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The Cardiopulmonary Consequences of the Trendelenburg Position in Patients Under General Anesthesia

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Abstract

Background: Acute hypotension has been treated with the Trendelenburg, or head down tilt (HDT), position for 100 years or more. Prior studies have suggested the HDT position increases central venous blood return and improves cardiac output, but the evidence is not consistent. This systematic review is designed to identify if there are known negative consequences of the HDT position on cardiopulmonary functioning, and determine if those risks outweigh the benefits.

Methods: An extensive search on Ovid Medline, CINAHL, and Evidence-Based Medicine Review Multifile was conducted to identify pertinent articles. The inclusionary criteria were, the use of HDT of greater than, or equal 1ð, and patients under general anesthesia.

Results: Six articles were identified and critically appraised. The data compiled in this systematic review suggest there is an increase in cardiac preload with no consequent increase in cardiac output or performance. The data suggest there are multiple negative consequences of HDT on pulmonary function including a decrease of functional residual capacity, an increase of atelectasis, and a decrease in oxygenation.

Conclusion: There is a lack of clear evidence to support the use of HDT as a treatment for acute hypotension. In the controlled environment of the surgical setting, head-down tilt should be utilized judiciously and for as short a duration as possible. HDT position should be avoided in patients who are obese, have pre-existing obstructive pulmonary disorders, have New York Heart Association class III heart failure, or other significant cardiopulmonary dysfunction

NOTICE TO READERS

The Cardiopulmonary Consequences of the Trendelenburg Position in Patients Under General Anesthesia

Aaron T. Carter

Biography

[Redacted for privacy]

Abstract

Background: Acute hypotension has been treated with the Trendelenburg, or head down tilt (HDT), position for 100 years or more. Prior studies have suggested the HDT position increases central venous blood return and improves cardiac output, but the evidence is not consistent. This systematic review is designed to identify if there are known negative consequences of the HDT position on cardiopulmonary functioning, and determine if those risks outweigh the benefits.
Methods: An extensive search on Ovid Medline, CINAHL, and Evidence-Based Medicine Review Multifile was conducted to identify pertinent articles. The inclusionary criteria were, the use of HDT of greater than, or equal $10°$, and patients under general anesthesia.
Results: Six articles were identified and critically appraised. The data compiled in this systematic review suggest there is an increase in cardiac preload with no consequent increase in cardiac output or performance. The data suggest there are multiple negative consequences of HDT on pulmonary function including a decrease of functional residual capacity, an increase of atelectasis, and a decrease in oxygenation. **Conclusion:** There is a lack of clear evidence to support the use of HDT as a treatment for acute hypotension. In the controlled environment of the surgical setting, head-down tilt should be utilized judiciously and for as short a duration as possible. HDT position should be avoided in patients who are obese, have pre-existing obstructive pulmonary disorders, have New York Heart Association class III heart failure, or other significant cardiopulmonary dysfunction.
Keywords: Trendelenburg position, head-down tilt, cardiac preload, cardiac output, perfusion, and anesthesia.

Acknowledgements

[Redacted for privacy]

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- A-aO$_2$ = Alveolar-Arterial Oxygen Gradient
- BMI = Body Mass Index
- CABG = Coronary Artery Bypass Graft
- Cdyn = Dynamic Compliance
- CI = Cardiac Index
- COH = Conventional Open Hysterectomy
- CV = Cardiovascular
- CVP = Central Venous Pressure
- E$_{cw}$ = Chest Wall Elastance
- E$_L$ = Lung Elastance
- E$_{rs}$ = Total Respiratory System Elastance
- FEV$_1$ = Forced Expiratory Volume in 1 minute
- FRC = Function Residual Capacity
- HDT = Head-Down Tilt; Trendelenburg Position
- HR = Heart Rate
- HUT = Head-Up Tilt
- ITBVI = Intrathoracic Blood Volume Index
- LCI = Lung Clearance Index
- LH = Laparoscopic Hysterectomy
- LVEDAI = Left Ventricular End-Diastolic Area Index
- MAP = Mean Arterial Pressure; Mean Arterial Blood Pressure
- MPAP = Mean Pulmonary Artery Pressure
- OLV = One-Lung Ventilation
- PaO$_2$ = Arterial Oxygen Tension
- PAW = Peak Airway Pressure
- PCWP = Pulmonary Capillary Wedge Pressure; Pulmonary Artery Occlusion Pressure
- PEEP = Peak End Expiratory Pressure
- P$_{insp}$ = Peak Inspiratory Pressure
- PVRI = Pulmonary Vascular Resistance Index
- Qs/Qt = Pulmonary Shunt Fraction
- R$_{cw}$ = Chest Wall Resistance
- R$_L$ = Lung Resistance
- RR = Respiratory Rate
- R$_{rs}$ = Total Respiratory System Resistance
- SpO$_2$ = Pulse oximeter oxygen saturation
- SVR = Systemic Vascular Resistance
- SVRI = Systemic Vascular Resistance Index
- SVV = Stroke Volume Variation
- T$_0$ = Baseline supine measurements
- T$_1$ = Time at first experimental measurements
- T$_2$ = Time at second experimental measurements
- TLC = Total Lung Capacity
- T$_P$ = Time post experimental intervention
- VATS = Video-Assisted Thoracoscopic Surgery
- V$_T$ = Tidal Volume

The Cardiopulmonary Consequences of the Trendelenburg Position

in Patients Under General Anesthesia

BACKGROUND

Shock is a state of inadequate blood flow and perfusion which is typically manifested by acute hypotension, a common finding in many critically ill patients. One treatment option that has been in practice for many years is the Trendelenburg, or head-down tilt (HDT) position. It has been suggested the HDT position increases central venous blood return and improves cardiac output (CO), but the evidence is not consistent.[1-3]

Dr. Friedrich Trendelenburg, a German surgeon, was one of the first people to describe the HDT position in the surgical patient. He suggested the HDT position is beneficial for exposure during surgical procedures for multiple pelvic conditions and when performing a pulmonary embolectomy.[4] Walter Cannon, a physiologist, introduced the HDT position as a treatment for shock during World War I. Cannon suggested HDT increased cardiac preload, CO, and improved blood flow to vital organs.[3]

