed areas of the skin, ocular, nasal and oropharyngeal tissues. In addition, they stimulate reflex movements and cause the release of peripheral neuropeptides, including substance P, which can lead to neurogenic inflammation, vasodilation and edema.^{3,16}

When directed at the face, effects are most prominent on the eyes, skin, nose and mouth. OC causes stinging in the eyes, conjunctival injection from vasodilation,

lacrimation, involuntary blepharospasm, and rarely corneal abrasions. Because of these immediate, severe ocular symptoms, many subjects lose their capacity to resist.

On contact with skin, OC spray causes severe burning sensation with local inflammation and erythema. OC spray also causes a local inflammatory reaction in the nose and oral mucosa, resulting in rhinorrhea, swelling, and pain. With acute exposures, these effects are transient and reportedly last anywhere from 30 to 60 minutes.¹⁴

Respiratory Effects. With inhalation or exposure to the oropharynx, OC spray causes a variety of immediate respiratory symptoms, including cough, gagging, inability to vocalize and subjective shortness of breath. In acute exposures, these symptoms are usually limited, lasting 15 to 30 minutes.¹⁴

Reports of Fatalities. The number of custody deaths following OC spray have paralleled the rise of its use nationwide and raised concern regarding its safety. In 1994, Granfield reported on 30 custody deaths associated with exposure to OC spray from 1990 through 1993.¹ An additional 60 or more deaths have been reported since that time.⁵

Concern has focused on the respiratory effects of OC spray as playing a potential causal role in these deaths. There have been reports of children who suffered significant respiratory compromise following accidental exposure to OC spray.^{13,17} Some suggest that when inhaled, the spray causes laryngospasm, airway edema, bronchoconstriction and pulmonary inflammation and edema, placing subjects at risk for respiratory compromise and arrest. Moreover, subjects with a history of asthma or other underlying pulmonary disease are opined to be at greater risk. Steffee et al reported on the custody death of a 24 year-old man with a history of asthma in which death was attributed to asphyxiation from bronchospasm precipitated by OC.³

Studies on Capsaicin. Evidence that OC may have significant respiratory effects is based on research on inhaled nebulized capsaicin. Because of its ability to induce cough, capsaicin has been studied extensively as a model for understanding the cough reflex. There has also been interest in capsaicin because of its ability to block pain sensation and pruritis, presumably by depletion of substance P and other neurotransmitters.

While animal and in-vitro human tissue studies suggest capsaicin induces significant increases in airway resistance and bronchoconstriction,^{18,19} clinical studies with nebulized capsaicin are less clear. In 1985, Fuller reported that inhaled nebulized capsaicin resulted in a transient dose-dependent increase in airway resistance, maximal at 20 seconds and lasting less than 60 seconds. There was no difference between normal subjects and those with a history of asthma or smoking.²⁰ The lack of spirometric evidence for sustained bronchoconstriction was further substantiated by Collier and Blanc, both of whom found no significant decrease in forced expiratory volume in 1 second (FEV1) in human subjects who inhaled nebulized capsaicin at concentrations sufficient to induce cough.^{21,22}

Both cough and deep inhalation however, have bronchodilatory effects, which may mask direct bronchoconstriction caused by capsaicin.²³ There is evidence that sub-tussive doses of inhaled capsaicin leads to marked changes in airway resistance.²⁴ Maxwell found transient increases in minute ventilation and respiratory rates in subjects exposed to sub-tussive doses of capsaicin.²⁵ Hathaway reported that asthmatic patients were more likely to decrease their FEV1 following inhaled capsaicin.²⁶

Capsaicin has been found to stimulate both C and A delta sensory afferent fibers in the airway and pulmonary parenchyma in animal studies, and has been implicated in precipitating a transient bronchoconstriction and cough reflex. In addition, it has been suggested that stimulation of these nerve endings could precipitate a pulmonary chemoreflex, resulting in bradycardia, hypotension, and apnea.¹⁶

Studies on OC Spray. Unlike capsaicin, research on the human effects of OC spray are limited.⁵ A two-year joint study by the FBI and US Army determined that no long-term health risks were associated with OC spray.²⁷ Other studies have been limited to retrospective reviews of law enforcement experience in the field. Granfield et al reviewed 30 cases of custody deaths that occurred after OC spray exposure. Twenty-two of these cases had enough information that cause of death could be determined. In no case was death attributed to OC exposure.¹ Since that time, another 60 deaths following OC spray exposure have been reported, of which one was attributed to asphyxiation from bronchospasm precipitated by OC spray.^{2,3,5,28}

In reviewing OC spray use in 1996, the California State Attorney General reported that no fatal consequences occurred in over 23,000 exposures.²⁹ Watson et al reviewed 908 OC spray exposures in their local jurisdiction and found less than 10% of subjects exposed required any medical attention. Moreover, less than 1% of these subjects complained of respiratory symptoms requiring medical attention, and none were determined to have any significant respiratory injury on evaluation.³⁰

Because of the lack of studies on the overall and respiratory effects of OC spray, it remains unclear whether the spray represents a significant health hazard to those who use and are exposed to the agent.^{3,11}

Positional Asphyxia

In their review, Granfield et al reported that all victims were placed in physical restraints during or following OC spray exposure. Granfield suggests the majority of deaths can be attributed to positional asphyxia rather than OC spray exposure.¹ In fact, law enforcement agencies have implemented policies limiting certain physical restraints and body positions after OC spray exposure.^{31,32,33}

Case Reports. The term positional or mechanical asphyxia has been used to explain the deaths of individuals who were found in body positions that caused upper airway obstruction or interfered with ventilatory function.³⁴ The term has also been applied to asphyxiation deaths where chest restraints wrapped around the necks of individuals resulting in strangulation.^{35,36} There have been reports of deaths caused by restraints that allegedly compressed the chest and abdomen to the point that mechanical ventilation was impaired.^{37,38}

More recently, the theory of positional asphyxia has been used to explain the sudden custody deaths of individuals who were placed in the maximal prone or hogtie (also known as hobble) restraint positions. Some have argued that this position, in which a subject lays prone with wrists and ankles bound together behind the back, prevents adequate chest and abdominal movement for ventilation, potentially placing subjects at risk for hypoventilatory respiratory compromise and asphyxiation.^{6,37,39,40}

Physiologic Studies. The role of positional asphyxia in the restraint position is based almost entirely on the work of Reay et al who found measurable physiologic effects in 10 healthy subjects placed in the prone restraint position. In 1988, Reay

reported prolonged recovery times for transcutaneous oxygen saturation and heart rate in subjects placed in the restraint position as opposed to sitting after mild exercise.⁴¹

However, Reay made no assessment of actual ventilatory function and respiratory mechanics in subjects placed in the restraint position. Transcutaneous oximetry is an indirect measure of blood oxygenation and frequently inaccurate under conditions of exercise.^{42,43} Also, Reay reported oxygen desaturations down to 85% with exercise, substantially lower than what would be expected at such relatively mild levels of exertion in healthy subjects.^{44,45}

Previously, we conducted a more comprehensive study examining the physiologic effects of this position.⁴⁶ Fifteen healthy volunteers underwent a two-phase randomized, cross-over controlled trial that evaluated static and dynamic respiratory function. First, subjects underwent pulmonary function testing (PFT) in four static positions: sitting, supine, prone, and restraint. We found a progressive restrictive pulmonary function pattern as subjects went from sitting to supine and prone to restraint positions. Mean forced vital capacity (FVC) fell progressively (declines of 7%, 7% and 13% of predicted for supine, prone and restraint positions respectively), as did mean FEV1 (declines of 8%, 9% and 14% respectively), and mean maximal voluntary ventilation (MVV, declines of 10%, 15% and 23% respectively).⁴⁶

Subjects were then placed in either the sitting or restraint position after a period of exercise. We found that exercise improved FVC and FEV1 in both the sitting and restraint positions. Moreover, we found no evidence of hypoxia by transcutaneous monitoring or direct arterial blood sampling. We also found no evidence of CO₂ retention suggestive of any significant hypoventilation. Finally, despite a more vigorous

exercise regimen, we found no evidence of a delay in heart rate recovery in subjects placed in the restraint position after exercise.⁴⁶

Thus, while our initial findings with static positions suggested the possibility of hypoventilation, our results in the dynamic phase found no evidence to support the theory of positional asphyxia. Moreover, as PFT measurements as low as 80% of predicted values are still normal, these static positional changes are of little clinical relevance.⁴⁷ The fact that these changes were clinically insignificant is further emphasized by the fact that small changes in pulmonary function were found simply in the supine and prone positions as well.

Schmidt et al also found no significant physiologic differences in their study of 18 subjects placed in the sitting and restraint positions after exercise. In addition, they studied subjects after a simulated pursuit and physical struggle regimen and found no differences in oxygen saturation.⁴⁸ Rogers et al studied 10 healthy volunteers in both the prone and lateral restraint positions and found no differences in peak expiratory flow rates or oxygen saturation after exercise.⁴⁹

Limitations. However, these studies examined positional restraint in healthy subjects. Our study was conducted with volunteers who were not severely overweight (body mass index or BMI less than 30 kg/m²) with no history of cardiopulmonary disease. Individuals with lung disease, and in particular asthma, may be more susceptible to small pulmonary function changes. Extreme obesity itself may result in abnormal pulmonary function as a result of changes in the chest wall and abdomen.^{50,51}

Furthermore, law enforcement personnel often place individuals in the restraint position for control and transport after initially subduing the combative subject with OC

spray. OC spray inhalation in combination with positional restraint has not been previously studied to determine if the combination of these force methods results in any detriment in pulmonary function that places individuals at risk for respiratory compromise and arrest.

III. SCOPE AND METHODOLOGY

A. Study Design and Location

We conducted a randomized, cross-over, controlled trial to assess the effect of OC spray exposure on pulmonary and respiratory function in the sitting and prone maximal restraint positions in volunteer human subjects.

Specifically, volunteers performed four (4) different experimental trials over two experimental days in random order:

- a. Placebo (no OC) spray exposure followed by sitting position;
- b. Placebo spray exposure followed by restraint position;
- c. OC spray exposure followed by sitting position;
- d. OC spray exposure followed by restraint position.

Subjects performed two of the trials during each experimental day. The order of the trials was randomized. However, to prevent two exposures to OC spray in a single experimental day, no subject was randomized to perform both trials c and d on the same day. Thus, on a given experimental day, subjects performed two of the trials (a and c, a and d, b and c, or b and d), and subsequently performed the remaining two trials on the next experimental day.

