Physiologic Mechanisms Associated with the Trendelenburg Position

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Abstract

Introduction: The Trendelenburg position is a common intervention used to stabilize patients in hemodynamic shock. It has been assumed that the head-down tilt position would create a hydrostatic gradient to improve venous return and, therefore, increase the cardiac output. However, numerous studies have shown this maneuver to be ineffective for hemodynamic enhancement. This study analyzed the physiologic mechanisms responsible for the limited benefits of Trendelenburg positioning.

Methods: Two-dimensional ultrasonography (Philips HDI 5000, Bothel,WA) was applied to healthy volunteers (four male; four female) to determine the changes in stroke volume (SV), cardiac output (CO) and inferior vena cava (IVC) area upon transition from the supine position to a 6º head-down tilt.

Results: In the eight subjects studied, the IVC area increased from an average of 10.76mm to 11.43mm ($P<0.05$) after transitioning to the head-down tilt position. Measurements of SV and CO showed small but clinically insignificant increases (7% and 8% respectively).

Conclusions: The Frank-Starling mechanism of the heart provides for the translocation of excess blood and fluid from the venous to the arterial side of the circulation. In the context of a functionally insignificant increase in CO during head-down tilt, the increase in the area of the IVC seen in this study may indicate a sequestering of blood in the venous system. It is considered that the weight of the abdominal organs may produce a fulcrum-like affect on the IVC when the patient is tilted to the head-down position. This external pressure would potentially increase the resistance to venous return and, thereby, limit the impact of the increased hydrostatic gradient on blood flow.

Introduction

The Trendelenburg position, or head-down tilt, has long been used during the treatment of patients in hemodynamic shock. According to Shampo, the use of the position has been around since the time of the Roman writer, Celsus (25 BC- 20 AD), for the treatment of abdominal injuries.[1] The position did not gain popularity until 1890 when Trendelenburg published his use of the position during an abdominal approach to repair a vesicovaginal fistula.[1] Since that time, the head-down tilt has been widely used in abdominal and pelvic surgeries. It was not until World War I that Walter Cannon made popular the use of this position for shock patients.[2] Trendelenburg positioning has now found widespread use and it has been reported that up to 99% of all critical care nurses surveyed have used the maneuver at some point in time.[3]

It has been generally assumed that the placement of a hemodynamically unstable patient in a head-down position would create a hydrostatic gradient facilitating the venous return of blood to the heart. An increase in venous return would then enhance cardiac output (CO) through the Frank-Starling mechanism and improve tissue perfusion, particularly for vital organs. However, this seemingly intuitive concept has not been found to be valid in several well documented studies.

Sibbald et al explored the hemodynamic effects of the Trendelenburg position in critically ill normotensive and hypotensive patients.[4] They found that in the normotensive patient group, the head-down positioning increased the preload, slightly increased CO, decreased systemic vascular resistance (SVR) and did not change the mean arterial pressure (MAP). Applying the same maneuver to the hypotensive patients resulted in a decrease in CO while the preload remained unchanged and the afterload increased only slightly. They concluded that there were no beneficial effects of this positioning for hypotensive patients.

Ostrow et al conducted a similar study on the effects of the Trendelenburg position on hemodynamics in 23 normotensive cardiac surgery patients.[5] In this study they found no statistically significant changes in CO, MAP, SVR or tissue oxygenation. These investigators also concluded that the study did not provide support for the general use of Trendelenburg positioning as a way to influence hemodynamic parameters in these patients.

Terai et al studied the hemodynamic effects of the Trendelenburg position at one minute and at ten minutes in ten healthy volunteers.[6] The results at one minute showed a marked increase in CO (16%) as well as an increase in left-ventricular end diastolic volume (LVEDV). These changes, nonetheless, had returned to baseline by ten minutes. They found that the MAP did not change from baseline, and while the internal jugular vein (IJV) velocity decreased and the IJV cross-sectional area increased at one minute, they both returned to baseline by ten minutes. The investigators proposed that Trendelenburg positioning produces a transient autotransfusion effect on hemodynamics, which is rapidly normalized in euvolemic patients.