Ostrow et al[5] performed a survey on the use of HDT by critical care nurses. Nearly all (99%) of the 494 respondents had utilized the HDT position. Thirty six percent reported they "almost always" utilized the HDT position to treat hypotension or low cardiac output, while 45% used it "sometimes." Twenty nine percent of the respondents reported HDT "almost always" improved blood pressure (BP) and CO, while 61% reported HDT "sometimes" improved these parameters. The most common non-emergent reason reported for utilizing HDT was to aid in the insertion of central IV lines. The authors suggest the utilization of HDT is "based on tradition rather than on scientific evidence."[5]

The effects of HDT on blood volume distribution in healthy, normovolemic men were studied by Bivinis et al.[6] With the use of technetium 99m marked blood, they were able to identify the blood volume distribution in supine and 15° HDT position. The data suggested an increase of blood volume in the upper compartment (above the diaphragm) and abdominal compartment (excluding urinary bladder) by 1.8% and 1.7% respectively. Simultaneously, there was a decrease of 3.2% in the lower compartment. The authors concluded the small increase of upper compartment blood volume was unlikely to have important hemodynamic or clinical effects.[6]

Ostrow et al[7] performed a straight forward assessment of HDT on non-anesthetized, post cardiothoracic surgery patients who were having a pulmonary artery catheter removed. Eighteen patients were included in the study and just prior to catheter removal patients were placed in two separate positions, 10° HDT and 30° leg elevation only. After 10 minutes in each position data were collected for CO, cardiac index (CI), mean arterial pressure (MAP), systemic vascular resistance (SVR), and partial pressure of oxygen (PO_2); no significant differences were found in any position[7].

Sibbald et al[8] studied the effects of 15-20° HDT on 76 patients admitted to a trauma unit including 15 identified as hypotensive with a mean arterial pressure (MAP) of ≤ 70 mmHg. In the hypotensive population there was no significant effect on MAP, CI, pulmonary capillary wedge pressure (PCWP), or systemic vascular resistance index (SVRI). Similarly, in the normotensive population there was no significant effect on MAP or CI, but there was a significant increase in PCWP and decrease of SVRI. The authors report there was no change of cardiac filling pressures in the hypotensive population that was seen in the normotensive

population. Additionally, they suggest there were no consistent beneficial or detrimental effects of HDT in the critically ill hypotensive or normotensive patient.[8]

Craig et al[9] looked at lung volumes and airway closure of 10 healthy individuals while in 4 positions: seated, supine, 15° HDT, and 15° HDT with the lithotomy position (hips and knees flexed). The authors found a greater impact in pulmonary function when patients changed from seated to supine position than when changed from supine to 15° HDT position. When moved to the supine position the total lung capacity (TLC), functional residual capacity (FRC), expiratory reserve volume (ERV), and residual volume (RV) all decreased significantly. Simultaneously, the closing volume increased significantly by 2.0% suggesting increased atelectasis. When moved from supine to 15° HDT the only significant changes were a further decrease in TLC and FRC; the closing volume decreased non-significantly suggesting a shift towards baseline.[9]

The current systematic review looks to compile the data of HDT on cardiopulmonary functioning in the anesthetized patient. The populations were limited to surgical patients under general anesthesia to efficaciously compare across included studies. The goal is to identify if there are known negative consequences of the HDT position on cardiopulmonary functioning, and determine if those risks outweigh the benefits.

METHODS

An extensive search on Ovid Medline, CINAHL, and Evidence-Based Medicine Reviews Multifile was conducted to identify pertinent articles. All databases were accessed through Pacific University's Library website. Search terms included: "head-down tilt," "cardiac output" "perfusion," "respiratory function tests," "anesthesia," and "hypovolemia." Limits used in the searches included "English" and "humans." The years included in the search were 1995 – 2010.

Pertinent articles were also identified in the works cited area of previously obtained articles. Selected articles were then evaluated using the "harm" critical appraisal form.

Inclusionary Criteria

Articles included utilized the head-down tilt (HDT) position of greater than, or equal to 10˚, and the populations studied were under general anesthesia. All studies were required to evaluate either cardiovascular function, pulmonary function, or both, of humans while in the HDT position.

Exclusionary Criteria

Articles excluded utilized patients with intracracranial hemorrhage, the modified Trendelenburg position (legs elevated, thorax horizontal), and simultaneous pneumoperitoneum when the effects of HDT were measured. Studies that used epidural analgesia in addition to general anesthesia, were excluded secondary to possible complicating factors. In addition, any studies that utilized lower body negative pressure devices, microgravity devices, or long-term bed rest as a component of the study were excluded.

RESULTS

Six articles were identified as fitting the criteria listed above and critically appraised. Two articles evaluated cardiovascular and pulmonary function simultaneously;[10, 11] two studies looked at pulmonary function alone,[12, 13] and another two articles looked at cardiovascular function.[14, 15] All studies were prospective and un-blinded, with only two being randomized and controlled.[10, 14] Four studies were quasi-experimental design where each subject served as his/her own control.[11-13, 15]

Cardiopulmonary Functioning

Choi et al[10] investigated the effects of head-down tilt (HDT) in the lateral decubitus position on intrapulmonary shunt and oxygenation. The populations studied were 34 adults undergoing either an open thoracotomy or video-assisted thoracoscopic surgery (VATS). All patients needed one-lung ventilation (OLV) during the procedure. Patients were excluded if they had occlusive coronary artery disease (CAD), had increased intraocular pressure, cerebrovascular disease, or their forced expiratory volume in 1 minute (FEV_1) was < 80%. The authors reported age, gender, body mass index (BMI), forced vital capacity (FVC), FEV_1, hemoglobin, and the operative side (either right or left) with no significant differences between groups.[10]

In the operating room, patients were anesthetized and intubated with a double lumen endobronchial tube. A radial artery catheter and a pulmonary artery catheter were placed and connected to sensors to collect data. Arterial and mixed venous blood samples were analyzed to determine oxygenation in addition to the cardiopulmonary parameters traditionally monitored in the surgical setting. All surgical interventions and vasoactive medications were held prior to the completion of the study protocol.[10]

The first data measurements were recorded 15 minutes after OLV had begun. Both the control and experimental groups remained in the lateral decubitus position throughout the study; the experimental group was placed in 10° HDT for a total of 10 minutes. Refer to Table 1 for study design and population; refer to Table 2 for anesthesia and ventilation interventions.