This study was conducted at the Pulmonary Function Laboratory at the University of California, San Diego Medical Center. The medical center is the only designated level-one trauma center and medical facility located in San Diego County.

B. Human Subjects

Volunteers were recruited among training cadets from the San Diego Regional Public Safety Training Institute, which trains law enforcement personnel for the San Diego City Police and San Diego County Sheriff's Departments. Recruitment was conducted in conjunction with personnel staff at the Training Center (Mr. T. Snowden and Mr. P. Schmidt). As a routine part of their training, cadets are exposed to OC spray on a voluntary basis in order to further their understanding of the effects of the spray when used in the field.

Informed written consent was obtained from all subjects prior to participating in the study. Potential subjects were told that participation in the study was completely voluntary and that participation would in no way affect their training or advancement.

No exclusion was made on the basis of race, ethnicity, age, obesity or history of pulmonary disease such as asthma. Subjects completed a short questionnaire regarding their health status, history of lung disease and asthma, smoking history, medication use, and respiratory inhaler medication use. Prior to experimentation, subjects underwent brief screening spirometry in the sitting position by means of a portable spirometry device to determine baseline pulmonary function. No exclusion was made on the basis of these results.

The research design and methods of this study were approved by the Human Subjects Committee and Institutional Review Board (IRB) of the University of California, San Diego (UCSD).

C. Experimental Procedures

Sitting and Restraint Body Positions. In the sitting position, the subject sat in a chair with feet flat on the floor and back upright against the back of the chair. In the restraint position, the subject lay prone on their stomach on a medical examination table with head turned to the side. The subject's wrists were bound together behind the back by means of police handcuffs. The subject's ankles were bound together and secured near the wrists by means of the maximal restraint cuff currently used by law enforcement agencies in San Diego County.

Placebo and OC Spray Exposure. OC and placebo spray exposure was facilitated by use of a 5 foot by 3 foot by 3 foot plastic exposure box (see EXHIBIT A). On one end of the exposure box, a large ventilation hood was attached. On the opposite end, a small opening was created from which OC or placebo spray was delivered.

The hood/exposure box was adapted for this study for a number of reasons. First, this method allowed for a more uniform and reproducible spray exposure and concentration within the box. Second, this method allowed the spray to be delivered from a standard distance of 5 feet (the length of the box) and targeted at the subject's face, focusing exposure on inhalation. Third, the exposure box prevented contamination of the laboratory. As we were most interested in OC inhalation, subjects were allowed to wear safety goggles to prevent ocular exposure.

OC was delivered via a standard duty aerosol spray canister commercially available in the United States. This aerosol contains 5.5% OC (0.92% capsaicinoids), 64% isopropyl alcohol as the carrier agent, and 30.5% isobutane/propane as the propellant. This particular delivery system is currently used by San Diego law

enforcement agencies. Placebo spray was delivered by a similar canister containing only the carrier and propellant agents (68%% isopropyl alcohol and 31.5% isobutane/propane.

The canister was used to deliver a 1 second OC or placebo spray into one end of the exposure box, approximately 5 feet from the subjects face. The subject's head remained in the hood of the exposure box for 5 seconds after the spray was released.

Pulmonary function testing. Pulmonary function testing was performed in the standard manner of the UCSD Pulmonary Function Laboratory. PFT measurements were obtained in accordance with the American Thoracic Society's criteria, including reproducibility within 5% on three repeat measurements.⁵² Spirometric measurements collected included forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1) obtained using a Medgraphics Cardiopulmonary Diagnostic System® machine.

Cardiopulmonary Monitoring. Subjects' cardiopulmonary status was monitored continuously throughout the experimental trials in the following manner.

1. Electrocardiographic (ECG) monitoring by 3-lead continuous monitor using a Quinton Instrument 4000®;
2. Serial blood pressure monitoring by automated sphygmomanometer located on the upper arm using an MDE Escort 100 Series® and Tango® blood pressure monitoring machine;
3. Pulse oximetry oxygen saturation monitoring by transcutaneous device placed on the index finger using an Ohmeda Biox 3740 Pulse Oximeter® machine;

4. Expired gases and end-tidal CO₂ monitoring by means of a quantitative CO₂ detector using a Medgraphics Cardiopulmonary Exercise System CPX/D® machine.

Arterial Blood Gas. Arterial blood gas (ABG) collection and analysis occurred in the following manner. UCSD PFT laboratory technicians collected 1-2 cc of arterial blood from either radial artery at the subject's wrist under sterile conditions in the standard technique with an ABG arterial puncture kit. ABG analysis was performed in the ABG laboratory of UCSD Medical Center in the standard fashion to determine pH, pO₂, pCO₂, and oxygen saturation by co-oximetry. All measurements were made in duplicate on two different ABG analyses.

Impedance Monitoring. Transthoracic impedance monitoring using an Edentec Sleep Recorder System® was performed on subjects during the 5 second spray exposure period to assess whether inhalation occurred during the exposure period.

D. EXPERIMENTAL PROTOCOL

At the start of each experimental day, the randomized order of trials was determined for each subject. On the first experimental day, the subject's weight, height, age and ethnicity were recorded. Continuous transcutaneous ECG, pulse oximetry and impedance monitors were placed on the subject for monitoring as described above.

At the start of each trial, baseline spirometry, including measurement of FVC and FEV₁, were performed in the sitting position. In addition, baseline measurements of oxygen saturation, end-tidal CO₂, heart rate and blood pressure were performed. The

subject then placed his or her head in the hood portion of the exposure box. The subject was allowed to wear safety eye goggles at his or her discretion.

OC or placebo spray was delivered into the box from a distance of 5 feet from the subject's face as described above. The subject remained in the hood of the exposure box for 5 seconds after the spray, during which time, impedance monitoring was used to assess that the subject inhaled.

After the 5 second exposure period, the subject was removed from the exposure box and immediately placed into the sitting or restraint position (as described above) depending on the particular experimental trial. The subject remained in this position for 10 minutes.

During this time, continuous cardiopulmonary monitoring was performed as outlined above. Data were collected and recorded in the following manner: transcutaneous oxygen saturation recorded at 1, 5, and 9 minutes; heart rate recorded at 1, 5, and 9 minutes; expired gas end-tidal CO₂ levels recorded at 1, 5, and 9 minutes; and arterial blood pressure measured at 3, 6, and 9 minutes.

At 1.5 minutes into the period, spirometric pulmonary function testing was performed as outlined above, and measurements of FVC and FEV₁ obtained. At 10 minutes into this period, repeat spirometry was performed with similar data collection. At 8 minutes into the period, arterial puncture and blood sampling were performed and sent to the ABG laboratory for analysis of blood pH, pO₂, and pCO₂.

After the 10 minute period, the subject was released from the designated body position (sitting or restraint). If the subject was exposed to OC spray, any residual OC on

the subject was removed by means of washing with soap and water as recommended by the manufacturer.

Once the trial was completed, all monitoring devices were removed and the subject rested for 1 hour to allow “washout” of any residual effects from exposure, body position or testing.

After the 1 hour rest period, the second trial as designated by randomization was performed in the manner of protocol outlined above. After the subject’s second trial and washout period, the experimental day was completed. As noted above, all 4 experimental trials for each subject were completed over 2 different experimental days in order to avoid OC exposure twice in a single day (See EXHIBIT B for the experimental trial protocol).

E. DATA COLLECTION AND ANALYSIS

Demographic data were collected on the subject’s age, weight, height and race. The subject’s weight and height were used to calculate body mass index (BMI). These data were used to stratify subjects as overweight (BMI>28 kg/m²). Additional data were collected on medical history, presence of lung disease (including asthma), smoking history, medication use, and respiratory inhaler medication use. These data were used to stratify subjects into those with potential respiratory abnormalities (asthma or lung disease history, tobacco history, inhaler medication use).

Experimental data were collected as delineated above on the subject’s heart rate, blood pressure, oxygen saturation, and expired gas end-tidal CO₂ levels. Data were also collected on arterial blood oxygenation (pO₂) and CO₂ levels (pCO₂). Pulmonary

function testing data were collected (FVC and FEV1) and converted to a percentage of predicted (% predicted, denoted % predicted FVC and % predicted FEV1) for each subject to allow for normalization for age, height and race as per standard practice.^{52,53}

Statistical Analysis. Analysis of Variance (ANOVA) for repeated measures with position (sitting or restraint) and exposure (OC or placebo) as factors was performed on all data to determine if any statistically significant differences existed between the four experimental trials. A probability value of less than 0.05 (after Bonferroni correction for independent variables) was considered statistically significant. Data analysis was performed by means of a computerized statistical software package (Stata 6.0 for Windows, Stata Corporation).

An *a priori* power analysis determined that 32 subjects would be needed to detect a 10% difference in pulmonary function testing parameters if such a difference existed between the four trial groups.

Clinically, the data were analyzed on multiple levels. First, data were analyzed as absolute values in comparison with known normal values. Hypoxemia, or abnormal oxygenation was defined as a pO₂ less than 85 mmHg or oxygen saturation less than 95%. Hypercapnia as a result of alveolar hypoventilation was defined as pCO₂ and end-tidal CO₂ levels greater than 45 mmHg. PFT measurements were considered abnormal if they fell below 80% of established predicted values (or below the fifth percentile of normal for a given age, body size and ethnicity⁵²).

Second, data were compared between the experimental trial groups. Cardiorespiratory parameters (heart rate, blood pressure, oximetry, pO₂, end-tidal CO₂

and pCO₂ levels) and PFT measurements (% predicted FVC and % predicted FEV₁), were compared between the different exposure and position groups.

In addition, the data were analyzed regarding questions related to the issues of OC spray and positional restraint. Data were stratified by BMI and potential for pulmonary abnormalities as delineated above. An additional analysis was conducted on these groups to determine if any clinically significant differences occurred in these subjects.

IV. DETAILED FINDINGS

A. Study Subjects

Thirty-seven (37) subjects from the training staff and cadets of the San Diego Regional Public Safety Training Institute were recruited and enrolled as subjects for this study. Two subjects were excluded prior to starting the study. One subject had fractured his ribs and was unable to adequately perform spirometric pulmonary function testing. The other subject had a fractured arm immobilized in a cast making it impossible to place the subject in the restraint position.

The third subject was excluded after he experienced an adverse reaction during his first trial. The subject suffered a syncopal event (fainted) during arterial puncture and blood drawing (phlebotomy) which was most likely due to vagal hyperstimulation. During this trial, the subject had been exposed to placebo spray and was in the sitting position when he fainted. This was his first trial and he was never exposed to OC spray nor placed in the restraint position at any time during the study. His recovery was uneventful and he suffered no lasting sequelae or injury.

