
Original Contributions

THE EFFECTS OF POSITIONAL RESTRAINT ON HEART RATE AND OXYGEN SATURATION

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□ Abstract—To study the effects of positional restraint on heart rate and oxygen saturation during recovery, a cross-over experimental design with prospective control trials was used in a two-phase study. In phase 1, serial resting oxygen saturations (RO1) measured on 18 volunteers were compared with those measured in the seated unrestrained (SU1) and the hogtie (HT) positions after submaximal cycle exercise. Additionally, serial heart rates were compared postexercise on subjects in the SU1 and HT positions. In phase 2, resting oxygen saturations (RO2) were compared with those measured in the seated unrestrained (SU2) position and an alternate maximal restraint (MR) position after a simulated pursuit and struggle scenario. No statistical differences were found between SU1 and HT recovery heart rates in phase 1. Small oxygen saturation differences (1%) were found in both phase 1 and phase 2 between resting (RO1 and RO2) positions and those measured in the seated unrestrained (SU1), hogtie (HT), and maximal restraint (MR) positions. Oxygen saturations taken during a 5-min period in each of the three situations in phase 1 (RO1, SU1, and HT) were averaged, yielding 97.8, 98.0, and 97.6%, respectively. In phase 2, mean oxygen saturations were 98.0, 97.4, and 96.8.0% for RO2, SU2, and MR, respectively. In our study population, the use of hogtie and an alternate maximal restraint method did not result in any clinical restrictions in heart rate or oxygen saturation recoveries. © 1999 Elsevier Science Inc.

□ Keywords—hogtie; in-custody death; positional asphyxia

INTRODUCTION

The sudden death of people in police custody is uncommon but appears to be increasing in conjunction with a rise in cocaine use in the general population (1,2). A number of sudden deaths have occurred when law enforcement and health care personnel have placed people who were behaving in an aggressive and violent manner in restraints (3-5). Thus, it is important that law enforcement and emergency care providers are aware of any risks related to using restraints to control excited or violent persons. Early methods of police restraint (i.e., hogtying) consisted of binding a person's ankles and wrists together behind his or her back while the person was in a prone position. This was achieved by using a combination of handcuffs, cords, chains, and hobble devices.

Recently, many in-custody fatalities have been attributed to "positional asphyxia," based on a belief that the hogtie position severely restricted respiration, leading to death (6). Ross found hogtying to be a precipitating factor in 22 sudden deaths reported in the United States from 1988 to 1993 (5). However, positional asphyxia has historically been used only to characterize incidents in which body position (e.g., neck hyperflexion) caused airway obstruction, suffocation, and death (7).

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The classification of hogtie-related deaths as positional asphyxia has arisen chiefly from the work of Reay et al., who suggested that prone positioning reduces breathing effectiveness, leading to hypoventilation, hypoxia, and eventually death (3). Reay and associates adopted this model based on their findings, demonstrating slowing of heart rate and oxygen saturation recovery after submaximal exercise as measured with co-oximetry (6). Reay's study is the sole investigation to suggest that prone restraint may lead to pulmonary distress (6). Curiously, Reay et al. detected oxygenation decreases in subjects who participated in moderate aerobic exercise (6). This is in contrast to an established concept in exercise physiology which holds that oxygenation increases as a result of increases in vascularization (8).

Several investigators have applied Reay's theory indicating positional asphyxia as the cause of sudden death occurring in conjunction with prone restraint (5–10). Numerous agencies have adopted expensive restraint devices, while others have modified the hogtie technique to reduce related risks as theorized by experts (9,10). Further, many law enforcement agencies have endured costly lawsuits or prohibited the hogtie method of restraint (9).

The theory that hogtie restraint induces positional asphyxia has been contradicted by the work of Chan et al., who found that prone restraint does not clinically restrict ventilation and oxygenation (11). Chan and associates assessed the impact of prone restraint by measuring pulmonary functions through standard spirometry, arterial blood gas-derived oxygen saturations, and pulse rate via electrocardiogram (EKG) tracing. Additionally, Chan et al. found favorable comparisons between earlobe, finger probe, and arterially measured blood gases (11). Reay et al. had used an earlobe-type pulse oximeter to monitor oxygen saturations and heart rates (6).

