The Effects of Thoracic Blood Volume on the Valsalva Maneuver

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Abstract

The Valsalva maneuver (VM) is frequently used to test autonomic function. However, the VM is also affected by changes in blood volume and by blood volume redistribution. We hypothesized that even a standardized VM may produce a wide range of thoracic blood volume shifts. Larger blood volume shifts in some normovolemic individuals may be sufficient to induce decreases in blood pressure which preclude autonomic restoration of BP in phase II of VM. To test this hypothesis we studied 17 healthy volunteers aged 15-22 years. All had similar supine and upright vasoconstrictor responses and normal blood volume. We assessed changes in thoracic blood volume by impedance plethysmography before and during the VM performed supine. In some subjects, large decreases in BP were produced by thoracic hypovolemia. The maximum fractional decrease in BP correlated well (r²=0.64, p<.001) with thoracic hypovolemia and with systolic blood pressure at the end of phase II of the Valsalva maneuver (r²=0.67, p<.001). The blood pressure overshoot in phase IV of the maneuver was uncorrelated to phase II changes suggesting intact autonomic vasoconstriction. We conclude that the blood pressure decrease during the Valsalva maneuver is related to a variable decrease in thoracic blood volume which may be sufficient to preclude pressure recovery during phase II even with normal resting peripheral vasoconstriction. The Valsalva maneuver depends on vascular as well as autonomic activation, which broadens its utility but complicates its analysis.

Key Words

Valsalva maneuver, vasoconstriction, veins, blood volume
Introduction

The Valsalva maneuver is classically defined as forcible exhalation against a closed glottis thereby increasing pressure within the thoracic cavity and impeding venous return of blood to the heart (11). Pleural pressure so obtained is highly variable; therefore, for comparative purposes investigators use the “quantitative Valsalva maneuver” wherein the subject exhales with open glottis against a known expiratory pressure (typically 30-40 mmHg) generating comparable increases in intrapleural and right atrial pressures (15). This is thought to reduce variation in venous return due to differences in straining between subjects providing a standard volume change stimulus.

The response of heart rate and blood pressure to the maneuver comprises one of the most frequently used tests of circulatory autonomic function (23). Typical arterial pressure and heart rate changes are shown in figure 1. There is a brief increase in blood pressure, denoted phase I, during which there is a mechanical increase in blood pressure due to the propulsion of blood from the thorax. This is followed by a decrease in blood pressure and baroreflex mediated rise in heart rate produced by decreased venous return and consequent decreased cardiac output and blood pressure during early phase II. Late phase II is marked by recovery of blood pressure produced by a combination of vasoconstriction, sympathetic cardiac stimulation and tachycardia. Once exhalation is complete, the release of strain will result in restoration of the normal negative intrathoracic pressure, leading to the blood pressure drop of phase III which results in rapid refilling of the thoracic vasculature. This is followed by phase IV hypertension and reflex bradycardia, the characteristic overshoot, produced by restored
venous return and cardiac output with continued sympathetic stimulation of the heart and arterial vasoconstriction (26; 36). Baroreceptor mediated tachycardia in phase II and bradycardia in phase IV are used as indices of cardiovagal integrity (21). BP recovery in phase II and hypertensive response in phase IV are often used as indices of baroreceptor mediated sympathetic integrity (10). However, both early and late phase II are affected by changes in blood volume (7) and therefore blood pressure changes cannot be regarded as entirely determined by autonomic responses.

Implicit in the interpretation of the maneuver is that a given increase in intrapleural pressure produces a well-defined decrease in venous return. Also, provided cardiac function is normal, a given decrease in venous return produces a given decrease in cardiac output and blood pressure. The decrease in venous return and cardiac output thus generate the early phase II decrease in blood pressure, which is the actual stimulus to the baroreflex resulting in the heart rate and BP response.

We hypothesized that even a standardized Valsalva maneuver with controlled expiratory pressure may produce a wide range of thoracic blood volume shifts; this would include large volume shifts producing decreased blood pressure during phase II that cannot be restored by autonomic compensation. Thus, the true vascular stimulus during normovolemia in the Valsalva maneuver, i.e. \( \downarrow \text{venous return} \rightarrow \downarrow \text{cardiac output} \rightarrow \downarrow \text{blood pressure} \), could be sufficiently decreased so that autonomic compensation fails to restore blood pressure in phase II.
Materials and Methods

Subjects and Experimental Outline

To test this hypothesis we studied 17 healthy volunteers aged 14-22 years (median=17 years, 7 male, 10 female). Volunteer subjects were recruited from among young people referred for innocent heart murmur. All subjects were evaluated with normal electrocardiograms and echocardiograms and had normal estimated resting cardiac outputs. Subjects with a history of syncope or orthostatic intolerance were specifically excluded. Only those found on cardiac exam to be free from heart disease were eligible to participate. There were no trained competitive athletes or bedridden subjects among subjects. Informed consent was obtained and all protocols were approved by the Committee for the Protection of Human Subjects (IRB) of New York Medical College.