Overall, 34 subjects completed the study. Of these, 24 were men and 10 were women. The mean age was 31.7 years with a range of 22 to 46 years of age. The mean weight was 79.1 kg with a range of 52 to 107 kg. Mean BMI was 25.9 kg/m² with a range of 19.2 to 31.6 kg/m². Seven (7) subjects were stratified as overweight with a BMI > 28 kg/m². Eight (8) subjects had a history of smoking, lung disease or asthma, or respiratory inhaler medication use.

The 34 subjects completed a total of 136 separate trials (4 trials each). Of these, 8 trials were excluded from analysis because the subject did not adequately inhale (as measured by impedance monitoring) when exposed to OC spray. As a result, 128 separate study trials were analyzed for purposes of this study.

For the spirometric and pulmonary function testing data, an additional 4 trials were excluded as testing did not meet American Thoracic Society criteria for reproducibility and variability.⁵² For the arterial blood gas data, 2 trials were excluded because venous rather than arterial blood was sampled. For the blood pressure data, 1 trial was excluded due to mechanical instrument error.

B. Effect of OC Exposure in the Sitting Position

Spirometric Findings. In the sitting position, OC exposure did not result in a statistically significant change in pulmonary function as measured by FVC and FEV1. OC exposure also did not result in any clinically significant abnormalities in pulmonary function (as previously defined as below 80% of predicted values).

There was no difference in baseline % predicted FVC prior to exposure in the sitting position. For the placebo group, mean baseline % predicted FVC was 102.8% of predicted (standard deviation [SD] of 9.2%; 95% confidence interval [CI] of 99.5%-106.1%); and for the OC group, 103.1% (SD 8.7%; CI 99.9-106.3%).

There were no significant changes in % predicted FVC at 1.5 or 10 minutes after exposure as well. For the placebo group, mean % predicted FVC was 102.0% (SD 9.0%; CI 98.8-105.1%) at 1.5 minutes, and 101.8% (SD 9.1%; CI 98.6-105.1%) at 10 minutes. For the OC group, mean % predicted FVC was 102.4% (SD 7.9%; CI 99.5-105.3%) at

1.5 minutes, and 102.3% (SD 8.6%; CI 99.2-105.5%) at 10 minutes after exposure (see EXHIBIT C).

Similarly, there were no differences in % predicted FEV1 between the OC and placebo exposure groups in the sitting position. For the placebo group, mean baseline % predicted FEV1 was 100.1% (SD 9.3%; CI 96.7-103.4%); and for the OC group, 100.3% (SD 9.1%; CI 97.0-103.6%). At 1.5 minutes after exposure, mean % predicted FEV1 was 98.9% (SD 9.6%; CI 95.6-102.3%) for the placebo group, and 98.9% (SD 9.4%; CI 95.5-102.4%) for the OC group. At 10 minutes, mean % predicted FEV1 was 99.2% (SD 10.1%; CI 95.6-102.8%) for the placebo group, and 99.0% (SD 9.5%; CI 95.6-102.5%) in the OC group (See EXHIBIT D).

Oxygenation. OC spray exposure did not result in any statistically significant differences in oxygenation (as measured by transcutaneous oxygen saturation and arterial pO2 levels) when compared to placebo in the sitting position. Moreover, there was no evidence of clinical hypoxemia (lower than normal amount of oxygen in the blood as previously defined as O2 saturation less than 95% or pO2 less than 85 mmHg) while in the sitting position after OC or placebo exposure.

For the placebo group, mean oxygen saturation level was 99.2% (SD 0.9%; CI 98.9-99.5%) at baseline, 99.0% (SD 1.1%; CI 98.6-99.4%) at 1 minute, 98.6% (SD 1.4%; CI 98.1-99.0%) at 5 minutes, and 99.5% (SD 0.6%; CI 98.3-100%) at 9 minutes after exposure. For the OC group, mean oxygen saturation level was 99.2% (SD 0.9%; CI 98.9-99.6%) at baseline, 99.1% (SD 1.1%; CI 98.7-99.5%) at 1 minute, 99.0% (SD 1.2%; CI 98.6-99.5%) at 5 minutes, and 98.0% (SD 3.6%; CI 96.7-99.4%) at 9 minutes after OC exposure. Similarly, the arterial pO2 at 8 minutes was 96.8 mmHg (SD 10.8mmHg; CI

92.9-100.6mmHg) for the placebo group, and 99.4 mmHg (SD 11.84mmHg; CI 95.0-103.7mmHg) for the OC group. Although this rise in pO₂ was not statistically significant, it does correspond well to the observed fall in pCO₂ levels in this group (see below) (SEE EXHIBIT E).

Ventilation and CO₂ Levels. Carbon Dioxide (CO₂) levels decreased after OC compared to placebo exposure in the sitting position. This small, but statistically significant finding suggests ventilation increased after OC exposure.

Mean end-tidal CO₂ levels were 38.0 mmHg (SD 3.5mmHg; CI 36.8-39.2mmHg) for the placebo group and 38.2 mmHg (SD 4.22mmHg; CI 36.6-39.7mmHg) for the OC group at baseline prior to exposure. At 1 minute after exposure, mean end-tidal CO₂ was 36.8 mmHg (SD 4.35mmHg; CI 35.3-38.3mmHg) for the placebo group, but dropped to 32.4 mmHg (SD 5.1mmHg; CI 30.5-34.3mmHg) for the OC group. At 5 minutes, mean levels were 36.5 mmHg (SD 5.1mmHg; CI 34.7-38.3mmHg) and 32.9 mmHg (SD 5.8mmHg; CI 30.7-35.0mmHg) for the placebo and OC groups, respectively. At 9 minutes, mean levels were 37.0 mmHg (SD 4.6mmHg; CI 35.4-38.6mmHg) and 35.2 mmHg (SD 5.9mmHg; CI 33.0-37.4mmHg) respectively. Similarly, mean arterial pCO₂ at 8 minutes was 39.4 mmHg (SD 3.9mmHg; CI 38.0-40.8mmHg) for the placebo group and 36.4 mmHg (SD 5.1mmHg; CI 34.5-38.3mmHg) for the OC group (SEE EXHIBIT F).

Cardiovascular Parameters. OC exposure led to small, but significant increases in both heart rate and blood pressure in subjects in the sitting position. In the placebo group, mean heart rate was 62.7 beats per minute (bpm) (SD 10.4; CI 59.1-66.4bpm) at baseline, 60.6 bpm (SD 7.8; CI 57.9-63.3 bpm) at 1 minute, 65.4 bpm (SD 9.4; CI 62.1-

68.7 bpm) at 5 minutes, and 60.3 bpm (SD 10.0; CI 56.9-63.8 bpm) at 9 minutes after exposure. In the OC group, mean heart was 67.8 bpm (SD 11.3; CI 58.4-66.2 bpm) at baseline, 77.4 bpm (SD 13.1; CI 72.6-82.2 bpm) at 1 minute, 69.0 bpm (SD 9.5; CI 65.5-72.5 bpm) at 5 minutes, and 64.6 bpm (SD 10.6; CI 60.7-68.5 bpm) at 9 minutes after exposure (see EXHIBIT G).

Mean arterial pressure (MAP, one-third of systolic pressure plus two-thirds of diastolic pressure) remained significantly elevated in the OC group at 3, 6 and 9 minutes after exposure in the sitting position. At baseline, mean MAP was 101.0 mmHg (SD 11.6; CI 96.9-105.0 mmHg) in the placebo group and 103.4 mmHg (SD 11.0; CI 99.3-107.4 mmHg) in the OC group. At 3 minutes, mean MAP was 103.8 mmHg (SD 9.1; CI 100.6-107.0 mmHg) in the placebo group, but increased to 116.8 mmHg (SD 12.8; CI 112.2-121.5mmHg) in the OC group. At 6 minutes, mean MAP was 100.7 mmHg (SD 10.4; CI 97.0-104.3 mmHg) and 112.1 mmHg (SD 13.5; CI 107.1-117.0mmHg), in the placebo and OC groups, respectively. At 9 minutes this difference remained, with mean MAP of 98.1 mmHg (SD 10.2; CI 94.6-101.7mmHg) and 107.8 (SD 13.7; CI 102.7-112.8 mmHg) in the two groups, respectively (See EXHIBIT H).

C. Effects of OC Exposure in the Restraint Position

Spirometric Findings. The restraint position resulted in a significant decrease in FVC and FEV1 similar to declines that have been previously reported. In the placebo group, mean % predicted FVC fell from a baseline of 101.9% (SD 10.0%; CI 98.4-105.4%) to 87.5% (SD 8.3%; CI 84.5-90.4%) at 1.5 minutes, and 87.9% (SD 8.3%; CI 84.9-90.8%) at 10 minutes into the restraint position. Mean % predicted FEV1 fell from

a baseline of 99.7% (SD 9.4%; CI 96.4-102.9%), to 83.2% (SD 9.7%; CI 79.7-86.7%) at 1.5 minutes, to 83.7% (SD 10.3%; CI 80.0-87.3%) at 10 minutes into the restraint position (see EXHIBITS C & D).

Exposure to OC made no statistical or clinical impact on pulmonary function in the restraint position. For the OC group, mean % predicted FVC fell from a baseline of 103.4% (SD 8.1%; CI 100.3-106.5%), to 87.5% (SD 7.3%; CI 84.7-90.3%) at 1.5 minutes and 87.2% (SD 7.3%; CI 84.3-90.0%) at 10 minutes. Similarly, mean % predicted FEV1 fell from a baseline of 101.1% (SD 8.0%; CI 98.0-104.2%), to 82.5% (SD 10.0%; CI 78.7-86.3%) at 1.5 minutes and 82.0% (SD 1.6%; CI 78.8-85.2%) at 10 minutes after OC exposure in the restraint position (see EXHIBIT C & D).

Oxygenation. Similar to the results in the sitting position, OC exposure followed by restraint did not result in statistically significant differences in oxygenation or evidence of hypoxemia. In the placebo group, mean oxygen saturation was 99.4% (SD 1.0%; CI 98.1-99.7%) at baseline, 97.9% (SD 2.3%; CI 97.1-98.7%) at 1 minute, 98.0% (SD 1.9%; CI 97.7-98.9%) at 5 minutes, and 97.4% (SD 3.7%; CI 96.1-98.6%) at 10 minutes into the restraint position. In the OC group, mean oxygen saturation was 99.3% (SD 1.1%; CI 98.9-99.7%) at baseline, 98.1% (SD 2.8%; CI 93.7-99.9%) at 1 minute, 98.8% (SD 1.3%; CI 98.3-99.3%) at 5 minutes, and 98.4% (SD 2.0%; CI 97.6-99.2%) at 9 minutes into the restraint position. Similarly, mean arterial pO₂ levels at 8 minutes were 90.2 mmHg (SD 10.2; CI 86.6-93.8 mmHg) in the placebo group and 90.0 mmHg (SD 15.2; CI 84.8-95.8 mmHg) in the OC group (see EXHIBIT E).

