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Effect of position and weight force on inferior vena cava diameter – Implications for arrest-related death

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ABSTRACT

Introduction: The physiology of many sudden, unexpected arrest-related deaths (ARDs) proximate to restraint has not been elucidated. A sudden decrease in central venous return during restraint procedures could be physiologically detrimental. The impact of body position and applied weight force on central venous return has not been previously studied. In this study, we use ultrasound to measure the size of the inferior vena cava (IVC) as a surrogate of central venous return in the standing position, prone position, and with weight force applied to the thorax in the prone position.

Methods: This was a prospective, observational study of volunteer human subjects. The IVC was visualized from the abdomen in both the longitudinal and transverse section in the standing and prone positions without weight force applied, and with 100 lbs (45 kg) and 147 lbs (67 kg) of weight force on the upper back in the prone position. Maximum and minimum measurements were determined in each section to account for possible respiratory variation of the IVC.

Results: The IVC significantly decreased in size with each successive change: from standing to prone, from prone to prone with 100 lbs (45 kg) weight compression, from prone with 100 lbs (45 kg) weight compression to prone with 147 lbs (67 kg) weight compression ($p < 0.0001$). The vital sign measurements had no statistical change.

Conclusions: The physiology involved in many sudden, unexpected ARDs has not been elucidated. However, in our study, we found a significant decrease in IVC diameter with weight force compression to the upper thorax when the subject was in the prone position. This may have implications for the tactics of restraint to aid in the prevention of sudden, unexpected ARD cases.

1. Introduction

The physiology of many sudden, unexpected arrest-related deaths (ARDs) proximate to restraint has not been elucidated. Previous work by Chan et al. did not suggest a relationship between position, restraint, and weight force (up to 102 kg) with a clinically important impact on respiration [1–3]. The impact of these variables on central venous return has not been studied. It is possible that a sudden decrease in central venous return with either body position or applied weight force in custodial arrest settings could be physiologically detrimental, especially in a compromised individual. In this study, we use ultrasound to measure the size of the inferior vena cava (IVC) as a surrogate of central venous return in the standing position, prone position, and with weight force applied to the thorax in the prone position.

2. Methods

This was a prospective, observational study of volunteer human subjects. The institutional review board at the Hennepin County Medical Center/Minneapolis Medical Research Foundation (Minneapolis, MN) approved the study. The subjects were a convenience sample of law enforcement officers and civilians. After providing informed consent, the study physician screened for exclusion criteria. The exclusion criteria included: known pregnancy, a musculo-skeletal condition that would preclude weight applied to the thorax, or a body mass index (BMI) calculation over 30 kg/m^2. The BMI exclusion criterion was chosen to ensure adequate visualization of the IVC with ultrasonography. Subjects also provided health histories for demographic data collection.

A commercial skin resistance analyzer (Omron Fat Loss Monitor HBF-306, Omron Healthcare, Inc., Bannockburn, IL) was used to determine body fat percentage. All subjects were in a rested condition at the time of the experiment. Subjects had an initial IVC ultrasound performed by a professional ultrasonographer with certification as a Registered Diagnostic Medical Sonographer (RDMS) using a Sonosite M-Turbo ultrasound machine (Sonosite Inc., Bothell, WA). Ultrasonographic images of the IVC

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Fig. 1. Experimental setup.

were obtained in B-mode with a phased array (5–2 MHz) transducer in the standing position. The IVC was visualized from the abdomen in a substernal location in both the longitudinal and transverse section. Maximum and minimum measurements were determined in each section to account for possible respiratory variation of the IVC. The ultrasonographer determined the recorded values, and rechecked them at her discretion. Vital signs (blood pressure and heart rate) were taken with an automated monitor (Nonin 2120, Plymouth, MN). Subjects were then laid prone on a flat, level table that had a 10 cm opening at the upper abdomen/lower chest to facilitate ultrasonography on the prone subject. The IVC measurements and vital signs were repeated in this position after approximately 1 min. Subjects then had 100 lbs (45 kg) of weight lowered onto their upper back and the IVC measurements and vital signs were repeated, approximately 1 min after placement of the weight. This delay was intentional to assess subject tolerance before measurements. Finally, the subjects had the weight increased to 147 lbs (67 kg) and the IVC measurements and vital signs were repeated, again, approximately 1 min after the placement of the weight. The 147 lbs of final weight was unintentional. The study authors intended to have the weight be 150 lbs (68 kg), but when the final constructed weight apparatus was weighed, it was 147 lbs (Fig. 1).