Choi et al[10] found a significant decrease in arterial oxygen tension at both 5 and 10 minutes post HDT without hypoxemia (SpO_2 < 90%). A significant increase in cardiac preload was identified, as measured by central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP), but with no significant change in cardiac index (CI). Refer to Tables 3 & 4 for results. The authors suggest patients with normal pulmonary function at base line can tolerate

HDT with OLV, but patients with decreased pulmonary function might not tolerate HDT as well.[10]

Rex et al[11] looked at multiple measures of cardiac pre-load in both the HUT and HDT positions to determine the efficacy of stroke volume variation (SVV) in predicting fluid responsiveness in ventilated patients. The study consisted of sixteen patients scheduled for elective CABG surgery. Patients were excluded if they had occlusive peripheral artery disease, intracardiac shunts, significant valvular disease, LVEF of $\leq$ 30%, or had undergone emergent CABG surgery. Of the 16 patients, two were also excluded from the study; one secondary to acute right ventricular heart failure post cardiopulmonary bypass weaning, and the other because of poor echocardiogram images.[11]

A variety of cardiac parameters were measured including SVV, which is a dynamic measurement "derived from cyclic changes of stroke volume induced by heart-lung interactions."[11] Additionally, static parameters including CVP, PCWP, and intrathoracic blood volume index (ITBVI) were measured. Baseline measurements were taken immediately following chest closure in the CABG surgery. Patients were then placed in a 30° HUT for 5 minutes, measurements were repeated, then placed in 30° HDT, and final measurements taken. Refer to Table 1 for study design and population; refer to Table 2 for anesthesia and ventilation interventions.[11]

The respiratory functions analyzed included peak inspiratory pressure (P_{insp}), peak end expiratory pressure (PEEP), tidal volume (V_T), and respiratory rate (RR). The inspiratory pressure was adjusted to maintain a V_T of approximately 8 ml/kg. The inspiratory-to-expiratory ratio was set at 1:1 throughout the study period. No significant differences were found in any of the respiratory variables.[11]

When compared to baseline, the HDT position showed a significant increase in cardiac pre-load as measured by CVP, PCWP, mean pulmonary artery pressure (MPAP), and left ventricular end-diastolic area index (LVEDAI). An increase in cardiac performance was also seen as measured by CI, mean arterial pressure (MAP), and stroke volume index (SVI). SVV showed no significant change in the HDT group when compared to baseline, but a significant increase was found in the HUT group when compared to baseline. Changes in SVI were significantly correlated with changes in CVP, ITBVI, and SVV. Refer to Tables 3 & 4 for results. The authors suggest HDT increases cardiac pre-load, and SVV was a reliable measure of cardiac pre-load in the cardiothoracic surgical patients.[11]

Pulmonary Functioning

Fahy et al[12] investigated lung and chest wall mechanics of patients in 10° HDT position. A total of 15 adult patients scheduled for elective laparoscopic surgery participated in the study. All data for the study were collected prior to pneumoperitoneum. The authors reported age, gender, height, weight, and smoking history. Eight patients had a history of smoking but none had a history of pulmonary disease.[12]

Two pressure transducers were used in this study: one placed in the esophagus, the other connected to a port on the endotracheal tube. Additionally, there was an airflow meter attached to the endotracheal tube. The differences in pressures and flow allowed the authors to calculate a variety of mechanical impedances to lung and chest wall inflation. At each data recording, 3 consecutive breaths were measured for each of the 8 ventilator settings. Each cycle of 3 breaths was repeated for a total of 3 sets of recordings each done 3 minutes apart. After baseline measurements were recorded, data were collected at 10° of head up tilt (HUT), then 15° HDT for

ten patients and the inverse order for the remaining five patients. Refer to Table 1 for study design and population; refer to Table 2 for anesthesia and ventilation interventions.[12]

Eight different combinations of respiratory frequency and tidal volume were measured at each body position in the following protocol: 10 breaths per minute at 800 ml, 250 ml, and 500 ml; 20 breaths per minute at 500 ml, 800 ml, and 250 ml; 30 breaths per minute at 250 ml and 500 ml. After a change in body position or change in ventilator settings, experimental data were collected after three breaths were given to allow for normalization at that position or setting. This protocol was followed except in a few instances when patient care dictated slight changes. The data were reported as impedance, a combination of elastances and resistances. Lung impedance was defined as a measure of alveolar pressure, while chest wall impedance was defined as a measure of intrathoracic pressure.[12] Elastance and resistance were not explicitly defined by the authors.[12]

The data in the supine position showed greater impedance in the lungs when compared to the chest wall. Additionally, as respiratory rate increased, there was an increase in elastances and a decrease in resistances.[12]

In the HDT position there was a significant increase of lung and total respiratory system impedance when compared to the supine position. There was no significant change in chest wall impedance. This suggests an increased effort to expand the lungs, but no concomitant increased effort to expand the chest wall in the HDT position. A positive correlation was identified between lung elastances and BMI. Refer to Table 3 for results. The authors suggest HDT causes a decrease in functional residual capacity (FRC) and causes microatelectasis secondary to the gravitational effects of abdominal contents. The authors suggest HDT increases the impedance of lung inflation, but not chest wall mechanics, and is tolerated by patients with normal

pulmonary function; they suggest patients with pulmonary disease or who are obese may have increased negative consequences.[12]

Regli et al[13] looked at the effects of HDT on FRC and the lung clearance index (LCI), a measure assessing peripheral airway collapse and atelectasis. Twenty children with known congenital heart defects undergoing cardiac surgery were studied. The authors reported the mean age, range of ages, height, and weight. No other population characteristics were reported. The study was conducted around the time of central line insertion when the patients were in a 30˚ HDT for 10-15 minutes.[13]

An airflow meter was connected to the endotracheal tube and a tracer gas was utilized to measure FRC and LCI. LCI is determined by measuring "the number of lung volume turnovers needed to clear the lungs of the marker gas," divided by FRC. Data were recorded 4 times: at 5 minutes post intubation, after the central line insertion (HDT), at 5 minutes after returning to horizontal position, and after a lung recruitment maneuver. The lung recruitment maneuver was done by increasing peak inspiratory pressure to 37-40 cmH$_2$O for 10 consecutive breaths. Refer to Table 1 for study design and population; refer to Table 2 for anesthesia and ventilation interventions.[13]