Ventilation and CO₂ Levels. Again similar to the results from the sitting position trials, CO₂ levels decreased slightly after OC exposure, suggesting an increase in

ventilation after OC. At baseline prior to exposure, mean CO₂ levels were 37.7 mmHg (SD 4.0; CI 36.3-39.1 mmHg) and 36.3 mmHg (SD 7.7; CI 36.0-39.0 mmHg) for the placebo and restraint groups, respectively. At 1 minute after exposure, levels were 38.8 mmHg (SD 4.1; CI 37.4-40.3 mmHg) in the placebo group, but decreased to 36.7 mmHg (SD 6.4; CI 34.3-39.2 mmHg) in the OC group. At 5 minutes, levels were 39.1 mmHg (SD 4.0; CI 37.7-40.5 mmHg) and 36.6 mmHg (SD 5.4; CI 34.5-38.7) and at 9 minutes, 39.5 mmHg (SD 3.8; CI 38.2-40.8 mmHg) and 37.7 mmHg (SD 4.7; CI 35.9-39.5 mmHg), respectively. Similarly, mean arterial pCO₂ levels at 8 minutes were 40.9 mmHg (SD 4.3; CI 39.3-42.4 mmHg) in the placebo group, and 39.1 mmHg (SD 5.2; CI 37.2-41.1 mmHg) in the OC group (see EXHIBIT F).

Cardiovascular Parameters. As with sitting position, there was a slight increase in HR and larger increase in MAP after OC exposure in the restraint position. For the placebo group, mean HR was 62.3 bpm (SD 11.3; CI 58.4-66.2 bpm) at baseline, 70.4 bpm (SD 10.9 bpm; 66.5-74.2 bpm) at 1 minute, 66.5 bpm (SD 12.2; CI 62.2-70.8 bpm) at 5 minutes, and 62.6 bpm (SD 8.6; CI 59.6-65.6 bpm) at 9 minutes. For the OC group, mean HR was 64.8 bpm (SD 8.4; CI 61.6-68.0 bpm) at baseline, 71.1 bpm (SD 13.9; CI 65.8-76.4 bpm) at 1 minute, 66.1 bpm (SD 9.8; CI 62.33-70.0 bpm) at 5 minutes, and 65.1 bpm (SD 7.3; CI 62.3-67.9 bpm) at 9 minutes (see EXHIBIT G).

Mean MAP at baseline prior to exposure was 102.2 mmHg (SD 9.4; CI 98.9-105.4 mmHg) and 102.7 mmHg (SD 11.0; CI 98.5-106.9 mmHg) in the placebo and OC groups, respectively. One minute after exposure, mean MAP was 102.0 mmHg (SD 11.4; CI 98.1-106.0 mmHg) in the placebo group, but had increased to 110.7 mmHg (SD 10.6; CI 106.7-114.7 mmHg) in the OC group. At 6 minutes, the mean MAP was 99.1 mmHg

(SD 10.0; CI 95.6-102.6 mmHg) and 108.2 mmHg (SD 12.42; CI 103.5-112.9 mmHg), and at 9 minutes, 100.1 mmHg (SD 11.52; CI 96.1-104.2 mmHg) and 110.0 mmHg (SD 10.6; CI 106.0-114.1 mmHg), respectively (see EXHIBIT H).

D. Effects of Other Factors

The effect of additional factors, body weight and the potential for underlying lung abnormalities, was assessed.

Body Weight. There were 7 subjects classified as overweight (BMI>28 kg/m²) in this study. Spirometric findings mirrored those for the entire study population. Mean % predicted FVC were similar between placebo and OC groups in the sitting position (103.7% vs. 103.7% at baseline; 104.4% vs. 104.3% at 1.5 minutes; 104.5% vs. 104.4% at 10 minutes, respectively). Mean % predicted FEV1 were similar between placebo and OC groups in the sitting position (102.8% vs. 102.9% at baseline; 103.0% vs. 104.3% at 1.5 minutes; 103.0% vs. 102.5% at 10 minutes, respectively).

Mean % predicted FVC were also similar between placebo and OC groups in the restraint position with typical decreases in pulmonary function resulting from body position (102.1% vs. 103.3% at baseline; 87.0% vs. 87.6% at 1.5 minutes; 85.8% vs. 87.4% at 10 minutes, respectively). Mean % predicted FEV1 also revealed typical declines between the placebo and OC groups with restraint (100.9% vs. 101.3% at baseline; 84.0% vs. 84.9% at 1.5 minutes; 83.8% vs. 83.8% at 10 minutes, respectively).

Mean oxygen saturation results from the overweight subjects were similar to the overall study results as well. In the sitting position, oxygen saturations were similar for the placebo and OC groups (99.3% vs. 99.1% at baseline; 99.0% vs 99.3% at 1 minute;

98.6% vs. 99.3% at 5 minutes; 99.3% vs. 98.7% at 9 minutes, respectively). Results were similar for the restraint position (98.6% vs. 99.3% at baseline; 97.6% vs. 97.0% at 1 minute; 97.0% vs. 98.4% at 5 minutes; 96.7% vs. 98.0% at 9 minutes).

Interestingly, in the overweight group, arterial pO₂ levels revealed a slightly lower pO₂ level in the placebo group that improved with OC exposure (85.4 mmHg vs. 101.6 mmHg, respectively) in the sitting position. In the restraint position, there was no improvement (82.3 mmHg vs. 82.8 mmHg, respectively).

Mean CO₂ levels in the overweight group mirrored those found in the overall study population. In the sitting position, there was no evidence of hypercapnia, or hypoventilation, with placebo or OC exposure (mean end-tidal CO₂ levels of 38.4 vs. 37.6 mmHg at baseline; 36.0 vs. 31.1 mmHg at 1 minute; 36.1 vs. 31.1 mmHg at 5 minutes; 37.9 vs. 33.4 mmHg at 9 minutes; mean arterial pCO₂ at 8 minutes of 41.6 vs. 35.0 mmHg, respectively). In the restraint position, findings were similar (mean end-tidal CO₂ levels of 38.0 vs. 37.8 mmHg at baseline; 37.9 vs. 35.8 mmHg at 1 minute; 38.7 vs. 36.4 mmHg at 5 minutes; 39.5 vs. 36.8 mmHg at 9 minutes; mean arterial pCO₂ levels at 8 minutes of 41.6 vs. 38.7 mmHg, respectively).

Heart rate and mean arterial pressure in the overweight subjects were similar to those of the main study population. In the sitting position, mean HR revealed a slight increase with OC exposure (59.9 vs. 71.4 bpm at baseline; 58.6 vs. 86.9 bpm at 1 minute; 62.6 vs. 75.6 bpm at 5 minutes; 58.7 vs. 69.7 bpm at 9 minutes for the placebo and OC groups, respectively). In the restraint position, a smaller difference was found (60.5 vs. 67.5 bpm at baseline; 71.4 vs. 71.0 bpm at 1 minute; 65.7 vs. 67.2 bpm at 5 minutes; 61.4 vs. 66 bpm at 9 minutes, respectively).

Mean arterial pressure markedly increased with OC exposure in both the sitting and restraint groups, similar to the findings for the entire subject population (in the sitting position: 108.8 vs. 107.7 mmHg at baseline, 107.8 vs. 120.8 mmHg at 3 minutes, 106.0 vs. 118.7 mmHg at 6 minutes, 103.3 vs. 111.6 mmHg at 9 minutes; and in the restraint position: 104.9 vs. 105.1 mmHg at baseline, 104.0 vs. 112.2 mmHg at 3 minutes, 102.3 vs. 110.6 mmHg at 6 minutes, 103.5 vs. 111.2 mmHg at 9 minutes for the placebo and OC groups, respectively).

Potential for Respiratory Abnormalities. Eight (8) subjects had either a history of lung disease, such as asthma, history of smoking, or used a respiratory inhaler medication on a regular basis. A separate analysis was performed on this group and resulted in findings similar to the overall subject population.

Pulmonary function testing revealed typical declines related to body position, but showed no differences between placebo and OC exposure. In the sitting position, mean % predicted FVC were similar in the placebo and OC groups (105.6 vs. 107.6% at baseline, 106.2 vs. 105.4% at 1.5 minutes; 105.4 vs. 101.1% at 10 minutes, respectively) as were mean % predicted FEV1 values (104.7 vs. 105.4% at baseline; 105.3 vs. 105.8% at 1.5 minutes; 104.9 vs. 105.7% at 10 minutes, respectively). In the restraint position, there were declines typical of body position, but no changes from OC exposure for mean % predicted FVC (105.9 vs. 106.9% at baseline, 91.0 vs. 90.6% at 1.5 minutes, 91.3 vs. 90.9% at 10 minutes) and mean % predicted FEV1 (104.2 vs. 105.7% at baseline, 87.9 vs. 88.7% at 1.5 minutes; 90.2 vs. 87.9% at 10 minutes for placebo and OC groups, respectively).

Oxygenation in this group of 8 subjects was similar to those of the overall subject population. In the sitting position, there was no evidence of hypoxemia (mean oxygen saturations of 99.4 vs. 99.4% at baseline, 99.1 vs. 99.6% at 1 minute, 98.4 vs. 99.6% at 5 minutes, 99.3 vs. 99.6% at 9 minutes; mean arterial pO₂ at 8 minutes of 101.7 vs. 96.6 mmHg for the placebo and OC groups, respectively). In the restraint position, findings were similar (mean oxygen saturations of 99.4 vs. 99.0% at baseline, 97.4 vs. 99.0% at 1 minute, 98.0 vs. 98.5% at 5 minutes, 95.9 vs. 98.8% at 9 minutes; mean arterial pO₂ at 8 minutes of 91.0 vs. 90.0 mmHg, respectively).