Data were entered into an Excel spreadsheet (Microsoft Corp., Redmond, WA) and exported into STATA 10.0 (STATA Corp., College Station, TX). Data were analyzed using descriptive statistics and k sample for equality of means test.

3. Results

A total of 25 subjects were enrolled in the study. One subject voluntarily withdrew from the study once the 100 lbs was placed due to discomfort. Of the 24 subjects that completed the study, 22 were male, and 2 were female. There was a failure to collect the demographic data on one of the subjects so their results were excluded from analysis. Therefore, a total of 23 subjects were included in the analysis. The median subject age was 35 years old (IQR, 26–43). The median subject height was 71 in. (IQR, 69–72) and the median subject weight was 185 lbs (IQR, 175–200). The median subject BMI was 26.6 kg/m^2 (IQR, 25–27.7). The subject health histories included: Achilles tendon surgery (1), cholecystectomy (1), shoulder surgery (2), nephrolithiasis (1), pilonidal cyst (1), appendectomy (2), knee surgery (1), orbital fracture (1), MRSA joint infection (1), traumatic brain injury (1), hip dislocation (1), foot surgery (1), hypertension (1), hypercholesterolemia (1).

The results are presented in Tables 1–2. The IVC decreased in size with each successive change: from standing to prone, from prone to prone with 100 lbs weight compression, from prone with 100 lbs weight compression to prone with 147 lbs weight compression ($p < 0.0001$). The vital sign measurements had no statistical change.

4. Discussion

The mechanisms involved in many sudden, unexpected ARDs are poorly understood. The infrequency, widespread geographical occurrence of the phenomenon, variable medical laboratory testing and treatment for cardiac arrest in these cases, and the varying data available at autopsy, as well as legal barriers to investigations, impairs the study of this phenomenon. Some associated risk factors for ARD have been identified through retrospective studies. Hick et al. identified a profound metabolic acidosis from forceful struggle, restraint preventing compensatory respiratory mechanisms, and stimulant drug use as risk factors [4].

Table 1
IVC measurements in centimeter.

	Standing	Prone	45 kg	67 kg
Longitudinal maximum, cm, median (IQR)	1.86 (1.57–2.16)	1.67 (1.05–2.26)	1.205 (0.83–1.58)	0.805 (0.46–1.29)
Longitudinal minimum, cm, median (IQR)	1.21 (1.01–1.51)	1.14 (0.64–1.61)	0.70 (0.45–1.02)	0.28 (0.0–0.79)
Transverse maximum, cm, median (IQR)	1.63 (1.43–1.93)	1.45 (1.17–2.02)	1.12 (0.76–1.65)	0.74 (0.46–1.13)
Transverse minimum, cm, median (IQR)	1.18 (0.93–1.39)	1.01 (0.77–1.47)	0.38 (0.0–1.15)	0.31 (0.0–0.52)

Table 2
Vital sign measurements.

	Standing	Prone	45 kg.	67 kg.
Heart rate, BPM (IQR)	74 (67–89)	72 (66–79)	70 (68–80)	80 (74–84)
Systolic pressure, mm Hg (IQR)	130 (124–142)	131 (126–147)	138 (129–151)	141 (128–151)
Diastolic pressure, mm Hg (IQR)	84 (80–88)	83 (70–89)	85 (80–90)	89 (80–95)

Stratton et al. identified forceful struggle, stimulant drug use, established natural disease, and obesity [5]. Ho et al. identified drug abuse and "bizarre" or "out-of-control" behavior [6]. Finally, O'Halloran and Frank identified acute delirium, significant exertion, stimulant drugs, and being restrained in the prone position [7]. In the study by O'Halloran and Frank, 17 of 21 subjects had body weight from the arresting officer(s) applied to the upper torso at the time they lost consciousness. Previous work has not suggested a relationship between weight force and respiratory impairment [1–3]. Our work examined the relationship between weight force and IVC collapse.