We assessed changes in estimated thoracic blood volume by impedance plethysmography, along with changes in heart rate and blood pressure before and during the Valsalva maneuver, which was performed supine. The Valsalva response has been shown to be strongly posture-dependent (16; 28). We chose to perform the maneuver in the supine position to separate autonomic stimuli arising from orthostasis from stimuli due to the Valsalva maneuver.

Details of the Method

Tests began in a temperature controlled room after an overnight fast. After a 30 minute acclimatization period, tests were performed in the following order allowing at least 15 minutes for recovery in between: Supine blood flow and arterial resistance
measurement, Valsalva maneuver, upright blood flow (at 35°) and arterial resistance measurement. Supine and upright peripheral blood flow and resistance measurements were used to demonstrate that current vasoconstrictor responses are consistent with data obtained from healthy control subjects during previous protocols (32; 33).

**Peripheral Blood Flow Venous Pressure (P_v) and Arterial Resistance (supine and upright)**

We used venous occlusion strain gauge plethysmography to measure forearm and calf blood flow. Supine measurements were made at the beginning of experiments. Occlusion cuffs were placed around the upper and lower limbs approximately 10 cm above a strain gauge attached to a Whitney-type strain gauge plethysmograph (Hokanson, Inc). Blood flow was estimated while supine using rapid cuff inflation to a pressure below diastolic pressure (e.g. 40 mmHg) to prevent venous egress (12). Briefly inflating a smaller secondary cuff to supra-systolic blood pressure prevented ankle blood flow. Systolic and diastolic blood pressures of the arm and leg were determined by oscillometry. Arterial inflow in units of ml/(100ml tissue)/min was estimated as the rate of change of the rapid increase in limb cross sectional area. For normative purposes we had previously collected peripheral blood flow data from 42 control subjects spanning a number of prior research protocols. For purposes of this study, decreased supine calf blood flow was defined as less than 1.2ml/min/100mls of tissue, which was the smallest calf blood flow we have measured in control subjects. Increased supine calf blood flow was defined as greater than 3.6 ml/min/100mls of tissue, which was the largest calf blood flow we have measured in control subjects. Capacitance
vessel pressure (venous pressure, $P_v$) was also assessed in the steady state. After
strain gauge dimension returned to baseline following blood flow measurement, we
measured $P_v$ by gradually increasing the occlusion cuff pressure until an increase in
limb volume was just detected which closely approximates invasive measurements of
venous pressure (2). Peripheral resistance was calculated using the formula $\frac{(MAP-}
\frac{R_{estating \ Flow})}{R_{estating \ Flow}}$ where MAP is the mean arterial pressure calculated as
(systolic BP + 2*diastolic BP)/3.

**Heart Rate and Blood Pressure Monitoring**

Electrocardiogram strips were monitored continuously. Upper extremity blood pressure
was continuously monitored with an arterial tonometer (Colin Instruments, San Antonio
TX) placed on the right radial artery and recalibrated automatically every 5 minutes
against oscillometric BP. Leg blood pressure was measured intermittently by
oscillometry on the calf contralateral to the strain gauge. EKG, and pressure data were
interfaced to a personal computer through an A/D converter (DataQ Ind, Milwaukee,
Wi). All data were multiplexed with strain gauge and impedance information and were
effectively synchronized.

**Dye Dilution Measurement of Blood Volume, Cardiac Output and Total Peripheral
Resistance**

Indocyanine green dye dilution technique (1) employing a noninvasive
spectrophotometric finger photosensor (DDG, Nihon-Kohden Inc) was used to estimate
blood volume, cardiac output, and total peripheral resistance. This technique has been
verified during clinical studies (13; 14; 14). First pass kinetics were used to obtain cardiac output by Stewart’s classical area under the curve method (31). Cardiac index was obtained by dividing the cardiac output by the patient surface area computed from the formula of Dubois and Dubois (4): 

$$\text{BSA}(\text{M}^2) = \text{weight}^{0.425} \times \text{height}^{0.725} \times 0.00718,$$

with weight in kg and height in cm. The dye decay curve is a monoexponential representing clearance by the liver. Once the hematocrit is measured, we extrapolated the dye decay curve to the time of dye injection (t=0) yielding estimated blood volume. Total peripheral resistance was estimated by dividing the mean arterial blood pressure measured while supine in the right arm by the cardiac index.