CO₂ levels for these 8 subjects also were similar to those found for the overall study population. In the sitting position, there was no evidence of hypercapnia or hypoventilation (mean end-tidal CO₂ levels of 38.1 vs. 37.9 mmHg at baseline, 37.8 vs. 31.6 mmHg at 1 minute, 38.3 vs. 33.7 mmHg at 5 minutes, 37.4 vs. 36.6 mmHg at 9 minutes; mean arterial pCO₂ at 8 minutes of 40.4 vs. 38.0 mmHg for the placebo and OC groups, respectively). In the restraint position, findings were similar (38.0 vs. 39.0 mmHg at baseline, 39.3 vs. 37.7 mmHg at 1 minute, 38.8 vs. 38.0 mmHg at 5 minutes, 39.3 vs. 39.5 mmHg at 9 minutes; mean arterial pCO₂ at 8 minutes of 40.9 vs. 42.2 mmHg, respectively).

Mean heart rate and blood pressure in this group of 8 subjects were similar to the findings for the overall subject population. There were variable findings regarding mean HR, but a marked elevation in MAP with OC exposure. In the sitting position, there was a slight increase in HR (64.5 vs. 66.6 bpm at baseline, 60.6 vs. 77.3 bpm at 1 minute, 68.1 vs. 72.0 bpm at 5 minutes, 58.8 vs. 67.9 bpm at 9 minutes for placebo vs. OC respectively), but a marked increase in MAP (104.8 vs. 106.4 mmHg at baseline, 109.9

vs. 120.8 mmHg at 3 minute, 105.7 vs. 120.5 mmHg at 6 minutes, 101.3 vs. 114.5 mmHg at 9 minutes, respectively).

In the restraint position, HR findings were variable (61.6 vs. 63.2 bpm at baseline, 77.7 vs. 76.5 bpm at 1 minute, 73.5 vs. 68.3 at 5 minutes, 64.3 vs. 65.8 bpm at 9 minutes for the placebo and OC groups, respectively), whereas MAP increased with OC exposure (106.5 vs. 106.9 mmHg at baseline, 110.3 vs. 115.7 mmHg at 3 minutes, 104.6 vs. 114.2 at 6 minutes, 107.1 vs. 115.6 at 9 minutes, respectively).

V. ANALYSIS AND DISCUSSION

The goal of this study was to assess the safety of OC exposure by itself and in combination with physical restraint. Specifically, we sought to determine if OC spray inhalation resulted in significant respiratory compromise such that individuals might be at risk for significant injury and even death.

We performed a randomized, cross-over controlled laboratory study in human subjects comparing the effects of OC spray and placebo followed by the sitting and restraint positions. Subjects performed 4 different trials (varying exposure and position) such that each served as their own control. The cross-over design eliminated potential confounding factors between control and experimental groups. In addition, randomization eliminated potential differences that may have resulted from the sequence of trials.

This study had 2 main objectives as well as 3 additional objectives addressing related issues regarding the physiologic effects of OC exposure.

A. MAIN OBJECTIVES

1. ***To determine if OC spray inhalation in the sitting position results in clinically significant respiratory compromise as measured by pulmonary function testing and assessment of oxygenation and ventilation.***

In this study, we found no evidence that OC spray inhalation and exposure resulted in any respiratory compromise in the sitting position. Statistically, there was no significant difference in % predicted for FVC or FEV1 on pulmonary function testing at

1.5 and 10 minutes after exposure between the OC and placebo groups. Clinically, these spirometric parameters remained within the range of normal (above 80% of predicted) at 1.5 and 10 minutes after OC exposure and inhalation.

Moreover, there was no difference between OC and placebo groups in terms of oxygenation, and no evidence of hypoxemia to suggest respiratory compromise after OC inhalation. There was also no evidence of hypercapnia, or CO₂ retention to suggest poor ventilation, after OC inhalation. In fact, CO₂ levels were lower in the OC group, suggesting an increase in ventilation after OC inhalation.

Our findings are consistent with a number of other human clinical studies that have examined the effect of nebulized capsaicin, the active agent of OC spray, on respiratory function. As discussed above, these studies reported transient changes in airway resistance, but no evidence of sustained bronchoconstriction beyond 1-2 minutes after the exposure.^{20,21,22}

While nebulized capsaicin has been studied extensively, this study assessed pulmonary and respiratory function after exposure to a commercially-available OC spray used by law enforcement agencies nationwide. In finding no evidence of respiratory compromise, this clinical experimental study in humans lends credence to the large retrospective field studies that have found little evidence suggesting OC causes significant respiratory injury.^{29,30}

2. To determine if OC spray inhalation in the restraint position results in clinically significant respiratory compromise as measured by pulmonary function testing and assessment of oxygenation and ventilation.

Our findings concerning the restraint position are consistent with our previous work on respiratory function and restraint. In our earlier studies, we found declines in pulmonary function (FVC and FEV1) with restraint, but no evidence of hypoxemia, hypercapnia or hypoventilatory respiratory dysfunction.⁴⁶

In this study, we found no evidence that OC exposure resulted in any additional change in respiratory function in the restraint position. In both the OC and placebo groups, we saw declines in % predicted FVC and % predicted FEV1 once subjects were placed in the prone maximal restraint position. While these declines indicate a restrictive pulmonary function pattern, mean spirometric measurements remained within the normal range. Moreover, there were no statistical differences between the OC and placebo groups relative to these declines in % predicted FVC and % predicted FEV1.

In additionally, just as we had found with in the sitting position, there was no difference in terms of oxygenation in the restraint position between the OC and placebo groups. There was also no difference in CO2 levels between the two groups in the restraint position, again suggesting that OC exposure had no adverse effect on ventilatory function in restrained subjects. Accordingly, OC inhalation had no effect on the pulmonary function changes, oxygenation or ventilation associated with restraint.

B. ADDITIONAL OBJECTIVES

- 3. Whether OC spray by itself or in combination with positional restraint results in any hemodynamic compromise as measured by pulse rate and blood pressure.*

In this study, we found OC exposure resulted in a small increase in heart rate after exposure when compared with placebo. While statistically significant, this difference is of probably of no clinical importance as mean heart rates for all groups, regardless of exposure or position, remained in the 60 to 80 bpm range, well within normal limits.

Mean arterial pressure however, was significantly elevated after OC exposure when compared to placebo in both the sitting and restraint positions. This difference, though small, persisted at 3, 6, and 9 minutes after exposure.

The cardiovascular effects of capsaicin remain unclear and complex.⁵⁴ Animal studies have shown capsaicin can result in both hypertension and hypotension.^{55,56} Accordingly, some investigators contend that capsaicin can precipitate bradycardia and hypotension, similar to the Bezold-Jarish reflex.⁵⁷ Others argue that OC exposure causes acute elevations in blood pressure, leading to potential hypertensive crises.⁵ Our study also suggests an elevation in blood pressure. However, the etiology of this elevation remains uncertain and may simply result from the discomfort and pain associated with OC exposure.

4. Whether body size and weight influence the effects of OC spray in regards to respiratory and pulmonary function as measured by pulmonary function testing and assessment of oxygenation and ventilation.

In this study, we found no evidence that OC spray inhalation and exposure resulted in respiratory compromise in subjects with BMI > 28 kg/m². In this subgroup, we found that OC exposure had no effect on pulmonary function in the sitting or restraint

positions. There was also no evidence of hypercapnia or hypoventilation for this group of subjects after OC inhalation in either the sitting or restraint positions.

Interestingly, while transcutaneous measurements of oxygen saturation were normal regardless of exposure or position, subjects in this group revealed a mild drop in pO₂ levels (on arterial blood gas sampling) in both sitting and restraint positions after placebo exposure. Arterial oxygenation improved with OC exposure in the sitting position, but did not change in the restraint position.

Morbid obesity can lead to a restrictive pulmonary dysfunction and increased airway resistance, as well as hypoxemia and hypercapnia, particularly when associated with obstructive sleep apnea.^{58,59} In our study, no subjects were morbidly obese (no BMI > 32 kg/m²). Of those who were overweight (BMI > 28 kg/m²), there was no evidence of restrictive dysfunction in the sitting or even restraint position with OC or placebo exposure. In this group, OC exposure did not hypoxemia or hypoventilation in either the sitting or restraint position, but did seem to improve oxygenation for the sitting position. Clearly, however, these conclusions must be tempered by the small number of subjects studied in this subanalysis and the fact that none of our subjects were morbidly obese.

5. Whether pulmonary disease (such as asthma), the use of respiratory inhaler medications, or history of smoking tobacco influence the effects of OC spray in regards to respiratory and pulmonary function as measured by pulmonary function testing and assessment of oxygenation and ventilation.

In this study, we found no evidence that OC spray inhalation and exposure resulted in respiratory compromise in subjects with the potential for pulmonary abnormalities (history of lung disease, asthma, smoking, and respiratory inhaler medication use). In this subgroup, we found that OC exposure had no effect on pulmonary function in the sitting or restraint positions. There was also no evidence of hypoxemia, hypercapnia or hypoventilation after OC inhalation for this group in either the sitting or restraint positions.

The effect of capsaicin in those with lung disease, particularly asthma, remains controversial. It has been suggested that asthmatics may have increased responsiveness to the respiratory effects of capsaicin and OC, possibility due to increased sensitivity of nerves in the lung parenchyma.^{26,60} Other clinical studies suggest there is little or no difference in the effects of capsaicin on airway resistance and bronchoconstriction in smokers, asthmatics or those with respiratory infections.^{20,61} Our findings from this small subgroup of subjects exposed to OC seem to support the latter contention.

While our results suggest OC exposure does not result in respiratory dysfunction in those with potential respiratory abnormalities at baseline, it is important to note that this study cannot make definitive conclusions due to the small number of subjects in this subgroup.

C. STUDY LIMITATIONS

This study was performed in a clinical laboratory on a healthy population of subjects. This study does not attempt to replicate all the conditions that may be

encountered in the law enforcement field setting where OC spray and positional restraint are employed.

Field subjects are often in a state of extreme agitation and “excited delirium” as a result of underlying psychiatric disease or intoxication from recreational drugs. Subjects are often involved in violent, physical struggles prior to, during and after the use of OC spray or positional restraint. There has been speculation that subjects in the field undergo extreme levels of exertion leading to exhaustion that may impact pulmonary function. While previous studies have attempted to replicate exertion and struggle,^{46,48} it is unlikely that all conditions, particularly the physiologic effects of psychological stress, psychological stress and trauma that occur in the field could be reproduced in the laboratory.

Moreover, as this study focused on inhalational exposure, all subjects had goggles to reduce ocular OC exposure. Eye irritation and pain from OC may exacerbate the physiologic stress of field subjects, which was not assessed by this study. In addition, when restrained, subjects were placed on a medical examination table rather than on a hard surface that often occurs in the field setting.