We endeavored to further examine the effects of positional restraint. A two-phase study was conducted to 1) attempt to replicate the work of Reay and associates (6), and 2) study the effects of struggle followed by restraint on oxygen saturation. In phase 2, an alternate restraint technique was tested.

MATERIALS AND METHODS

Eighteen healthy students from a regional public safety training institute (RPSTI) volunteered to participate in the study. The design for the experiment was approved by the RPSTI's human subjects committee and dean of public safety. The student volunteers were 12 males and 6 females. All were nonsmokers. None of the volunteers was taking any medications that regulate heart rate or metabolism. Ages ranged from 21 to 42 years, and body

Table 1. Participant Demographics

Subject Number	Age	Sex	Wt	Ht	1.5 M	$\dot{V}O_2$	% Fat
1	27	M	214	72	12:18	42.1	28.1
2	35	M	195	71	14:13	32.5	22.6
3	27	F	133	69	10:07	48.3	17.4
4	26	M	148	69	12:39	41.1	14.8
5	36	F	135	64	17:28	30.0	20.2
6	39	M	145	69	13:00	34.3	13.8
7	37	F	121	62	11:20	45.0	15.5
8	23	M	223	73	14:03	36.1	21.5
9	33	F	130	66	12:47	41.1	16.7
10	30	M	230	74	12:45	34.7	22.8
11	21	F	130	67	13:46	36.6	19.5
12	24	M	170	70	10:57	45.5	17.4
13	42	M	191	70	11:30	44.0	25.5
14	22	F	111	62	11:08	45.6	16.0
15	25	M	154	70	12:01	42.1	12.5
16	40	M	195	70	12:31	40.6	21.6
17	21	M	150	65	12:19	42.6	12.5
18	30	F	145	68	14:49	35.0	20.0

Ht = height (inches), Wt = weight (lbs), 1.5 M = 1.5-mile time, $\dot{V}O_2$ = volume of oxygen consumed per minute, % Fat = body fat estimate.

weights ranged from 50.5 to 104.5 kg (111 to 230 pounds). Body fat was estimated using a Lange caliper with the Jackson and Pollock three-site equation (12). Percent body fat estimates ranged from 12.5 to 28.1 percent. The subjects' aerobic fitness ranged from low to moderate, 30.0 to 48.3 mL \cdot min⁻¹ \cdot kg⁻¹ (Table 1), based on 1.5-mile running times that were converted using the American College of Sports Medicine metabolic equations (13). Resting heart rates and resting oxygen saturations (RO1 and RO2) were measured using portable electromagnetic heart-rate monitors (Polar Vantage XL) and a finger-probe oxygen saturation analyzer (BCI model 3301). The heart rate monitors were calibrated through auscultation. Calibration of the finger-probe oximeter was accomplished by means of hyper-ventilation in the seated resting position.

Phase 1

In phase 1, students were randomly assigned to seated unrestrained (SU1) or hogtie (HT) groups for their initial test, and switched protocols after a 15-min rest. Each student then exercised on a cycle ergometer until a steady-state heart rate of more than 120 beats/min (range 124–150) was achieved (see Table 2). Students from the SU1 group were then positioned seated and unrestrained, while those from the HT group were hogtied immediately postexercise. Heart rates and oxygen saturations were measured every 15 s during a 5-min recovery period. Exercise heart rates were similar during both recovery protocols (± 6 beats/min).

Table 2. Comparison of Mean Seated Unrestricted (SU1) and Mean Hogtie (HT) Recovery Heart Rates. Significance at $p < 0.05$ ($n = 18$)

Recovery minute	Seated/ Unrestrained (SU1)	Standard Deviation	Hog tied (HT)	Standard Deviation	<i>t</i> value	<i>p</i> value
1	97.0	12.59	101.3	14.10	1.36	0.19*
2	87.0	13.86	91.4	15.47	1.78	0.09*
3	85.7	13.21	89.3	14.16	1.80	0.09*
4	86.1	12.58	88.8	13.71	1.98	0.06*
5	85.8	12.79	87.5	13.04	1.06	0.30*

* Not significant at $p < 0.05$.