The data showed a significant decrease in FRC and an increase in LCI in the 30˚ HDT position indicating increased atelectasis. When the patients were returned to horizontal position both the FRC and LCI improved significantly, but were still significantly impaired when compared to baseline measurements. Post lung recruitment maneuver there was a significant improvement in FRC and LCI and results returned to baseline levels. Refer to Table 3 for results. The authors suggest a 30˚ HDT causes atelectasis which is not reversed with a return to

horizontal position. Additionally, they suggest a lung recruitment maneuver should be performed after the use of HDT to restore baseline pulmonary functioning.[13]

Cardiovascular Functioning

Kardos et al[14] looked at whether the HDT position would compensate for the known drop in cardiac output (CO) secondary to induction with propofol. Thirty children who were scheduled for elective orthopedic surgery were randomized to the HDT group or the horizontal control group. Non-invasive measurements were taken using an impedance cardiograph to calculate stroke volume, from which many other cardiac parameters could be calculated. Measurements were taken prior to induction, at 3 and 5 minutes post HDT, and then finally at 3 minutes post return to horizontal position; the patients were placed in HDT immediately after induction. Refer to Table 1 for study design and population; refer to Table 2 for anesthesia and ventilation interventions.[14]

Heart rate was the only parameter found to be significantly different between the HDT and control groups, and only at 3 minutes post HDT. The expected drop in CO, as measured by the cardiac index (CI), a calculation of CO / body surface area (BSA), was seen post induction in all patients. There was no difference in CI found between the HDT and the control groups. A decrease in mean arterial blood pressure (MAP) was also seen post induction in all patients. The stroke volume index (SVI), stroke volume (SV) / BSA, increased significantly at 3 min. post HDT, but not at 5 minutes. The Heather Index (HI), a measure of cardiac contractility, showed a significant decrease in contractility at all time measurements post induction, including after a return to horizontal position. Refer to Tables 3 and 4 for results. The data suggest there was a significant decrease in cardiac output and performance post induction as measured by MAP, CI,

and HI despite the increase in SVI. The authors suggest a 20˚ HDT does not prevent a decrease in CI post induction with propofol in children.[14]

Reuter et al[15] studied the effects of HDT on the intrathoracic blood volume index (ITBVI) and cardiac performance in the hypovolemic patient. All patients had just undergone elective coronary artery bypass grafting (CABG) procedure and had normal pre-op left ventricle ejection fraction (LVEF). On a post-op transesophageal echocardiogram, all patients were found to have "kissing papillary muscles" suggesting a hypovolemic status. No ages or other basic demographics were reported in the article.[15]

A central venous catheter, pulmonary artery catheter, and femoral artery catheter were accessed to obtain data via thermodilution and dye injection. These processes, in addition to the transoesophageal echocardiogram and hemodynamic monitoring provided the means of data collection. Cardiac output was measured via arterial thermodilution as well pulmonary artery thermodilution. Similarly, the ITBVI was measured via thermodilution as well transpulmonary arterial dye dilution. Baseline data were collected, patients were placed in 30˚ HDT for 15 minutes, and then returned to horizontal position. Data were collected in each position in triplicate, with a 3 minute interval between recordings, and after a 3 minute window post change to a new position. The three readings for each position were averaged. Refer to Table 1 for study design and population; refer to Table 2 for anesthesia and ventilation interventions.[15]

The cardiac filling pressures, as measured by CVP and pulmonary capillary wedge pressure (PCWP), showed a significant increase. The ITBVI measurements, also measuring cardiac preload, showed significant increases of 4.6% and 3.8% in the thermodilution and dye dilution methods respectively. No significant improvement of cardiac performance, as measured by MAP and CI, was found. There was a significant decrease in both MAP and CI when the

patients were brought back to the horizontal position after 15 minutes of 30° HDT. Refer to Tables 3 & 4 for results. The authors suggest the HDT is not an efficient method to increase cardiac preload after cardiac surgery with cardiopulmonary bypass.[15]

DISCUSSION

An extensive search was completed to identify the six articles that have been included in this systematic review. Each of the articles looked at head down tilt (HDT) of greater than, or equal to 10°, on the effects of cardiopulmonary functioning in the anesthetized patient. Only two articles were randomized and controlled studies, while the remaining four were quasi-experimental. The study size ranged from 12 to 20 subjects per group. Three studies looked at adults,[10-12] 2 studies looked at children,[13, 14] and 1 study did not report ages.[15]

Study Design Limitations

Choi et al[10] provided the most complete picture of cardiopulmonary functioning of any study found while in the HDT position, and was one of only two randomized and controlled studies identified for this systematic review. Their data, however, should be carefully interpreted, especially when extrapolated to other populations. The patients in this study were in the lateral decubitus position throughout the study period, and were the only population undergoing one-lung ventilation (OLV).[10]

The study design isolated the HDT variable during the study period, and after baseline data were collected, 2 measurements were taken in the 10° HDT position. The authors reported significant changes in parameters when compared to baseline as well as compared to the control group. They did not, however, report statistical differences between the 2 readings in the HDT position. This would have identified if the study parameters had stabilized during the HDT position.   This study adds valuable information by suggesting HDT causes a decrease in oxygen

delivery capacity but without clinically significant hypoxemia, as well as an increase in cardiac

preload but without subsequent increased cardiac output.[10] This study can be used to suggest

physiologic trends in the HDT position, but should not be directly applied to the general patient

population secondary to complicating factors of OLV such as the influence of intrapulmonary

shunting.[10]

Rex et al[11] primarily looked at a new method, stroke volume variation (SVV), of

assessing fluid responsiveness. They did, however, also report cardiopulmonary functioning at

30 HDT compared to baseline. The design study utilized a 30 HUT position to cause a

relative decrease in central blood volume, followed by a 30 HDT to cause a relative increase of

intrathoracic blood volume. The application of HUT prior to HDT, and presumed decrease in

central blood volume, is a possible complicating factor when interpreting the results of this

study.[11]

The authors report, "all patients were anaesthetized and underwent mechanical

ventilation,"[11] but did not report any of the anesthesia medications utilized. Using medications,

such as propofol, can cause an expected drop in CO.[14] By not reporting the type or dosing of

anesthetics utilized, it decreases the reproducibility of the study and leads the reader to make

assumptions.[11]