We attempted to replicate OC exposure in the field as much as possible in the laboratory setting. In doing so, exact capsaicin dosing was not standardized. Rather, subjects were exposed to a standard 1 second spray directed from 5 feet away as they might in the field setting (though impedance monitoring assured that subjects did not simply hold their breath during the exposure period). The duration of spray and exposure distance were dictated by both manufacturer recommendations and local police policies regarding the use of OC. As a result, we did not study spray exposures longer than 1

second that can occur in the field setting. Moreover, we did not study exposures from close distances, which also may occur in the field. However, spray distances less than 5 feet generally do not allow for adequate aerosolization of OC and likely reduce the amount of inhalational exposure.

Exposure in the box was limited to 5 seconds while in the laboratory. While this may seem a short period of time, spray in the field usually occurs in an open setting where OC dissipates rapidly. Moreover, by containing the spray within the exposure box, it is likely that subjects were exposed to a much higher concentration of capsaicin than might have occurred in the open air. The concentration of active capsaicinoids (approximately 26 mg delivered per spray into a $2 \times 10^6 \text{ cm}^3$ space) in our study was similar if not higher than other clinical studies on capsaicin inhalation.

We did not study repeated OC spray exposures that commonly occur in the field setting. We also used an aerosol form of OC spray, rather than the liquid or foam forms that are also used by law enforcement agencies. We believe the aerosol form was more likely to be inhaled than other forms and, thus more appropriate for our study on the respiratory effects of OC.

While we closely monitored subjects for 1 hour after each trial, as well as had follow-up with the SDRPSTI staff for any significant adverse reactions, we did not assess for any other delayed or long-term effects from exposure that may have occurred. Moreover, this study does not address issues regarding the potential for long term complications (such as mutagenic or carcinogenic properties) from chronic, occupational exposure to OC.

Finally, we must again stress the limited nature of the additional analyses performed on the subgroups of subjects who were overweight or had potential respiratory abnormalities. These groups were small in number and our analyses lacked sufficient statistical power to make any definitive conclusive findings. Moreover, as our study population was recruited from training cadets at the local police academy, our subjects were generally healthy, young individuals.

D. IMPLICATIONS FOR LAW ENFORCEMENT

This study provides scientific and physiologic data assessing the safety of OC spray by itself and in combination with positional restraint, commonly used by law enforcement personnel to subdue violent, dangerous suspects in the field. This study focused on two force methods, namely OC and prone maximal restraint, directly associated with law enforcement procedures. Findings from this study directly impact upon policing practices and policies regarding the use of these force options.

This study supports the contention that OC spray inhalation does not represent any significant risk to subjects in terms of respiratory and pulmonary function even when combined with positional restraint. In this study, OC exposure did not result in any evidence of pulmonary dysfunction, hypoxemia, or hypoventilation, in either the sitting or restraint positions. These findings also applied to subgroups of overweight subjects and those with potential respiratory abnormalities.

This study provides new information on the issue of “in-custody” deaths. Determining a cause of death in these cases is often difficult as there are few pathologic findings at autopsy.^{7,9} Accordingly, “in-custody” deaths have been attributed to a number of possible causes, including respiratory compromise from OC exposure, positional asphyxia, drug and alcohol intoxication, excited delirium, psychiatric illness, cardiopulmonary disease, and obesity. This study indicates that OC inhalation and exposure does not cause significant respiratory injury, and should not lead to an increased risk for respiratory compromise, arrest and death.

Our findings will aid law enforcement agencies by providing data supporting the safety of OC spray, even in combination with restraint. First, this study will provide law

enforcement personnel some measure of comfort in the knowledge that they are employing force methods, namely OC spray, that have been tested and found safe on humans in clinical studies.

In addition, these findings may improve the relationship between local agencies and their communities as the general public will be aware that officers in their communities are employing force methods that have been rigorously studied in a clinical laboratory on human subjects. Moreover, questions regarding OC use in cases of custody deaths will less likely contribute to the public controversy and contentiousness that often follows these cases.

Second, this study will aid law enforcement agencies when facing accusations of excessive force based on the unfounded contention that OC exposure results in respiratory compromise. Data from this study will assist law enforcement agencies in deterring and defending themselves from litigation that can negatively impact the well-being and morale of their agencies as a whole and, more directly, their personnel and officers in the field.

Third, on a general public policy level, this study provides solid scientific evidence regarding the search for safer restraint methods. In the past, controversy regarding police force methods and restraint have been based on anecdote and case reports, rather than scientific study of human physiology. While many other controversies remain, such as the impact of physiologic and psychological stress, external weight compression during restraint, and the cardiovascular effects of stress and restraint, this study provides a physiologic and scientific basis from which to investigate and assess law enforcement force methods and custody restraint procedures.

CONCLUSIONS

We conducted a randomized, cross-over controlled study investigating the effects of OC inhalation and prone maximal restraint on respiratory function in human subjects. In our subjects, OC exposure resulted in no evidence of pulmonary dysfunction, hypoxemia, or hypoventilation when compared to placebo in both the sitting and restraint positions. Our findings support the contention that OC spray use by law enforcement personnel in the field does not result in respiratory compromise or increased risk for respiratory arrest and death in exposed subjects.

VI. ENDNOTES

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EXHIBIT A

Exposure Box (2 views)

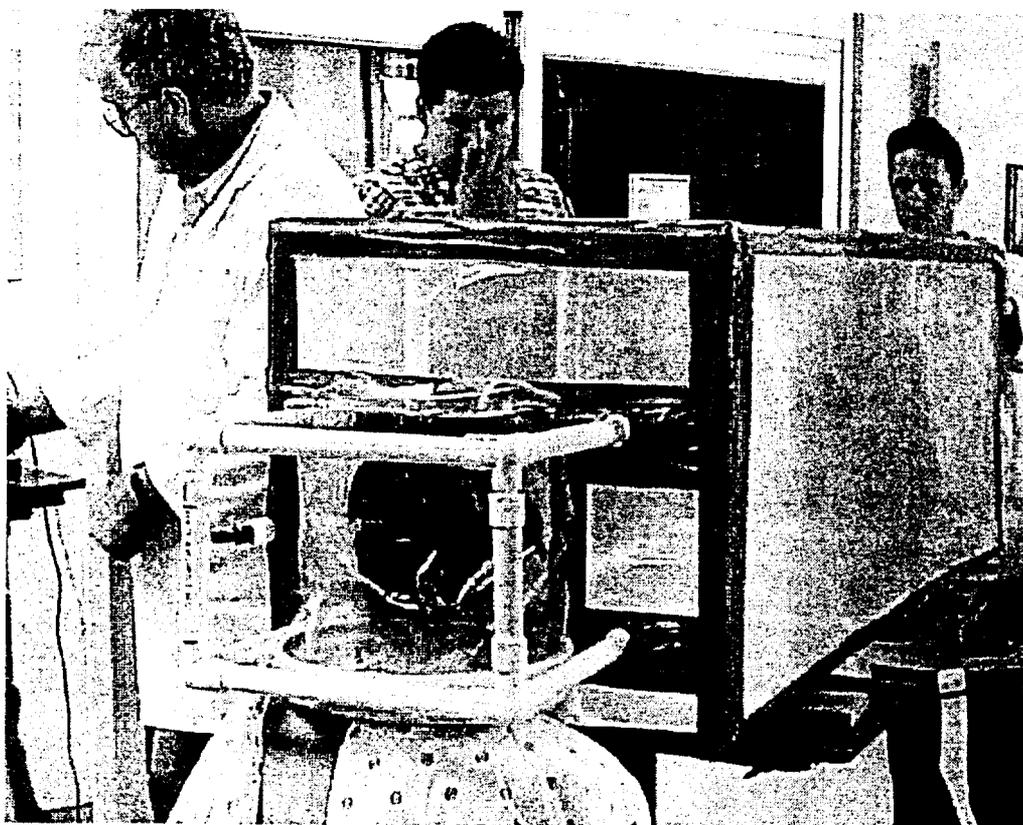
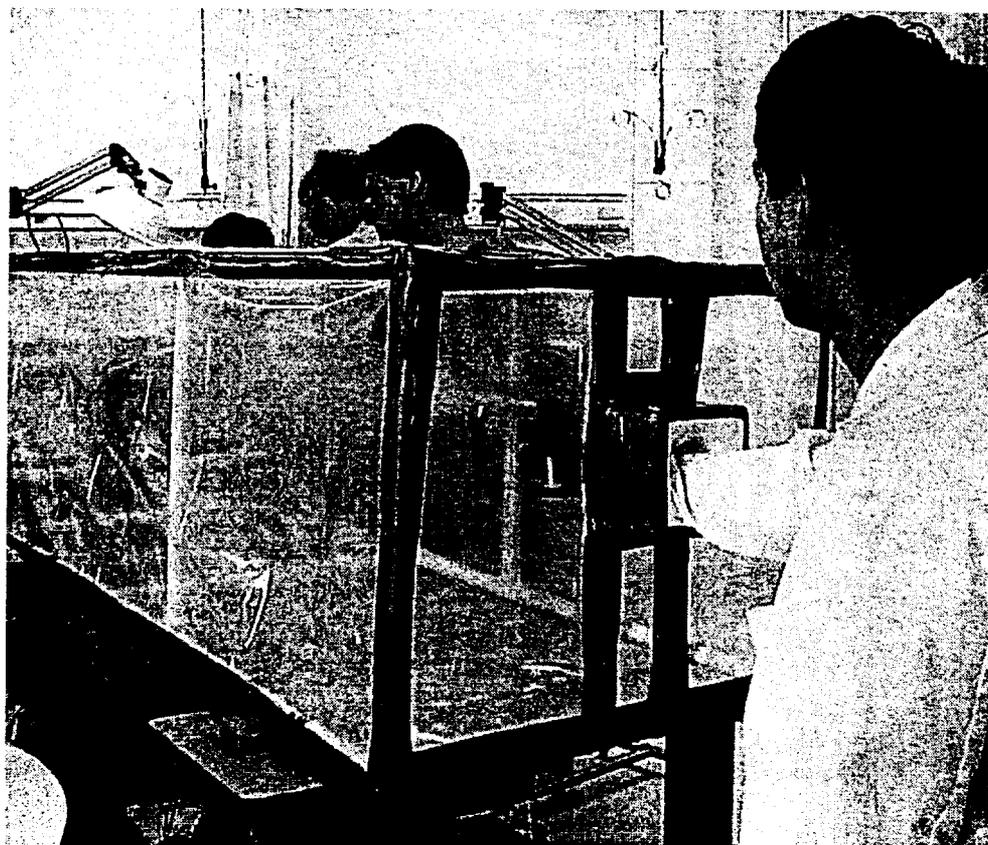


EXHIBIT B

Trial Study Procedure

<u>Time</u>	<u>Procedure</u>
<i>Baseline/Preparation</i>	Trial exposure (OC or Placebo) and position (Sitting or Restraint) determined. Baseline Pulmonary Function Testing (FVC and FEV1) performed. Continuous monitoring devices placed on Subject. Baseline oxygen saturation, end-tidal CO2, heart rate and blood pressure recorded.
<i>0 time</i>	Subject placed in Hood/Exposure Box 1 second of OC or placebo spray delivered into box. Impedance monitoring assesses inspiration/expiration
<i>5 seconds</i>	Subject removed from Hood/Exposure Box Subject placed in position (Sitting or Restraint).
<i>1 minute</i>	Oxygen saturation, end-tidal CO2 level and heart rate recorded.
<i>1.5 minutes</i>	Pulmonary Function Testing (FVC and FEV1) performed.
<i>3 minutes</i>	Blood Pressure recorded.
<i>5 minutes</i>	Oxygen saturation, end-tidal CO2 level and heart rate recorded.
<i>6 minutes</i>	Blood Pressure recorded.
<i>8 minutes</i>	Arterial blood gas sample drawn from radial artery at wrist. pH, pCO2, and pO2 levels determined.
<i>9 minutes</i>	Oxygen saturation, end-tidal CO2 level and heart rate recorded. Blood Pressure recorded.
<i>10 minutes</i>	Pulmonary Function Testing (FVC and FEV1) performed.
<i>Trial Completed</i>	Subject released from position. Residual exposure (OC or placebo) washed off. Subject allowed to rest a minimum of 1 hour.