Gaffney et al. also studied the autotransfusion effect related to passive leg raising, a modified version of the Trendelenburg positioning.[7] In this study, the stroke volume (SV) and CO increased transiently after three minutes of leg raising. However, by seven minutes, the changes had returned to baseline. They concluded that passive leg-raising does not result in a sustained increase in CO or SV. More recently, Zorko et al. showed that Trendelenburg positioning most enhances cardiac output when intravenous fluids were given concurrently.[8]

Even though these and many other studies have shown the ineffectiveness of Trendelenburg positioning alone to improve hemodynamic performance, the physiologic rationale surrounding these findings is still poorly understood.[9,10,11,12,13] This study analyzes the possible physiologic mechanisms responsible for the observed limited benefits of this common clinical maneuver.

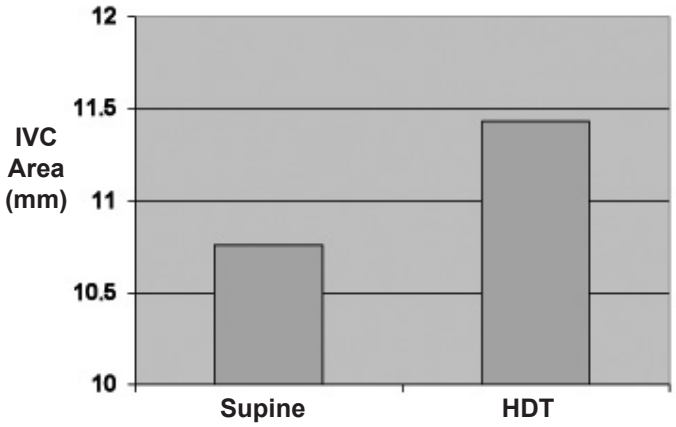
Methods

Two-dimensional ultrasonography (Philips HDI 5000, Bothel, WA) was applied to healthy volunteers to determine the changes in SV, CO and inferior vena cava (IVC) area upon transitioning from the supine attitude to a 6° head-down tilt (HDT) position. Stroke volume was determined using a standard technique in which two-dimensional echocardiography images and continuous-wave Doppler measures were used to determine aortic cross-sectional area (parasternal long-axis view at the point of cusp insertion) and flow (systolic velocity integral aortic cross-sectional area).[14] From the values of stroke volume, heart rate and mean arterial pressure, all the other hemodynamic parameters are derived. After a five-minute period of equilibration in the supine position, an image of the IVC was localized at a level just caudal to the lower liver margin. Measurements of the cross-sectional IVC area were recorded as well as SV and CO. While maintaining the ultrasound probe at the same level, the subjects were rapidly transitioned to the 6° head-down tilt position (moderate Trendelenburg). After five minutes of equilibration in this posture, the IVC area was again measured as well as the SV and CO. Changes in these measurements were analyzed using a standard student's test (significance $p < 0.05$). The study was performed under the auspices of a NASA Johnson Space Center Institutional Review Board approved experimental protocol.

Results

In the eight subjects studied (four female; four male), the IVC area increased from an average of 10.76mm to 11.43mm ($p < 0.05$) after transitioning to the head-down tilt position (Figure 1). Measurements of SV and CO showed small, but clinically insignificant increases (7% and 8% respectively, $p<0.05$).

Figure 1: Impact of Trendelenburg positioning on IVC area



Discussion

The Frank-Starling mechanism of the heart provides for the translocation of blood and fluid from the venous to the arterial side of the circulation. In a normally functioning circulatory system, any means through which there is an enhancement in venous return should result in an increased left ventricular end-diastolic volume or preload, and, therefore, augment the SV and CO. The intuitive concept behind the potential beneficial effects of the Trendelenburg maneuver is grounded in the assumption that placing the patient in the head-down position will increase venous return by a gravitationally driven force to move blood toward the heart.[1,2] It is further supposed that this movement of blood would result in a sustained increase in cardiac output in the normally functioning circulation.