Phase 2

In phase 2, students were paired with others who were within five pounds of their weight. Each pair randomly selected one student to act as either a suspect or peace officer during a simulated pursuit. These were the same volunteers from phase 1, but subjects 17 and 18 were eliminated because of mild illness (Table 1). All role players' resting oxygen saturations (RO2) were measured for 5 minutes before they participated in the scenario. Each pair of participants ran all-out for 75 meters (82 yards) to the base of a stairway, continued up and then down a 65-step, 50-meter stairway (55 yards), and then returned over the initial 75-meter course. The total distance traveled by both participants was 250 meters (274 yards). Next, the participants wrestled continuously for 1 minute. Exercise intensity was high, with heart rates ranging from 175 to 212 beats/min. At the end of 1 minute, the "peace officer" volunteer was placed in the seated unrestrained (SU2) position while the "suspect" was placed in maximal restraints (MR) and then rolled onto a side (see Figures 1 and 2). The "suspect" role-player struggled against the restraints for 30 s after being

restrained. Oxygen saturations of both role-players were measured immediately at 15 s intervals for a 5-min recovery period. Recovery heart rates were monitored but were not used for comparison purposes because exercise intensity was not controlled. After a rest period (30 min minimum), the participants changed roles and recovery oxygen saturation measurements were repeated on all 16 subjects.

The maximal restraint technique (MR) consisted of placing the suspect in the prone position, handcuffing the suspect behind the back, cord-cuffing the ankles, cord-cuffing the waist, connecting the ankle and waist cords in the front of the suspect, and rolling the subject onto a side (Figures 1 and 2). This technique differs from early hogtying because the foot-restraints are not connected to the wrist-restraints, but to a cord wrapped around the waist. Therefore, when a restrained person kicks the legs, there is no pulling on the wrists or subsequent bowing of the chest as found in the hogtie method. This technique was developed by a local police department in 1992 and is part of the basic training at the RPSTI (14).



Figure 1. Subject is initially placed in the prone position while handcuffs and leg restraints are applied.



Figure 2. Subject is rolled on one side, and a cord is wrapped around the waist and connected through the legs to the foot restraint.

Table 3. Phase 1 Comparisons of Oxygen Saturation Means for Minutes 1 Through 5 Resting (RO1) to Means for Minutes 1 Through 5 of Seated Unrestrained (SU1) and Hogtie (HT) Recovery ($n = 18$)

Minute	Rest	Standard Deviation	Seated/ Unrestrained	Standard Deviation	Prone/ Hog tied	Standard Deviation	F	p
1	97.8	1.00	98.0	0.87	97.3*	0.94	3.52	.041
2	97.8	0.86	98.0	0.62	97.6	0.63	5.39	0.09
3	97.7	1.03	98.0	0.77	97.8	0.67	1.66	0.21
4	97.8	0.86	98.0	0.77	97.7	0.76	2.33	0.11
5	97.7	0.93	98.0*	0.79	97.6	0.76	4.21	0.02

Means for five minutes of recovery 97.7, 98.0, and 97.6 for RO1, SU1, and HT, respectively.

* denotes significant difference at $p < 0.05$.

Statistical Analysis

Phase 1 recorded postexercise heart rates taken during each minute while the subjects recovered in the seated unrestrained (SU1) position were averaged and compared on a minute-by-minute basis with those measured during recovery in the hogtie (HT) position via an ANOVA for repeated measures (15). This statistical method also was used to compare resting oxygen saturations (RO1) with postexercise recovery saturations for the seated unrestrained (SU1) and hogtie (HT) positions (15). Phase 2 recovery oxygen saturations, SU2 and MR, also were compared with resting saturations (RO2) using an ANOVA (15). A probability of 0.05 was used to determine if any differences were statistically significant. In the event of a significant F ratio, a Newman-Keuls multiple factorial comparison was used to determine which differences were significant (15).