**Impedance Plethysmography (IPG) to Measure Changes in Thoracic Blood Volume**

IPG has been used to quantify relative regional body fluid volumes (22; 27) but cannot quantify absolute total blood volumes with accuracy (hence the green dye measurements). Relations between impedance and changes in fluid compartment volumes and transient blood flows have been quantitated during orthostasis (3; 5). A Tetrapolar High Resolution Impedance Monitor (THRIM) digital impedance plethysmograph (UFI, Inc) was used to measure volume shifts in the thoracic segment. Disposable EKG electrodes were attached to the foot of each subject's left leg, on the same side of the body at the lower rib cage near the xyphoid process, at the left shoulder, and on the left arm at the back of the hand. The IPG introduces a high frequency (50 kHz), low amperage (0.1 mA RMS) constant current signal between the foot and hand electrodes. Electrical resistance values were measured using the
shoulder and rib electrode as sampling electrodes. Anatomic features were selected as the most appropriate locations for comparing changes within and across subjects. This combination of electrodes gives highly repeatable regional volume shifts and has been tested in a wide range of experiments by our group (19; 20; 35). The distance between the sampling electrodes (L) was measured.

We estimated the change in blood volume during the Valsalva maneuver from the formula:

\[ \Delta \text{volume} = \rho \left( \frac{L}{R_0} \right)^2 \cdot \Delta R \]  

(8).

Where \( \rho \) is electrical conductivity of blood which we estimated as 53.2*exp(Hct*.022) (Hct=hematocrit, or packed cell volume which we measured) given by Geddes and Sadler (9), \( R_0 \) is the baseline resistance and \( \Delta R \) is the time dependent change in resistance during the maneuver. Volume change was calculated from the maximum decrease in \( \Delta R \) during the maneuver using the value of \( R_0 \) immediately preceding initiation of exhalation as the starting point for calculation as shown on figure 2. An initial transient \( \Delta R \) was relatively constant until release (see figure 3). However, sometimes there was a systematic change as shown in figure 2. The average value of \( R_0 \) was used and \( \rho \) was regarded as constant during the course of the maneuver. IPG measurements allow us to track acute blood volume changes in the thoracic compartment during the Valsalva maneuver.

**Quantitative Valsalva Maneuver**

The quantitative Valsalva maneuver was performed with the subject supine by exhaling with an open glottis into a mouthpiece connected to the mercury column of a sphygmomanometer with an air leak. A 35-40 mmHg pressure was maintained for 15
seconds. Pressure was released taking care to prevent deep breathing. Two attempts with 10 minutes of intervening quiet breathing were made to secure an adequate Valsalva maneuver with sustained intraoral pressure. The first adequate exhalation was used for data acquisition. All subjects were able to produce the maneuver, although in one case the pressure was unsustained (see below). Blood pressure, electrocardiogram, heart rate, and thoracic impedance were recorded continuously throughout the maneuver. The BP response was quantified during straining and during the pressure overshoot. We used the maximum decline in BP to indicate the end of early stage II. Typically this occurred at approximately 7 seconds into exhalation. We used the point of subsequent maximum systolic blood pressure preceding release to indicate late stage II. Early blood pressure changes are independent of the sympathetic nervous system which requires at least some seconds to exert any effect (29; 34).

**Size Measurement**

The inlet circumference ($C_{in}$) of the thorax was measured by tape measure by encircling the chest under the axillae. The thoracic outlet circumference ($C_{out}$) was measured by encircling the waist at the level of the xyphoid process. The thoracic inlet and outlet cross-section areas were, respectively $\pi \cdot (C_{in}/2\pi)^2$ and $\pi \cdot (C_{out}/2\pi)^2$. We estimated thoracic volume as the average of inlet and outlet cross sections multiplied by L or

$$L \cdot 0.5 \cdot \left[ \pi \cdot (C_{in}/2\pi)^2 + \pi \cdot (C_{in}/2\pi)^2 \right] = L \cdot \frac{(C_{in})^2 + (C_{out})^2}{8\pi}.$$

**Orthostatic Challenge**
We used a low angle tilt test to produce well defined changes in peripheral resistance in response to orthostasis. An electrically driven tilt table (Cardiosystems 600, Dallas, Texas) with a footboard was used. After supine measurements were complete, the subjects were tilted to 35° for 15 minutes to obtain steady state measurements. Earlier work indicated that 35° upright tilt produces an adequate orthostatic response (33). $P_v$ and limb blood flows were remeasured at steady state and forearm and calf arterial resistances calculated.