In our study, we found a significant decrease in the IVC dimensions with position change and applied thoracic weight force. ARDs often occur in subjects under significant physiologic stress. A study by Ho et al. of simulated arrest encounters demonstrated that the behaviors seen in a resistive subject, such as fighting and fleeing, are physiologically stressful and can lead to a profound acidosis, a catecholamine surge, and significant vital sign changes [8]. In this study, the mean change in heart rate after the simulated fight was 72 beats per minute (range of heart rate was 115–181). It is, at least theoretically, a concern that a subject who has a very elevated heart rate from exertion, mental excitement, heat stress, sympathomimetic abuse, or a combination of these, and who has a sudden decrease in central venous return could have decreased cardiac filling, decreased cardiac output, decreased coronary perfusion pressure, and cardiac ischemia that could lead to a arrhythmic cardiac arrest. In the O'Halloran and Frank series, it was estimated that the time the subject was held in a prone position was 2–12 min [7]. This is enough time to lead to cardiac ischemia. Our subjects did not have a significant drop in systemic blood pressure, but our subjects had normal blood pressures at the start (and presumed normal hearts), were not exerting themselves (sudden cessation of muscle activity can lead to a sudden drop in preload as well), and did not have impaired autonomic control as is typically the case in the compromised subjects in field situations. Additionally, these changes may be more likely to occur in the obese subject in the prone position since obesity can lead to increased abdominal pressures and increased risk of collapse of the IVC [9]. A high BMI has been reported in studies of arrest related death [5,7]. Our subjects had a low mean BMI intentionally in order to ensure adequate visualization of the IVC.

Some authors have reported a high incidence of hyperthermia in cases of sudden, unexpected ARD. In their landmark paper, Wetli and Fishbain reported on a series of seven "cocaine excited delirium subjects" who died in custody. In their series, 4 were hyperthermic, two did not have a temperature noted, and one was not hyperthermic [10]. Wetli et al. reported an average temperature of 105 °F in their Miami cohort [11]. Hyperthermia can lead to an increased cardiac output and a relative hypovolemia that may make the subject more susceptible to decreases in central venous return [12,13]. In addition, subjects may be relatively hypovolemic from decreased oral intake due to drug binging. Some authors have reported a higher incidence of these deaths in warmer climates [11]. Hot ambient temperatures, especially in combination with exertion, may also predispose subjects to being relatively hypovolemic due to dehydration. Hick et al. hypothesized that the sudden collapse with restraint could be related to the loss of vascular tone with catecholamine depletion from the struggle [4]. If there is a loss of vascular tone from catecholamine depletion, decreasing central venous return would exacerbate this. So, there are several factors in ARDs that could possibly exacerbate a reduction in central venous return from thoracic compression: obesity, hyperthermia, and catecholamine depletion.

In addition to a reduction in cardiac output and subsequent ischemia, the decreased filling could also, theoretically, invoke maladaptive neurocardiac reflexes that could lead to a brady-asystolic arrest. The negative cardiovascular effects of IVC compression have been well documented in gravid patients. Huang et al. reported on a gravid patient who had 9 s of sinus arrest leading to hypotension and syncope caused by a 54% decrease in the diameter of the IVC with position change from supine to sitting [14]. In this paper, it was hypothesized that the IVC occlusion leads to decreased cardiac filling that may activate pressure-sensitive afferent nerves in the heart. These afferent nerve signals presumably inhibit sympathetic output and stimulate vagal efferent nerves leading to brady arrhythmias and hypotension. While such effects may be generally benign in healthy, young persons, this may not be the case in the acidotic, hyperthermic, drug-intoxicated and catecholamine surge/depleted subject in arrest-related situations. Sprung et al. reported on a 54-year-old male who had "cardiac arrest due to a vasovagal syncope" during the insertion of an epidural catheter. The patient had 42 s of asystole and was resuscitated with atropine and epinephrine [15]. An inappropriate neurocardiogenic reflex as described above can cause cardiac arrest. At least three such reflexes have been proposed including: (1) collapse firing of low pressure baroreceptors located in the right atrium, (2) a reflex involving pacemaker cells whereby heart rate is proportional to stretch, and (3) the paradoxical Bezold–Jarisch reflex in which mechanoreceptors in the left ventricle cause bradycardia. In each mechanism, the end pathway is increased vagal tone [16].