RESULTS

Phase 1

Postexercise mean heart rates for subjects recovering in either SU1 or HT positions were not significantly different for minutes 1 through 5 when compared on a minute-by-minute basis (Table 2). The 1- through 5-min mean oxygen saturation levels determined from measurements taken on all subjects for RO1, SU1, and HT positions were 97.8, 98.0, and 97.6 percent, respectively, ranging from 97.3 to 98.1. Resting (RO1) and seated unrestrained (SU1) oxygen saturation means were similar during the first minute of recovery, but both differed significantly ($p < 0.05$) from hogtying (HT). No significant differences were found between the means of any conditions measured during minutes 2, 3, or 4. Resting (RO1) and hogtie restraint (HT) oxygen saturation measurements were similar during minute 5 of recovery but differed statistically from seated unrestrained (SU1) means (Table 3).

Results: Phase 2

The 5-minute oxygen saturation means were 98.0, 97.3, and 96.8 percent, for RO2, SU2, and MR, respectively. Oxygen saturations measured during 5 minutes of rest and 5 minutes of recovery in the seated unrestrained and maximal restraint positions after participation in a simulated arrest scenario ranged from 97.1 to 98.2 (Table 4). Oxygen saturations measured during minutes 1 and 2 differed statistically between MR and both RO2 and SU2, while during minutes 3, 4, and 5, both SU2 and MR differed from RO2 ($p < 0.05$; Table 4).

DISCUSSION

The results from phase 1 of our study revealed no significant differences in heart-rate recoveries between subjects who were seated unrestrained (SU1) and those hogtied (HT) while placed prone. Statistical differences were found between oxygen saturations in both phase 1 and phase 2, but this may be attributed to a very small standard error of measurement. Measured oxygen saturations ranged from 95–99%, with similar means (SU1, 97.9; HT, 97.6%; MR, 96.8%). Therefore, from our study, it appears that healthy individuals are at little risk of suffering life-threatening desaturation when placed in either the hogtie (HT) or maximal restraint (MR) position.

Our results are in contrast to those of Reay and associates (6) but are supported by the work of Chan et al. (11), who found little differences in either heart rate or oxygen saturation during postexercise recoveries in the hogtie position. The prolonged heart-rate recovery times reported by Reay et al. may be due to instrument-related measurement error or inadequate control of exercising heart rates. For example, if the heart rates attained by subjects while exercising in Reay's study were inconsistent between trials, recovery heart rates could vary greatly.

Reay found reductions in oxygen saturation levels to

Table 4. Phase 2 Comparisons of Resting Oxygen Saturation (RO2) Means and Seated Unrestrained (SU2) and Maximal Restrained (MR) Oxygen Saturation Recoveries ($n = 16$)

Minute	Rest	Standard Deviation	Seated/Unrestrained	Standard Deviation	Maximal Restraint	Standard Deviation	F	p
1	97.9	1.10	97.6	0.92	96.7*	0.87	8.85	.001
2	98.0	1.02	97.6	0.85	97.0*	0.84	7.53	.002
3	98.0*	0.98	97.4	0.84	97.0	0.71	8.22	.001
4	98.0*	1.04	97.2	0.92	96.7	0.77	11.6	.001
5	97.9*	0.96	97.0	0.85	96.6	0.73	17.7	.001

Means for 5 minutes of recovery 98.0, 97.4, and 96.8 for RO2, SU2, and HT, respectively.

* denotes significant differences at $p < 0.05$.