Rex et al[11] found a significant increase in all 3 pressure cardiac preload parameters

studied, but interestingly only 1 of the 3 volumetric cardiac preload parameters showed a

significant increase. This could be secondary to the study design of inducing a relative low

central blood volume just prior to the HDT treatment. This was also the only study to identify an

increase in all three cardiac performance parameters studied (MAP, SVI, and CI).[11]

Fahy et al[12] had a good design study that looked directly at the effects of lung compliance in the HDT and HUT positions. As with Rex et al,[11] however, this study implemented the HUT position just prior to the HDT treatment. After a change in position or ventilator settings, the study protocol only allowed 3 breaths for parameters to stabilize prior to taking experimental data. The limited amount of time might not have allowed blood volume re-distribution to occur and therefore underestimate the influence of intrathoracic pressure and venous return caused by a blood volume shift. This effect, however, would likely have less influence on pulmonary function when compared to cardiovascular function.[12]

The authors defined impedance as a combination of elastance and resistance and provided a working definition of lung and chest wall impedance, however, they did not define elastance and resistance.[12] Stedman's dictionary defines elastance as, "a measure of the tendency of a structure to return to its original form after removal of a deforming force." Resistance is defined as, "the flow of gases during ventilation resulting from obstruction or turbulent flow in the upper and lower airways."[16]

The important contribution of this study is that it suggests in a population with normal pulmonary function there is increased atelectasis and lung impedance with the HDT position. Additionally, they found that chest wall impedance was not affected by body position. The authors suggest the mechanism of increased lung impedance and atelectasis was secondary to abdominal contents pushing on the diaphragm.[12] These pulmonary changes were tolerated well by patients with normal pulmonary function, however, patients who are obese or have pre-existing pulmonary conditions, such as chronic obstructive pulmonary disease (COPD), are at increased risk of negative pulmonary consequences.[12]

Regli et al[13] added interesting data about pulmonary functioning during $3\mathring{o}$ HDT, but did not provide much information about the study population. The participants included children with congenital heart defects that required surgery. The genders, body mass index (BMI), cardiac output (CO), and other cardiac functioning tests were not reported for the study population. Lung functioning tests, functional residual capacity (FRC) and lung clearance index (LCI), were studied in the HDT position, horizontal position, and post lung recruitment maneuver. The authors, however, do not report duration in HDT, or time post HDT the lung recruitment maneuver was performed.[13]

This study suggests there is a decrease of FRC and an increase of atelectasis with a $3\mathring{o}$ HDT that is not resolved by a return to horizontal position. In the population studied, this provides strong evidence against the use of HDT, especially since a lung recruitment maneuver was required to return to baseline pulmonary functioning. The data from this article, however, should be cautiously applied to other populations secondary to limited information about the study population.[13]

Kardos et al[14] presented data on cardiovascular functioning and $2\mathring{o}$ HDT after induction. The authors looked at the question if HDT could prevent the expected drop of CO secondary to propofol use. This study met the criteria of inclusion in the present systematic review, however, the study design makes it difficult to compare to other studies secondary to baseline measurements being taken prior to induction. The baseline measurements were then compared to experimental data post induction and post HDT treatment. This means the effects of HDT were not isolated from the effects of induction in the experimental group.[14]

The only data from this article that is useful in the present systematic review is the comparison between the HDT group and the supine group (SG). Heart rate was the only variable

found to be significantly different between groups, and only at 3 minutes post HDT. There was no significant change in cardiac output between groups as measured by MAP, SVI, CI, and HI. The expected drop in cardiovascular parameters was seen post induction, and the authors suggest HDT does not prevent a decrease in CO post induction with propofol in children.[14]

Reuter et al[15] developed a good study design, but similar to Regli et al,[13] reported limited population demographics making it difficult to apply the results to a larger population. The population studied had normal pre-operative left ventricular ejection fraction (LVEF), were immediately post elective coronary artery by-pass graft surgery (CABG), and were identified as having "kissing papillary muscles" on transesophageal echocardiogram; no other pertinent population characteristics were reported including age, gender, BMI, or other characteristics.[15]

The study design included data measurements at 3, 6, and 9 minutes post HDT. The authors reported the data as an average of all three measurements as opposed to individual data points. This eliminates the possibility to identify a temporal relationship between HDT and the parameters studied.[15]

This study was unique in this systematic review because of the population inclusion criteria of hypovolemia. The major contribution of this study is the finding of consistent and significant improvement in cardiac preload, but no concomitant increase of cardiac performance, as measured by MAP and CI.[15] This is in agreement with prior research of HDT in hypovolemic patients.[8]

Major Findings by Parameters

None of the pulmonary parameters looked at were repeated across studies, therefore no cross validity could be analyzed. Oxygenation parameters studied by Choi et al[10] found a decrease in arterial oxygen tension and an increase in both arterial-alveolar oxygen gradient and

pulmonary shunt fraction. Lung mechanics showed a decrease in FRC, an increase in lung and total respiratory system impedance, as well as an increase in atelectasis as measured by LCI.[12, 13]

No significant effects on heart rate were found by any study with the exception of Kardos et al.[14] These changes, however, are more likely explained by the use of propofol during induction than the use of HDT position.

Cardiac preload was the variable found to be the most consistent across the studies. Cardiac preload, as measured by pressure sensors of CVP, PCWP, and MPAP showed a significant increase in 8 out of 9 individual data collection points across three studies. Choi et al,[10] the only exception, showed no change in CVP at 5 minutes post 10° HDT but found a significant increase at 10 minutes post HDT. The only significant difference found between the experimental and control groups, however, was an increase of CVP 5 minutes post HDT.[10]

Cardiac preload, as measured by volume indices of ITBVI, LVEDAI, and SVV showed a significant increase in 3 out of 5 individual data collection points across three studies. Rex et al [11] did not find a significant increase in ITBVI in 30° HDT, though this might have been complicated by the study design of 30° HUT followed by 30° HDT. In the HUT position there was a non-significant decrease in ITBVI when compared to baseline, therefore, an even greater absolute increase of ITBVI in HDT position would be required to show a significant increase when compared to baseline. Stroke volume variation was the other volumetric cardiac preload parameter that did not show any significant change.