It has also been hypothesized that a stress cardiomyopathy may be contributory in some of these unexplained, ARDs [13,17]. Wittstein et al. studied 19 previously healthy patients with "myocardial stunning" after sudden emotional stress. These patients presented with chest pain, pulmonary edema, and shock. The median ejection fraction was 0.20. In 17 of the 19, there was a mild elevation in troponin. Wittstein et al. proposed several mechanisms for the association of sympathetic stimulation and myocardial stunning: (1) epicardial coronary artery spasm, (2) microvascular spasm, and (3) direct myocyte injury [18]. Abraham et al. studied 9 cases of acute stress cardiomyopathy precipitated by iatrogenic infusion of epinephrine and dobutamine. Six subjects developed symptomatic heart failure with a mean Killip class of 3. Subjects had a mean troponin of 4.07, and a mean ejection fraction of 35%. The authors concluded that excess sympathetic stimulation was central to the pathogenesis of acute stress cardiomyopathy [19]. The work by Ho et al. demonstrated that there is excessive sympathetic stimulation with common subject behaviors during resistive custodial arrests simulations [8]. With a sudden decrease in central venous return, such subjects might not be able to maintain adequate perfusion pressure. In addition, Otahbachi et al. described a left ventricular outflow tract obstruction secondary to the hypercontractile cardiac base in some of these subjects. A sudden decrease in central venous return could cause cardiovascular collapse [13]. Finally, there is also evidence implicating central neurogenic factors in stress-induced arrhythmias [20]. So, the sudden reduction in central venous return due to IVC collapse from prone positioning and applied thoracic weight force during arrest situations could cause decreased coronary artery perfusion and lead to arrhythmias in the already sensitized heart.

Prior to this study, there was no work examining the effects of restraint on central venous return. As written by Stratton et al. "The entire process of subduing and restraining an uncooperative individual may have high risk for causing ventilation compromise" [5]. The work by Chan et al. examined the ventilatory question in healthy volunteers [1–3]. The effects of the restraint processon central venous return have not been examined. While the etiology of these unexpected, arrest related deaths is likely multi-factorial, the decreased central venous return with certain methods of restraint may have a role. Our study indicates that thoracic compression does reduce the size of the IVC perhaps reducing

central venous return. More study is recommended, including studies of methods of restraint that minimize this risk.

5. Limitations

The primary limitation of our study is the use of IVC measurements as a marker of cardiac filling and, by inference, cardiac output. In addition, the small number of subjects is a limitation.

The authors limited the BMI of subjects in order to ensure adequate visualization of the IVC. We feel that this limitation likely caused underreporting of our findings. We hypothesize that obese subjects will likely have increased compression of the IVC due to a reduced abdominal compliance and increased abdominal compartment pressures, especially in the prone position. This is of particular relevance since the available demographic literature supports that ARDs occur with a high preponderance in overweight to obese individuals based on BMI.

Finally, in our study, we only examined weight compression on the upper thorax and not the lower thorax or while in the supine position. The effects of weight force to this area or in this position on IVC diameter are not known. We also did not examine different restraint methods that would be used in the field with weight compression (e.g., handcuffing and hobble) to secure a subject under custodial arrest.

6. Conclusion

The physiology involved in many sudden, unexpected ARDs has not been elucidated. We found a significant decrease in IVC diameter with applied weight force compression to the upper thorax while in the prone position. We recommend further study in this area to determine its relevance to ARD.

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