85–90% after submaximal exercise. Conversely, Rowell (16) stated that “it is commonly assumed that oxygen saturation levels remain constant up to $\dot{V}O_2$ max” (6). Rowell found that only a few elite athletes experience minor oxygen saturation decreases (from 96 to 93%) at maximal exercise. This is due to a rightward shift of the oxygen association curve (Bohr effect) that has been attributed to a reduction in arterial pH, coupled with a rise in temperature, which then decreases saturation at a given PO_2 (16). Reay’s findings of desaturation after low-intensity exercise are atypical and may be due to measurement error inherent to earlobe pulse oximetry, which has been shown to be potentially unreliable for use during clinical exercise testing in previous studies (17,18). In an extensive review of recent studies comparing ear and finger-probe pulse oximeters with arterial oxyhemoglobin, Mengelkock et al. concluded that pulse oximetry has variable precision (19). They found that the accuracy of pulse oximetry declines with subject hypoxia and smoking. Additionally, they found that recent finger-probe oximeters may be more accurate than the ear-probe models. Our use of a finger-probe oximeter may explain why our results were similar to those of Chan et al. and different from those of Reay et al.

Further, Reay et al. based their theory on hogtie-related positional asphyxia, postulating that the prone position decompensated normal pulmonary ventilation, but they did not measure pulmonary functions in their studies. Conversely, Chan et al. found only minimal and nonclinically relevant reductions in pulmonary functions measured on subjects while hogtied.

The pursuit scenario included in phase 2 of our research is an attempt to study the effects of a high-intensity chase with struggle on restraint. Further, in an attempt to duplicate conditions cited by Welti (20), we had our subjects struggle for 30 s against the restraints. We theorized that isometric struggle while in restraint may decompensate the cardiorespiratory system, where venous return would be reduced because of lack of dynamic muscle action. The combination of drugs or alcohol, adrenalin, intense muscular contraction, and el-

evated body temperature may synergistically induce cardiorespiratory distress regardless of positioning or restraint.

A shortcoming of this study is the use of finger-probe pulse oximetry to estimate oxygen saturation levels instead of arterial hemoglobin. However, several investigators have demonstrated that the newer versions of finger-probe oximeters are highly accurate in recent exercise studies (21–23). Further, we employed portable heart-rate monitors to determine our subjects’ pulse rates. These devices have been shown to accurately measure normal pulse rate, but they will not precisely quantify abnormal cardiac rhythms (24). Additionally, our study cohort was small ($N = 18$) and included only healthy adult volunteers who were tested for brief periods of time (5 min). The physiological response of people with cardiopulmonary disease may be quite different. This may limit the application of our findings to the general population. Finally, when police personnel or emergency care providers contact a person who must be restrained, the individual often is psychotic or under the influence of alcohol, cocaine, or other drugs. The physiological response of intense struggle and restraint coupled with any of these secondary factors may differ from the results of our research.

Our findings and that of Chan and associates refute the premise that positional restraint alone produces physiological stress that places healthy persons (disease-, drug-, and alcohol-free) at risk for sudden death. What, then, is the mechanism for sudden in-custody deaths?

The use of cocaine that precipitates excited delirium (EXD) followed by sudden death appears to be a common element reported by several investigators (1,2,4,5,7,9,10). Insight into the cause and prevention of sudden death during restraint may be found in the work of Rutenber et al. (25). They found that chronic cocaine users who die suddenly after displaying signs of EXD have much higher dopamine levels than those dying from a toxic cocaine overdose. The researchers suggest that high levels of dopamine (due to lack of normal clearance), and not any physiological deficiencies related to

restraint, may lead to EXD and finally to fatal cardiac trauma. Additionally, Rutenber suggests that black male crack cocaine users from 20–34 years old have the greatest risk of death through excited delirium.

In conclusion, law enforcement and medical personnel must continue to respond to calls for assistance when persons become aggressive and violent as part of their duty to protect the welfare of the public. Hence, it is recommended that law enforcement and medical first-responders learn to identify persons at risk for sudden

death (as described by Rutenber et al.) and ensure that they are thoroughly evaluated and treated. All persons placed in maximal restraint should be positioned on their side or seated as soon as possible to facilitate the monitoring of their alertness, respiration, and pulse. Monitoring should be vigilant and should include basic life signs and EKG and oxygen saturation, when available. High-risk persons should be medically evaluated as needed before transport. Maximal restraint devices should be removed as soon as it is possible and safe.