**Statistics**

Tabular data concerning supine and upright blood flow, $P_v$, heart rate, blood pressure, and peripheral resistance were compared by one-way analysis of variance before and after the maneuvers. When significant interactions were demonstrated the ratio of $F$ values was converted to a $t$ distribution using Scheffe’s test and probabilities were thereafter determined. Correlations were obtained using the Spearman rank order correlation statistic. All tabular results are reported as mean $\pm$ standard error of the mean.
Results

All subjects were able to perform the supine quantitative Valsalva maneuver maintaining the expiratory pressure at 35-40 mmHg for at least 15 seconds. Results are shown in Tables 1, and in figures 2-5.

Hemodynamics and Size Measurements

Table 1 shows results concerning size and peripheral hemodynamic measurements in all subjects. Total blood volume, thoracic volume, cardiac index, total peripheral resistance and body surface area were similar for all subjects and did not relate to the magnitude of volume changes during Valsalva maneuver. Blood volume was also uncorrelated with changes in blood pressure during the Valsalva. While there is copious evidence that the Valsalva response relates strongly to blood volume (7; 16; 28), all our subjects had similar normal blood volumes. Resting heart rate, mean arterial pressure, and venous pressures were also similar and, except for heart rate, changed similarly with upright tilt to 35°. The leg MAP and P_v increased to a similar extent with tilt in all subjects. Peripheral arterial resistance increased in all subjects indicating postural vasoconstriction.

Impedance, Thoracic Blood Volume and Pressure Changes during the Valsalva Maneuver

Young subjects often have wide ranges of blood pressures and thoracic blood volumes. Thus, for example, for a subject whose resting systolic pressure is 95 mmHg (within normal range for a 14 year old) a decrease in blood pressure of 20 mmHg will be
relatively more important than for another subject whose blood pressure is 120 mmHg. Hence, for purposes of comparison we chose to examine changes in blood pressure as fractional changes in pressure and similarly changes in thoracic blood volume as fractional changes in blood volume. Figure 2 shows changes in impedance, blood volume and blood pressure for a representative subject. There is a decrease in thoracic blood volume exceeding 25% associated with a large change in blood pressure. Resting and orthostatic blood pressures, venous pressures, and arterial blood flows and resistances were completely normal.

Figure 3 compares several subjects with varying degrees of change in calculated thoracic blood volume and associated blood pressure changes. Two of the three subjects shown did not have complete blood pressure recovery during late phase II because thoracic blood volume and thus cardiac filling were insufficient and precluded recovery. Peripheral arterial resistances supine and during orthostatic testing were similar to results for healthy volunteers in prior experiments (32; 33). As shown in figure 4, early phase II systolic blood pressure is poorly correlated with supine and upright arm and leg arterial resistances. In all subjects the expected phase II tachycardia and phase IV bradycardia and pressure overshoot were present consistent with intact autonomic functioning. Changes in phase II BP parallel changes in thoracic blood volume in these subjects. The phase II fractional change in blood pressure for volunteer subjects was 0.20±.03. The fractional change in thoracic blood volume for volunteer subjects was 0.15±.02.
In order to demonstrate the relationship between blood pressure and intrathoracic filling during early phase II of the Valsalva maneuver we plotted the fraction BP as a function of fraction intrathoracic volume measured by impedance methods. This appears in figure 5. We used a linear fit, SBP= a_0*V, constrained to pass through the origin. The best fit ($r^2=.64$, $p<.0001$) is shown superimposed on the data. The graph suggests that there is a good relation between phase II volume and BP change during the Valsalva maneuver, which is similar from subject to subject once normalized.

In order to demonstrate that the decrease in systolic BP during phase II is related to systolic BP in late phase II we plotted fraction systolic BP during late phase II normalized to baseline BP against fraction BP during early phase II. As shown in figure 6 the best fit to all subject data is a straight line ($r^2=.67$, $p<.0001$) An exaggerated decrease in blood pressure during early phase II is related to a reduction in late phase II blood pressure.