Exhibit C: FVC by Exposure and Position

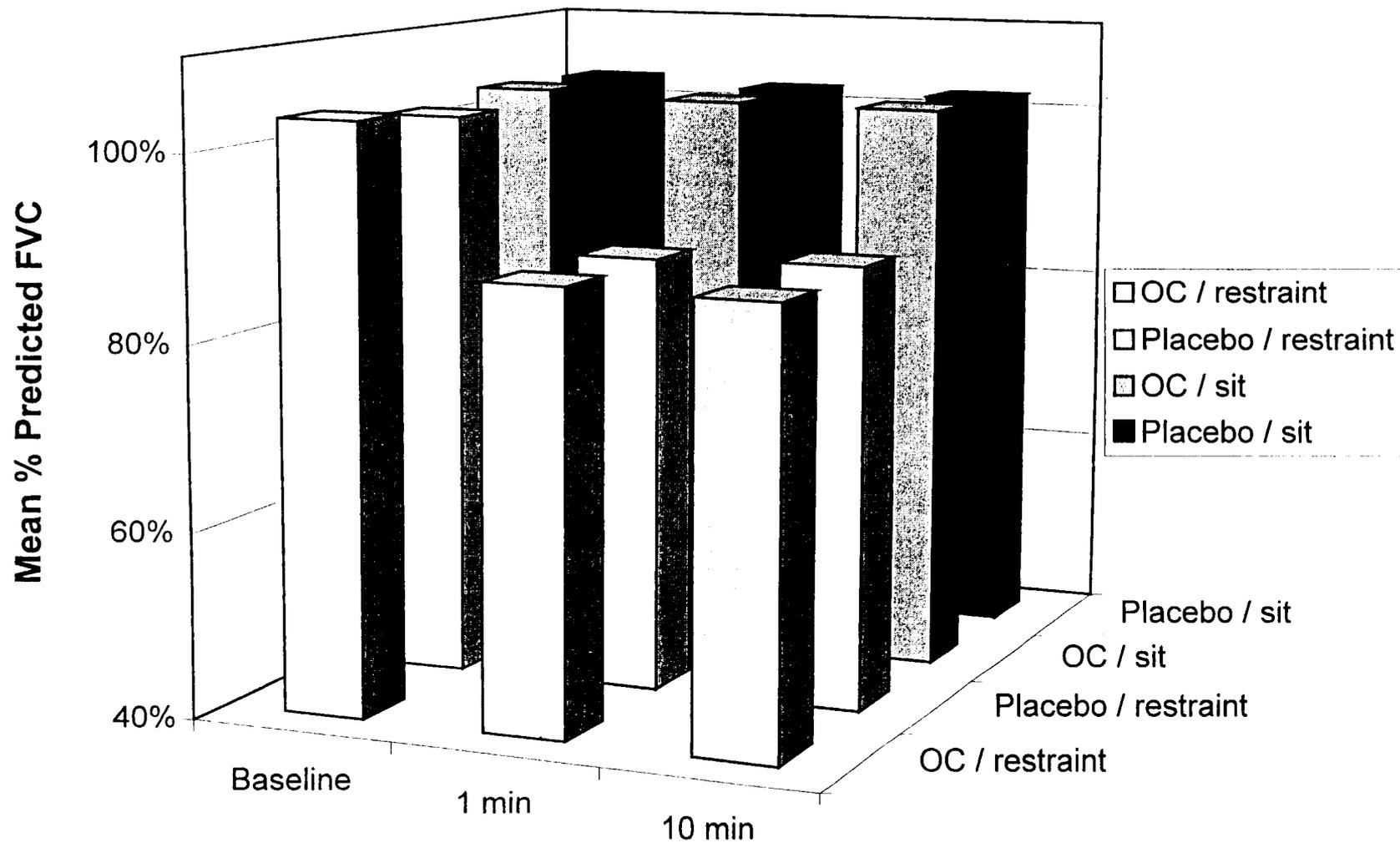


Exhibit D: FEV1 by Exposure and Position

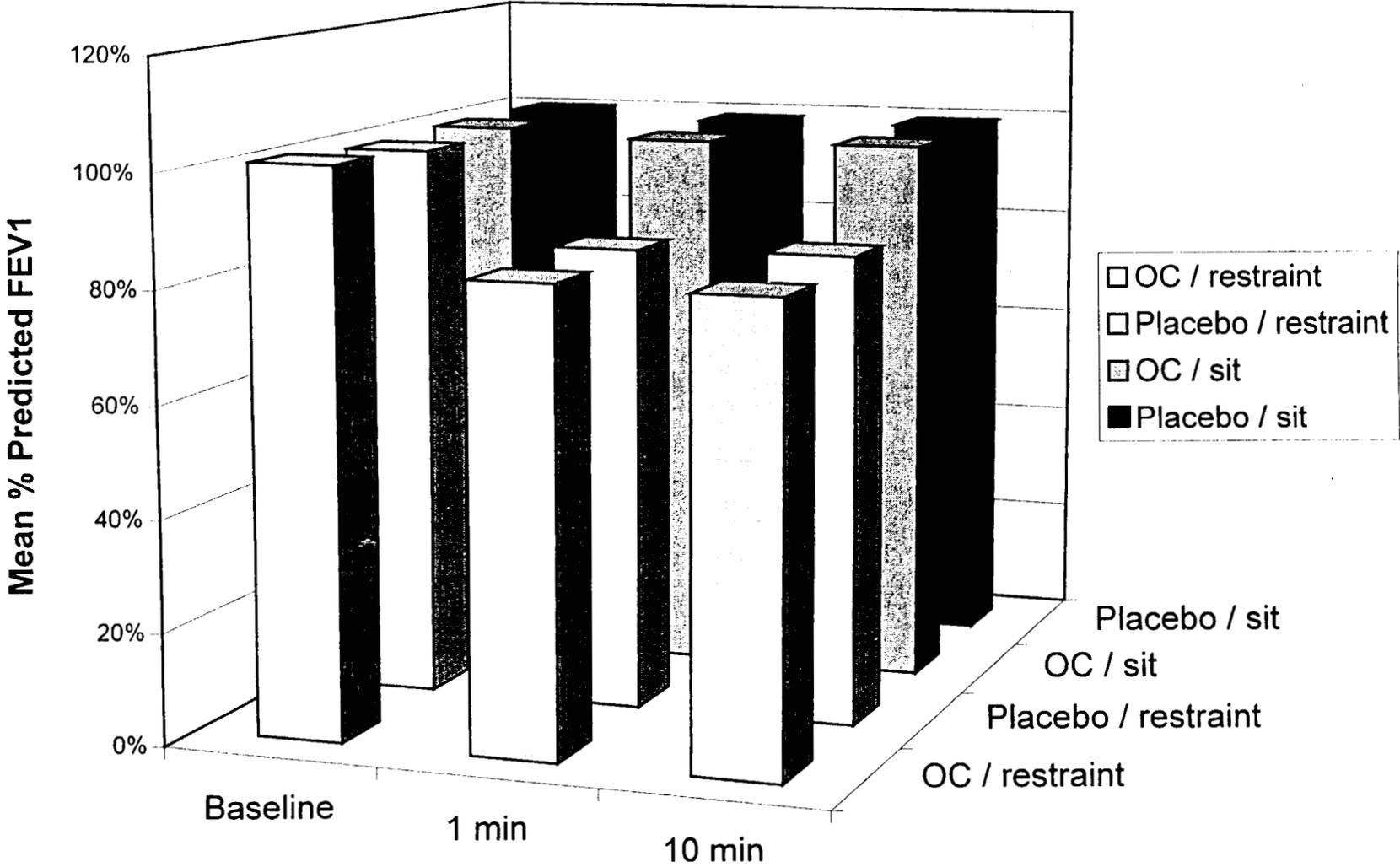


Exhibit E: Oxygenation by Exposure and Position

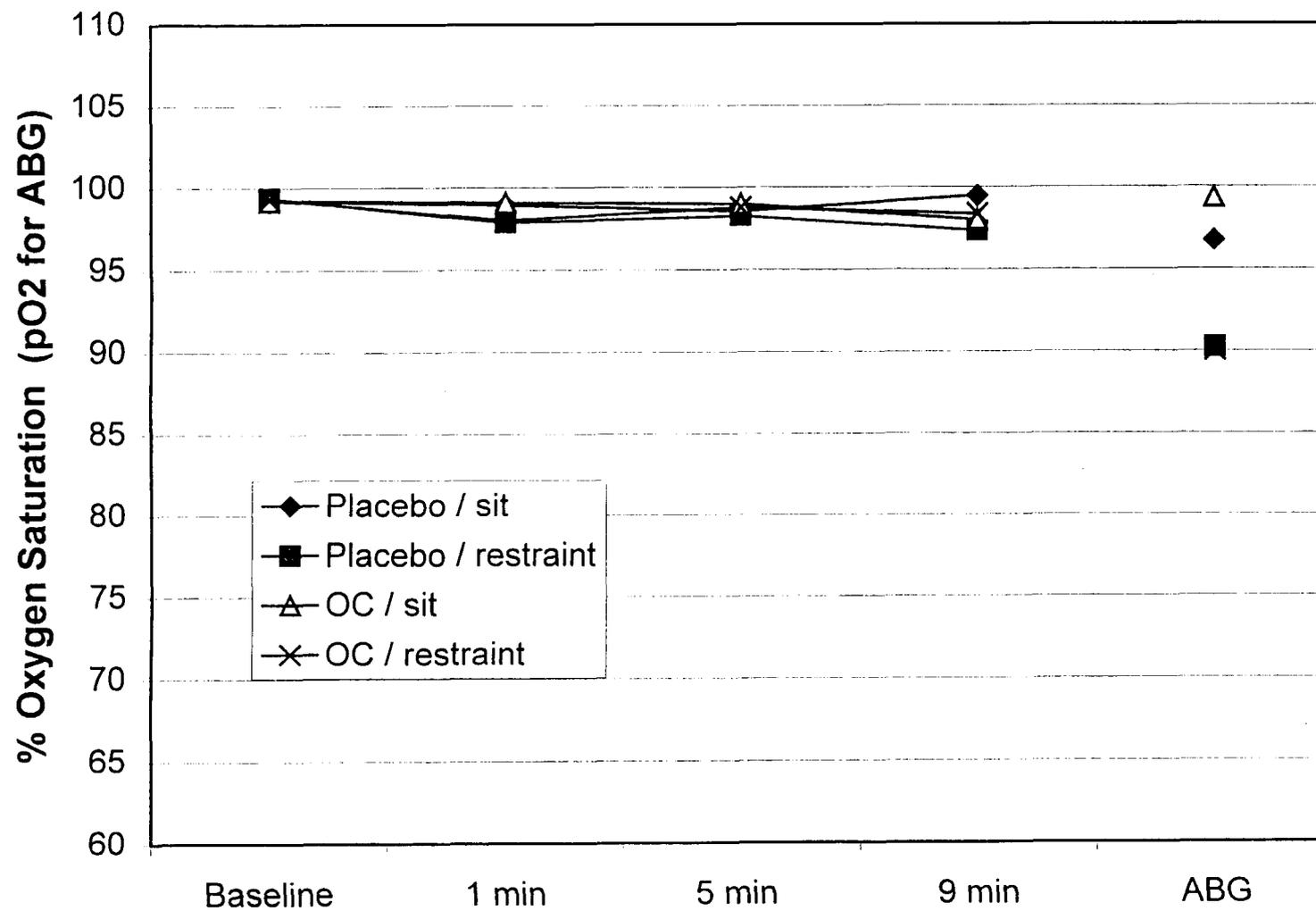
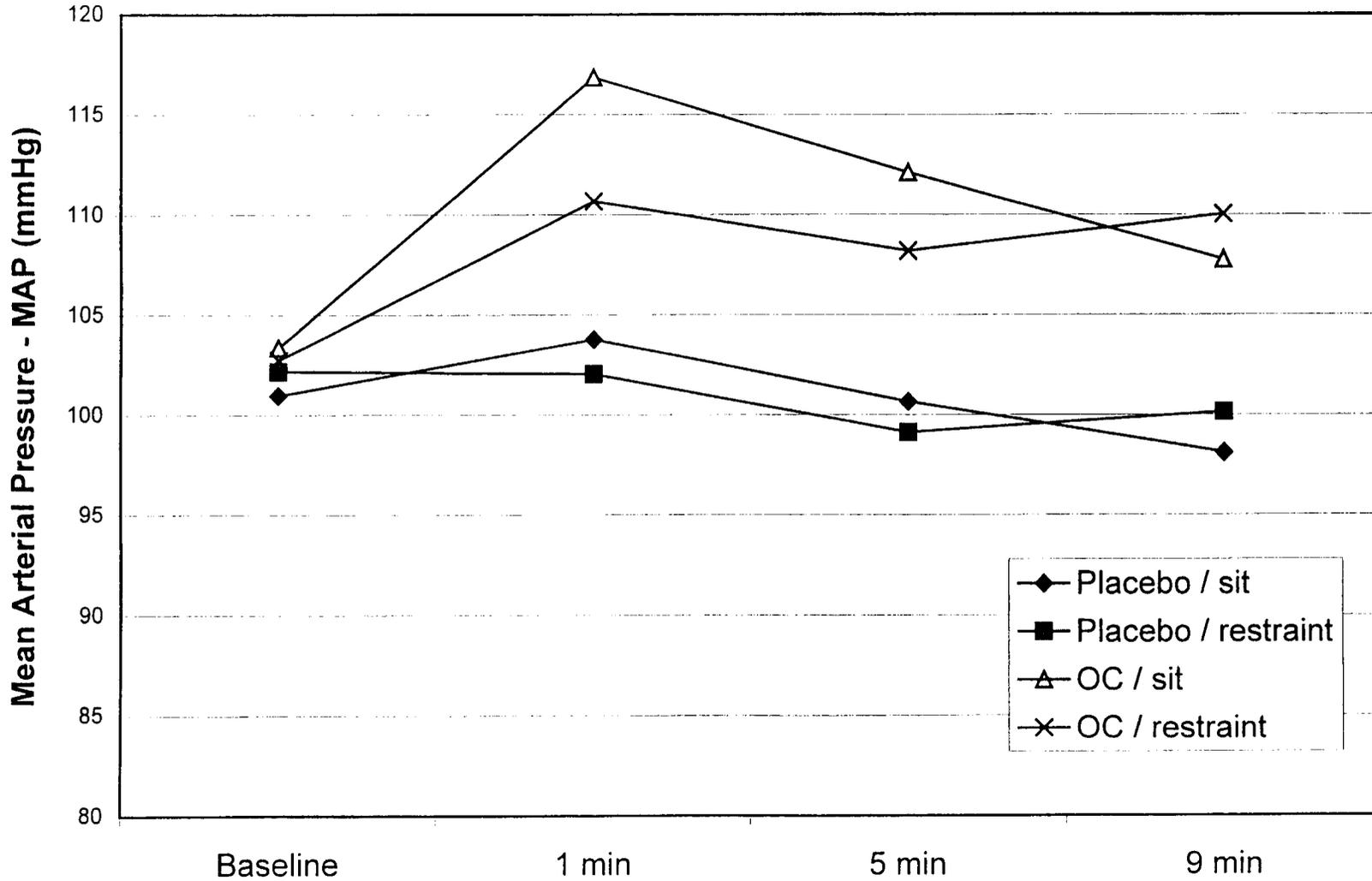