REFERENCES

1. Raval MP, Welti CV. Sudden death from cocaine-induced excited delirium: an analysis of 45 cases. *Am J Clin Pathol* 1995;104:329.
2. Welti CV, Fishbain DA. Cocaine-induced psychosis and sudden death in recreational cocaine users. *J Forensic Sci* 1985;30:873–80.
3. Reay DT, Flinger CL, Stilwell AD, Arnold J. Positional asphyxia during law enforcement transport. *Am J Forensic Med Pathol* 1992;13:90–7.
4. O'Halloran RL, Lewman LV. Restraint asphyxiation in excited delirium. *Am J Forensic Med Pathol* 1993;14:289–95.
5. Ross D L. An analysis of in-custody deaths and positional asphyxiation. *Police Marksman* 1996; March/April:16–8.
6. Reay DT, Howard JD, Fligner CL, Ward RJ. Effects of positional restraint on oxygen saturation and heart rate following exercise. *Am J Forensic Med Pathol* 1988;9:16–8.
7. Bell MD, Rao VJ, Welti CV, Rodriguez RN. Positional asphyxia in adults: a series of 30 cases from the Dade and Broward County, Florida, medical examiner offices from 1982 to 1990. *Am J Forensic Med Pathol* 1992;13:101–7.
8. Lait M. *Los Angeles Times*. May 23, 1997; B1.
9. Krosch C, Binkered V, Blackbourne B. Final report of Custody Death Task Force. San Diego Police Department, 1992.
10. Ross D. Medical risk factors of sudden in-custody deaths. *Police Marksman* 1997;Nov/Dec:42–7.
11. Chan TC, Vilke GM, Neuman T, Clausen JL. Restraint position and positional asphyxia. *Ann Emerg Med* 1997;30:578–86.
12. Jackson AS, Pollock M. Generalized equations for predicting body density of man. *Br J Nutr* 1978;:497–504.
13. American College of Sports Medicine. Guidelines for exercise testing and prescription, 4th edn. Philadelphia: Lea and Febiger; 1991:285–300.
14. San Diego Police Department. Policy and Procedure Manual. City of San Diego. 1992; DP - 6.01.
15. Elliot AC. Kwikstat, Winks Professional Windows version: 1st edn. TexaSoft: Cedar Hill; 1996.
16. Rowell L. In: Human circulation regulation during physical stress. New York: Oxford University Press; 1986:227–8.
17. Norton LH, Squires B, Craig NP, et al. Accuracy of pulse oximetry during exercise stress testing. *Int J Sports Med* 1992;13:523–7.
18. Hansen JE, Casaburi R. Validity of ear oximetry in clinical exercise testing. *Chest* 1987;91:333–7.
19. Mengelkoch LJ, Martin D, Lawler J. A review of the principles of pulse oximetry and accuracy of pulse oximeter estimates during exercise. *Phys Ther* 1994;74:40–9.
20. Welti CV. Fatal cocaine intoxication: a review. *Am J Forensic Med Pathol* 1987;81–2.
21. Barthelemy JC, Geysant A, Riffat J, et al. Accuracy of pulse oximetry during moderate exercise: a comparative study. *Scand J Clin Invest* 1990;50:533–9.
22. Martin D, Powers S, Circale M, et al. Validity of pulse oximetry during exercise in elite endurance athletes. *J Appl Physiol* 1992; 72:455–8.
23. Mengelkoch LJ, Martin D, Cicale M, Huang D. Validity of pulse oximetry during maximal treadmill exercise Abstract. *Med Sci Sports Exerc* 1992;24(Suppl):159.
24. Godsen R, Carroll T, Stone S. How well does the Polar Vantage XL heart rate monitor estimate actual heart rate? *Med Sci Sports Exerc* 1991;23(Suppl):14.
25. Rutenber AJ, Lawler-Heavner J, Yin M, Welti CV, Hearn WL, Mash C. Fatal excited delirium following cocaine use: epidemiological findings provide new evidence for mechanisms of cocaine toxicity. *J Forensic Sci* 1997;42:25–31.