We examined phase IV overshoot blood pressure. As shown in figure 7 there is no significant correlation between minimum phase II systolic blood pressure and phase IV overshoot ($r^2=.06$, $p=NS$). However, an overshoot is noted even when there is failure of pressure recovery (see figure 3).
Discussion

Our results demonstrate a quantitative relation between early and late blood pressure changes and the decrease in thoracic blood volume during phase II of the Valsalva maneuver. That decreased venous return produces decreased thoracic blood volume is not new. The new observation comprises the wide variation in venous return from subject to subject, sufficient to produce large decreases in blood pressure which cannot be compensated by sympathetic vasoconstriction. We demonstrated an uncompensated decrease in phase II blood pressure which is independent of baseline vasoconstrictive ability and unrelated to decreased blood volume but which can produce late phase II findings suggesting sympathetic impairment when none is present. Results suggest a variable redistribution of blood volume in individual subjects during the maneuver. The vascular as well as autonomic dependence of the Valsalva maneuver broadens its utility but complicates its analysis.

Pressure decrease during early Valsalva maneuver is Independent of Sympathetic Activation

Sympathetic activation takes time. Based on data from Tyden (34), Rowell estimated that a lag of 5-15 seconds occurs before vasoconstriction or venoconstriction takes place (25). During the early Valsalva maneuver, blood volume redistribution may therefore occur dependent on basal resistance and compliance properties. This coincides with early phase II. Cardiac activation may occur more rapidly, but exerts only a modest effect on pressure recovery, which instead depends on compensation for inadequate venous return. Indeed Smith et al (29) have carefully demonstrated a
similar, although somewhat shorter, delay in the onset of muscle sympathetic nerve activity following Valsalva straining. Baseline sympathetic tone could play a role, but there is no evidence among our subjects of any difference in baseline peripheral vascular resistance or phase IV variation. We propose, along with others (24), that during expiratory strain there is a rapid decrease in venous return detected here as an increase in thoracic impedance.

The decrease in blood pressure during phase II depends on the decrease in thoracic filling, which varies from subject to subject (figures 3-5). Thoracic filling depends on blood volume, on the time dependent changes of venous resistance and venous pressure in regional circulations, and on right atrial pressure. Intrapleural pressure is very similar to intraoral pressure (6). Right atrial pressure appears to change in a deterministic way with increasing intrapleural pressure, although the increase in atrial pressure is only about 70% of the increase in intrapleural pressure (15). Therefore, in subjects of similar total blood volume, thoracic filling depends on venous properties and provides insight into venous mechanisms. The data suggest that large intersubject variations in venous resistance, peripheral venous pressure, or both determine intersubject variation in venous return during early phase II. Given that resting arterial constriction and venoconstriction (inferred from peripheral venous capacity) are similar in all subjects, the data may indicate that individual differences relate to differences in venous mechanical properties. Prior work has supported the ability to generate well-defined venous return curves from graded use of the quantitative Valsalva maneuver (18), while other investigators have shown that such graded expiratory pressures
produce graded changes in splanchnic venous pooling. (17). In this regard figure 4 resembles a ventricular function curve, albeit one obtained from a number of subjects, reflecting the ability to generate blood pressure as a function of thoracic blood volume. Anecdotally, large and dramatic venous function variations have occasionally been reported during the Valsalva maneuver up to and including complete collapse of large collecting veins with rapid onset of syncope despite ongoing tachycardia (30).

**Pressure recovery during phase II of the maneuver may not occur despite adequacy of the sympathetic nervous system**

Peripheral venous properties may so severely limit thoracic venous return during early phase II, that no degree of sympathetic vasoconstriction or sympathetic cardiac activation can restore blood pressure. Similar uncompensated phase II hypotension can be contrived by limiting blood volume. Thus, Fritsch-Yelle et al (7) could increase or decrease end phase-II blood pressure by infusing saline or by infusing intravenous furosemide. Similar effects are seen with a change in posture, particularly if combined with relative hypovolemia (16; 28; 28). But our subjects were normovolemic and supine.

**Limitations**

We did not measure vasoconstriction during the Valsalva maneuver. This was not feasible using occlusion plethysmography but may be addressed in the future by using peripheral impedance flow methods currently under consideration. We have used resting and orthostatic peripheral vasoconstriction as surrogates of sympathetic
vasoconstrictive adequacy. Clearly selective regional sympathetic abnormalities can also exist and could alter our conclusions.