Cardiac output and performance as measured by MAP, SVI, CI, and HI did not show much consistency across studies, and is one of the more important parameters in determining actual benefits of the HDT position. Two studies[10, 15] found no significant difference of MAP. Rex et al[11] found a significant increase in MAP while in HDT , though as discussed above, the

study design of 3⁰ HUT position just prior to 3⁰ HDT may have influenced the data collected. Kardos et al[14] also found a significant decrease in MAP, though the influence of induction with propofol probably had a greater influence on MAP than the HDT position. The two randomized, controlled studies[10, 14] did not find any significant differences in MAP between the experimental and control groups.

Stroke volume index showed a significant increase in 2 out of 3 individual data collection points across 2 studies[11, 14]. Kardos et al[14] found a significant increase of SVI at 3 minutes post 2⁰ HDT, but found no difference at 5 min. post HDT. No significant difference was found between the experimental and control groups.

Cardiac index was studied for a total of 6 individual data collection points across 4 studies, with 3 data points finding no difference, 2 finding a decrease, and 1 finding a significant increase. Choi et al[10] found no significant differences when compared to baseline or the control group. Kardos et al[14] found a significant decrease in CI when compared to baseline, but similarly, did not find any difference between groups. Reuter et al[15] found no difference, while Rex et al[11] was the only study to find a significant increase in CI.

The Heather index (HI) is a measure of cardiac contractility as measured by aortic ejection acceleration. Kardos et al[14] were the only authors to look at the HI, and found a significant decrease. The findings of decreased cardiac performance (MAP, CI, and HI) when compared to baseline measurements found by Kardos et al, however, is most likely a reflection of the effects of induction with propofol as opposed to the effects of HDT.

Systematic Reviews

Shammas et al[3] performed a systematic review of 5 articles looking at HDT in a variety of populations including healthy subjects and anesthetized patients; two articles were also

included in the present study.[12, 15] The authors report the "research findings do not provide strong support for the use of Trendelenburg positioning as an intervention for hypotension," and report adverse consequences have been identified in the literature. Additionally, they further suggest patients who are obese, have compromised right ventricular ejection fraction, pulmonary disorders, or head injuries should not be placed in the HDT position. Their conclusion is the HDT position should not be used to treat shock until more conclusive data on the effects of the HDT position is identified.[3]

Bridges et al[1] reviewed 8 articles and 3 text books pertaining to HDT in both healthy subjects and anesthetized patients; one article was also included in the present study.[15] The authors report the literature on HDT is scarce, lacks strength, and state HDT is probably not useful for fluid resuscitation. The authors suggest the following clinical guidelines:

HDT is useful for:

      Insertion or removal of central catheters

      Certain spinal anesthetic techniques

HDT is probably not indicated or may have harmful effects in:

      Resuscitation of patients who are hypotensive

      Patients in whom mechanical ventilation is difficult, or patients with decreased vital capacity

      Patients who have increased intracranial pressure

      Patients who have cerebral edema

      Patients who have increased intraocular pressure

      Patients with ischemia of the lower limbs[1]

CONCLUSION

This systematic review attempted to compile the current data on the effects of head-down tilt (HDT) on cardiopulmonary functioning in the anesthetized patient. One striking point is the lack of clear evidence to support a treatment procedure that has been in use for almost 100 years or more. The data from this review support the theory that HDT increases cardiac preload, but overall, there is no consequent increase in cardiac performance. This is also found when confining the research to hypovolemic patients.[8, 15]

The data compiled in this review suggests there are multiple negative consequences of HDT on pulmonary functioning. Head down tilt causes a decrease in functional residual capacity (FRC), an increase in atelectasis, and a decrease in oxygenation.[9, 10, 12, 13] The general consensus of the research is that this decrease of pulmonary capacity is tolerated well by patients with normal pulmonary function. A majority of the studies, however, concluded that patients with pulmonary disease, such as obstructive pulmonary conditions, could be more susceptible to negative pulmonary consequences in the HDT position.

In the controlled environment of surgery, the HDT position should be utilized judiciously and for as short a duration as possible. Likely scenarios where HDT would be advantageous would be to aid in the insertion of a central venous catheter or increase the exposure during a surgical procedure. Despite the lack of clear evidence, HDT should be avoided in patients who are obese, have pre-existing obstructive pulmonary disorders, have New York Heart Association class III heart failure, or other significant cardiopulmonary dysfunction.

The evidence in anesthetized patients suggests HDT provides limited and inconsistent cardiovascular benefits along with adverse pulmonary consequences. The trends gathered from this data can be applied to the general patient population and non-anesthetized patients. Patients

who present with acute hypotension, or shock, would be better served initiating parenteral fluid resuscitation as opposed to the HDT position, especially when pulmonary capacity is unknown. Further education about the adverse consequences of HDT position should be disseminated through the healthcare system to ensure the HDT position is used judiciously and to ensure the risk to benefit ratio has been fully explored for each patient.

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TABLES
Table 1: Study Design and Population

Parameters		Cardiopulmonary		Pulmonary		Cardiovascular	
Study		**Choi et al[10]**	**Rex et al[11]**	**Fahy et al[12]**	**Regli et al[13]**	**Kardos et al[14]**	**Reuter et al[15]**
Study Design		All studies were prospective and un-blinded*					
		•Randomized controlled trial	•Quasi-experimental	•Quasi-experimental	•Quasi-experimental	•Randomized controlled trial	•Quasi-experimental
Population							
Total #		•34 patients; 17/group	•14 patients	•15 patients	•20 patients	•30 patients; 15/group	•12 patients
Age		•Mean control: 52.7 y/o •Mean HDT: 53.9 y/o	•45 – 84 y/o •Mean: 63	•26 – 78 y/o	•3 mo. – 8 y/o	•7 – 16 y/o •Median control: 12 y/o •Median HDT: 12 y/o	•Not stated
Surgical intervention		•Thoracotomy or VATS •One-lung ventilation needed	•Immediately post elective CABG	•Elective laparoscopic surgery	•Congenital heart surgery	•Minor orthopedic surgery	•Immediately post elective CABG •Hypovolemic
Procedure							
Intervention		•10˚ HDT in lateral decubitus position for 10 min. •Control: lateral decubitus throughout	•30˚ HDT for 5 min.	•15˚ HDT for 15 min. •All data prior to pneumoperitoneum	•30˚ HDT for 10-15 min.	•20˚ HDT for 5 min. •Control: horizontal throughout	•30˚ HDT for 15 min.
Protocol	T_0	•15 min. post OLV	•Immediately post CABG	•Immediately post induction†	•5 min. post intubation	•Prior to anesthesia	•Immediately post CABG‡
	T_1	•5 min. post 10˚ HDT	•5 min. post 30˚ HUT	•Post 3 initial breaths in 10˚ HUT†	•10-15 min. post 30˚ HDT	•3 min. post 20˚ HDT / induction	•3, 6, & 9 min. post 30˚ HDT, averaged together
	T_2	•10 min. post 10˚ HDT	•5 min. post 30˚ HDT	•Post 3 initial breaths in 15˚ HDT†	•N/A	•5 min. post 20˚ HDT / induction	•N/A
	T_P	•10 min. post horizontal position	•N/A	•N/A	•5 min. post horizontal position •Post lung recruitment maneuver (time not specified)	•3 min. post horizontal position	•3 min. post horizontal position‡