The current study and several previously noted studies have not shown a significant sustained increase in cardiac output of more than 5 - 10%.[4,5,6] Through the application of gravitational hydraulics to Pascal's Principle (pressure $= \rho g h \sin\theta$) it would be expected that the force for driving venous return and cardiac output would increase by ~20% using traditional Trendelenburg tilt angles. In normotensive, euvolemic patients this differential between the expected and observed hemodynamic changes might be explained by a physiologic counter-regulatory modu-

lation of the vascular compliances and resistances in an attempt to normalize the hemodynamics after a postural perturbation. There would be no need for an increase in cardiac output or arterial pressures in these circumstances, and the circulatory control mechanisms would respond to mitigate these state changes. This explanation would be consistent with the time dependent changes in CO that have been observed with the HDT maneuver. The transient increases in CO found with a HDT are often found to recede within minutes of the change in position.[6,7]

While normal physiologic control responses may account for the lack of hemodynamic augmentation during Trendelenburg positioning in the euvolemic, normotensive patient, the same cannot be concluded for the hypotensive, shock patient.[4] In the hypotensive patient we would expect that normal circulatory physiologic control mechanisms would work to amplify rather than modulate any attempts to increase CO. However, hemodynamics in hypotensive patients do not appear to be enhanced by a Trendelenburg positioning. In some circumstances, the CO may be even reduced further by a HDT maneuver.[4] These findings suggest that another mechanism beyond the typical circulatory controls is involved in the abrogation of the hemodynamic response to the Trendelenburg positioning.

In our study, a statistically significant increase in subhepatic IVC area ($p<0.05$) was found within five minutes of placing subjects in the HDT position. It is thought that this finding is a result of the abdominal viscera acting as a fulcrum on the IVC while in the Trendelenburg position in a manner similar to that previously described by Gauer.[13] Such a compression on the IVC would result in an impedance to venous return to the heart, decreasing preload, and, thereby, restricting the impact of the increased hydrostatic gradient on CO. There are findings in the literature that support this idea.

Reuter et al. conducted a study on the hemodynamic effects of the Trendelenburg position in 12 hypovolemic patients post-cardiac surgery.[15] By measuring the intrathoracic blood volume by transpulmonary indicator dilution, they found that the HDT maneuver caused only a slight increase in preload volume and did not significantly change CO or MAP. They concluded that the Trendelenburg positioning causes a no significant increase in venous return to the heart.

While the impact of small increases in vena cava resistance on venous return and CO were first described by Guyton more than 40 years ago, these effects are not always considered in many clinical scenarios.[16,17] However, external pressures on the vena cava are known to commonly influence hemodynamics during laparascopic surgery and in the perinatal condition.[18,19,20] Placing patients in the Trendelenburg position is a very common practice in clinical medicine.[3] The utility of this maneuver is in question, though the reasons for its failures are poorly understood.[2,10] Evidence supports the idea that changes in the resistance to venous return may play a central role in the limitations.[13,14] Even in the supine position, the abdominal organs appear to have some compressive effect on the central venous system.[13] In this study, changes in the venous system caudal to the liver and diaphragm were measured. It seems likely that the positioning and relative weights of these organs make them more likely candidates for providing a fulcrum force. If the abdominal organs are limiting venous return, it may be possible to modify the maneuver to circumvent this problem. While we used a 6º head-down tilt position in this study, a different angle might result in more optimal hemodynamics. The current clinical study is limited in its scope and represents a preliminary analysis of the potential associated mechanisms involved. Further study is required for a better understanding of all the physiologic mechanisms in play during Trendelenburg positioning and to determine the best postural angle needed to optimize hemodynamics in patients with circulatory shock.

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