Alternatively, a direct measure of sympathetic activity such as muscle sympathetic nerve activity (MSNA) could have enhanced our ability to state that volume changes per se, in the presence of intact sympathetic vasoconstriction, account for our findings. However, such instrumentation is often regarded as problematic in subjects of the age range used in our studies and was therefore not pursued. Nevertheless, in the absence of direct measures of sympathetic vasoconstrictor tone such as MSNA one cannot with certainty assert that vasoconstrictor responses and therefore autonomic vasomotor function was normal during the Valsalva maneuver or even during non-Valsalva intervals.

Posture: We chose to examine subjects in the supine position because we wanted to separate contributions from orthostasis from contributions due only to the Valsalva maneuver. Clearly, the Valsalva maneuver response has been shown to relate strongly to posture (16; 28).

Effects of Lung Volume: Thoracic blood volume is affected by lung volume. The amount of initial inspiration and leakage flow during the Valsalva maneuver can affect impedance measurements. Also inspiratory volume can vary among subjects. Thus, it would be best to include measurements of tidal volume for subjects; this could not be accomplished using current experimental design. However, intrapleural pressure was
controlled across subjects, and is a critical external stimulus for blood and impedance changes.

Age limitations to generalizability may exist. Young adults and adolescents may not perfectly represent findings for mature adults. However, cardiovascular structure and function is essentially mature by puberty and therefore results can be regarded as at least qualitatively similar to older age groups. Moreover, younger subjects generally have the advantage of absence of confounding illness such as heart disease, renal disease, hypertension, and diabetes. Also, the threshold for abnormal tachycardia in adolescents may be higher.
Acknowledgements

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Disclosures

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Reference List


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* = p<.05 compared to supine
Figure 1 A representative Valsalva maneuver is shown. The top panel shows arterial blood pressure while the lower panel shows heart rate. After inspiration, the subject blows against a resistance of approximately 40 mmHg. This initially produces a brief increase in BP in phase I, a fall in BP which is later restored in phase II, a brief decrease in BP with inspiration in phase III, and an overshoot in phase IV. Hypotension in phase II is associated with a tachycardia while hypertension in phase IV is associated with bradycardia. See text for further details.
Figure 2 shows that impedance increases and blood volume decreases precede the decrease in blood pressure (related to blood volume transit times). Blood pressure then decreases steadily through early phase II. Impedance decreases and calculated thoracic volume from the impedance measurement increases throughout the latter portions of phase II suggesting venoconstriction. Blood pressure never regains resting levels, however, despite autonomically mediated tachycardia and intact orthostatic vasoconstriction in this subject. Phase IV hypertension and bradycardia are evident.
Figure 3 shows fractional thoracic volume (upper panel) and fractional blood pressure (lower panel) during the Valsalva maneuver. Minimum phase II blood pressure is respectively mildly, moderately, and markedly decreased in association with mildly, moderately, and markedly decreased thoracic blood volume. Blood pressure recovery in late phase II is complete for the mild subject, nearly complete for the moderate subject (a normal volunteer) and incomplete for the marked subject.
Figure 4 shows the relationship between the minimum fractional systolic blood pressure during early phase II of the Valsalva maneuver and peripheral arterial resistance in arm and leg while supine (upper panel) and upright (lower panel). Blood pressure is expressed as the fraction of baseline systolic blood pressure preceding the maneuver. Resistances are poorly correlated to early phase II pressure.
Figure 5 depicts the relationship between the fraction change (decrease) in blood pressure and the fraction (decrease) intrathoracic blood volume calculated from impedance plethysmography. A linear fit to these data is constrained to pass through the origin since a zero volume change produces no pressure decrement.
Figure 6 depicts the relationship between the maximum fractional systolic blood pressure at end phase II of the Valsalva maneuver (i.e. recovery) and the minimum systolic blood pressure during early phase II. Blood pressure is expressed as the fraction of baseline systolic blood pressure preceding the maneuver. The least squares linear fit to all data is shown. Lower early phase II blood pressure correlates to lower late phase II blood pressure.
Figure 7 depicts the relationship between the minimum fractional systolic blood pressure at during early phase II and the maximum systolic blood pressure during phase IV. Blood pressure is expressed as the fraction of baseline systolic blood pressure preceding the maneuver. The least squares linear fit to all data is shown. There is no significant correlation.