HDT = head-down tilt; HUT = head-up tilt; VATS = video-assisted thoracoscopic surgery; CABG = coronary artery bypass graft; OLV = one lung ventilation; T_0 = baseline supine measurements; T_1 = time at first experimental measurements; T_2 = time at second experimental measurements; T_P = Time post experimental intervention

* Reuter et. al. echocardiograms were read by blinded radiologists

† 3 consecutive breaths were measured, each set was at one of 8 different respiratory frequency and tidal volume combinations

‡ Measurements taken in triplicate 3 min. apart and averaged together

Table 2: Anesthesia and Ventilation Interventions

Parameters		Cardiopulmonary		Pulmonary		Cardiovascular	
Study		Choi et al[10]	Rex et al[11]	Fahy et al[12]	Regli et al[13]	Kardos et al[14]	Reuter et al[15]
Anesthesia							
Pre-Medication(s)		•NR	•NR	•No pre-medication	•Midazolam 0.5 mg/kg 30 min. prior	•Midazolam 0.3-0.5 mg/kg 60 min. prior	•NR
Induction Medication(s)	**Muscle Relaxant**	•Rocuronium 0.9 mg/kg	•NR	•Mivacurium 0.1-0.2 mg/kg •Succinylcholine 1.0-1.5 mg/kg (when risk for aspiration)	•Atracurium 0.5 mg/kg	•NR	•Pancuronium (dose NR)
	General Anesthetic	•Thiopental 5 mg/kg		•Propofol 1-2 mg/kg •Thiopental 5 mg/kg	•Sevoflurane up to 5% OR propofol 2-3 mg/kg •Ketamine 0.15 mg/kg	•Propofol 3 mg/kg	•Midazolam (dose NR)
	Analgesia	•Fentanyl 2μg/kg		•NR	•Sufentanyl 0.5 μg/kg	•Fentanyl 1.5 μg/kg	•Sufentanyl (dose NR)
Maintenance Medication(s)	**Muscle Relaxant**	•Vecuronium 1-2 μg/kg/min	•NR	•Mivacurium (dose determined by peripheral nerve stimulation)	•NR	•NR	•NR
	General Anesthetic	•Isoflurane 0.8%-1%		•Isoflurane 0.4%-1.0% •Nitrous Oxide 70%	•Propofol 8-10 mg/kg/h •Ketamine 0.1 mg/kg	•Propofol 8 mg/kg/h •Nitrous Oxide 70%	•Isoflurane (dose NR)
	Analgesia	•Remifentanil 0.05-0.2 μg/kg/min		•NR	•Sufentanil 0.5-1.0 μg/kg	•NR	•Sufentanil (dose NR)
Ventilation				•Except study period†			
Frequency		•8-12 breaths/min.	•12*	•10 breaths/min.	•Determined by ETCO$_2$	•NR	•NR
Tidal Volume		•8-10 mL/kg	•6.65 ml/kg*	•Determined by PCO$_2$	•10 "mg/kg"	•NR	•NR
PEEP		•None	•3.43*	•5 cm H$_2$O	•3 cm H$_2$O	•5 cm H$_2$O	•5 cm H$_2$O
Peak Airway Pressure		•NR	•NR	•NR	•NR	•12-14 cm H$_2$O	•14-16 cm H$_2$O
Peak Inspiratory Pressure		•NR	•22 mm Hg*	•NR	•NR	•NR	•NR
Arterial PCO$_2$		•33-38 mm Hg	•NR	•30-40 mm Hg	•NR	•NR	•NR
End-Tidal CO$_2$		•NR	•NR	•NR	•5 kPa	•4.3-5.2 kPa	•NR
Inspiratory/Expiratory Ratio		•1:1.9	•1:1	•1:1	•NR	•NR	•NR

NR = not reported; ETCO$_2$ = end-title carbon dioxide; PEEP = peak end expiratory pressure; PCO$_2$ = partial pressure of carbon dioxide

*Averages measured during HDT; inspiratory pressure was adjusted to maintain a V$_T$ of about 8 ml/kg

†See "Results" section for a description of ventilator settings during study period

Table 3: Cardiopulmonary Parameters Studied and Significant Results, Grouped by Authors