Exhibit H: Blood Pressure by Exposure and Position



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Exhibit F: Ventilation (CO2 levels) by Exposure and Position

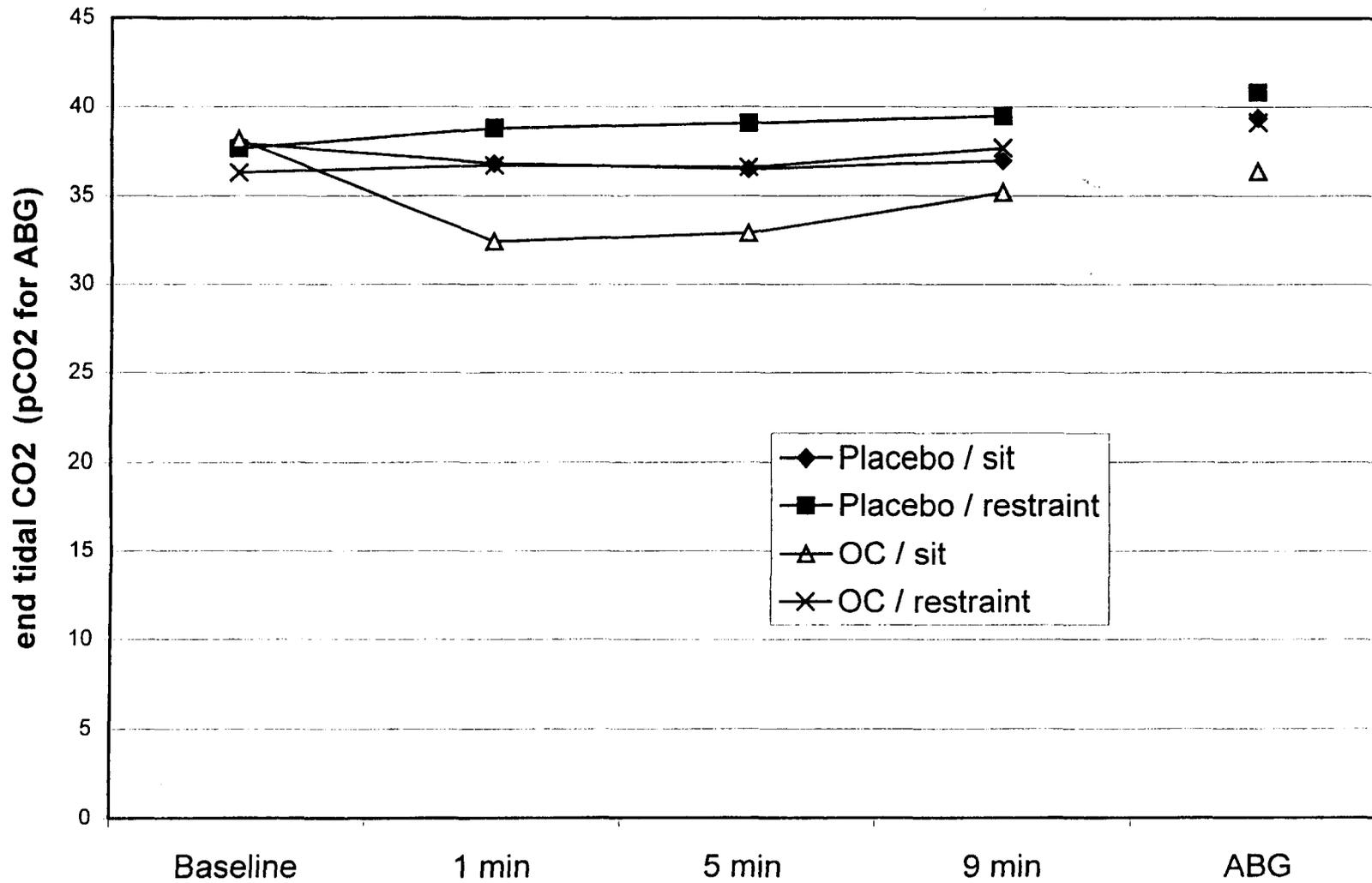


Exhibit G: Heart Rate by Exposure and Position