Parameter	Cardiopulmonary				Pulmonary		Cardiovascular	
Study	Choi et al[10]		Rex et al[11]		Fahy et al[12]	Regli et al[13]	Kardos et al[14]	Reuter et al[15]
Parameters Reported	RESP: •PaO_2 •$A\text{-}aO_2$ •Qs/Qt •PAW •Cdyn	CV: •HR •MAP •PCWP •CVP •CI •SVRI •PVRI	RESP: •P_{insp} •PEEP •V_T •RR	CV: •HR •MAP •CVP •MPAP •PCWP •CI •SVRI •SVV •ITBVI •LVEDAI	•E_L •R_L •E_{cw} •R_{cw} •E_{rs} •R_{rs}	•FRC •LCI	•HR •MAP •CI •SVI •SVRI •HI	•MAP •CI (2 methods) •SVRI •CVP •PCWP •ITBVI (2 methods) •LVEDAI
Significant Results of HDT Position (P = < 0.05 for either T_1 or T_2)	RESP: HDT vs. control: •↓ PaO_2 •↑ $A\text{-}aO_2$ HDT vs. baseline: •↓ PaO_2 •↑$A\text{-}aO_2$ •↑ Qs/Qt	CV: HDT vs. control: •↑ CVP •↑ PVRI HDT vs. baseline: •↑ CVP •↑ PCWP	(None)	CV: •↑ CI •↑MAP •↑ SVI •↑ CVP •↑PCWP •↑ MPAP •↑LVEDAI	•↑ E_L •↑ R_L •↑ E_{RS} •↑ R_{RS}	•↓ FRC •↑ LCI	HDT vs. control: •↓ HR HDT vs. baseline: •↓ HR •↓ MAP •↓ CI •↑SVI •↓ HI	•↑ CVP •↑ PWCP •↑ ITBVI (both methods) •↑ LVEDAI
Conclusions by Authors	•Data showed sig. ↓ of PaO_2 with no dangerous hypoxemia; ↑ in cardiac preload but no Δ in CI •sig. ↑ of intrapulmonary shunt •No sig. negative effects of 10 min. 1ð HDT in OLV patients with normal pulmonary function		•Data showed sig. ↑ in cardiac preload and cardiac output measures •HDT causes an intrathoracic fluid shift that can be accurately assessed by SVV		•Data showed sig. ↑ in lung and total resp. sys. impedances •HDT causes a ↓ in FRC and causes microatelectasis but is tolerated well in non-obese Pt's w/ norm. pulmonary function	•Data showed sig. ↓ in FRC and ↑ atelectasis •3ð HDT required a lung recruitment maneuver to restore baseline pulmonary functioning •HDT has neg. consequences on pulm. function in children	•Data showed sig. ↓ in HR between groups •CI was decreased in all groups post induction •20˚ HDT does not prevent a drop in CI post induction	•Data showed sig. ↑ in cardiac preload by pressure and volume measures •No improvement in cardiac performance •3ð HDT is not an efficient maneuver to ↑ cardiac preload

HDT = head-down tilt; PaO_2 = arterial oxygen tension; $A\text{-}aO_2$ = alveolar-arterial oxygen gradient; Qs/Qt = pulmonary shunt fraction; PAW = peak airway pressure; Cdyn = dynamic compliance; HR = heart rate; MAP = mean arterial pressure; CVP = central venous pressure; MPAP = mean pulmonary artery pressure; PCWP = pulmonary wedge capillary pressure; CI = cardiac index; SVRI = systemic vascular resistance index; SVV = stroke volume variation; ITBVI = intrathoracic blood volume index; LVEDAI = left ventricle end-diastolic area index; E_L = lung elastance; R_L = lung resistance; E_{cw} = chest wall elastance; R_{cw} = chest wall resistance; E_{rs} = total respiratory system elastance; R_{rs} = total respiratory system resistance; FRC = functional residual capacity; LCI = lung clearance index; HI = Heather index

Table 4: Results of Hemodynamics in Head-Down Tilt, Grouped by Parameter

Parameter Grouping	Parameter	Study	HDT Compared to Supine Baseline (P = < 0.05)				HDT Compared to Control Group (P = < 0.05)			
			T_1	T_2	T_P		T_0	T_1	T_2	T_p
HR	HR	Choi et al[10]	=	=	=		=	=	=	=
		Kardos et al[14]	↓↓	↓↓	↓↓		=	↓↓	=	=
		Rex et al[11]	N/A	=	N/A		N/A	N/A	N/A	N/A
Cardiac Preload (Pressure)	CVP	Choi et al[10]	↑↑	↑↑	=		=	↑↑	=	=
		Reuter et al[15]	↑↑	N/A	↓↓		N/A	N/A	N/A	N/A
		Rex et al[11]	N/A	↑↑	N/A		N/A	N/A	N/A	N/A
	PCWP	Choi et al[10]	=	↑↑	=		=	=	=	=
		Reuter et al[15]	↑↑	N/A	↓↓		N/A	N/A	N/A	N/A
		Rex et al[11]	N/A	↑↑	N/A		N/A	N/A	N/A	N/A
	MPAP	Rex et al[11]	N/A	↑↑	N/A		N/A	N/A	N/A	N/A
Cardiac Preload (Volume)	ITBVI	Reuter et al[15]	↑↑	N/A	=		N/A	N/A	N/A	N/A
		Rex et al[11]	N/A	=	N/A		N/A	N/A	N/A	N/A
	LVEDAI	Reuter et al[15]	↑↑	N/A	=		N/A	N/A	N/A	N/A
		Rex et al[11]	N/A	↑↑	N/A		N/A	N/A	N/A	N/A
	SVV	Rex et al[11]	N/A	=	N/A		N/A	N/A	N/A	N/A
Cardiac Output and Performance	MAP	Choi et al[10]	=	=	=		=	=	=	=
		Kardos et al[14]	↓↓	↓↓	↓↓		=	=	=	=
		Reuter et al[15]	=	N/A	↓↓		N/A	N/A	N/A	N/A
		Rex et al[11]	N/A	↑↑	N/A		N/A	N/A	N/A	N/A
	SVI	Kardos et al[14]	↑↑	=	=		=	=	=	=
		Rex et al[11]	N/A	↑↑	N/A		N/A	N/A	N/A	N/A
	CI	Choi et al[10]	=	=	=		=	=	=	=
		Kardos et al[14]	↓↓	↓↓	↓↓		=	=	=	=
		Reuter et al[15]	=	N/A	↓↓		N/A	N/A	N/A	N/A
		Rex et al[11]	N/A	↑↑	N/A		N/A	N/A	N/A	N/A
	HI	Kardos et al[14]	↓↓	↓↓	↓↓		=	=	=	=
Vasculature Resistance	SVRI	Choi et al[10]	=	=	=		=	=	=	=
		Reuter et al[15]	=	N/A	=		N/A	N/A	N/A	N/A
		Rex et al[11]	N/A	=	N/A		N/A	N/A	N/A	N/A
	PVRI	Choi et al[10]	=	=	=		=	↑↑	↑↑	=

↑↑ = Sig. Increase; (=) = No Sig. Change; ↓↓ = Sig. Decrease; N/A = Not Applicable

T_0 = baseline supine measurements; T_1 = time at first experimental measurements; T_2 = time at second experimental measurements; T_P = Time post experimental intervention; HR = heart rate; CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; MPAP = mean pulmonary artery pressure; ITBVI = intrathoracic blood volume index; LVEDAI = left ventricular end-diastolic area index; SVV = stroke volume variation; MAP = mean arterial pressure; SVI = stroke volume index; CI = cardiac index; HI = heather index; SVRI = systemic vascular resistance index; PVRI = peripheral vascular resistance index