The effect of breathing manner on inferior vena caval diameter†

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Aims

Although the inspiratory ‘collapse’ of the inferior vena cava (IVC) has been used to signify normal central venous pressure, the effect of the manner of breathing IVC size is incompletely understood. As intra-abdominal pressure rises during descent of the diaphragm, we hypothesized that inspiration through diaphragmatic excursion may have a compressive effect on the IVC.

Methods and results

We measured minimal and maximal intrahepatic IVC diameter on echocardiography and popliteal venous return by spectral Doppler during isovolemic inspiratory efforts in 19 healthy non-obese volunteers who were instructed to inhale using either diaphragmatic or chest wall expansion. During inspiration, the maximal diaphragmatic excursion and popliteal vein flow were compared between breathing methods. The IVC ‘collapsibility index,’ IVCCI, was calculated as (IVCmax2IVCmin)/IVCmax. The difference in diaphragmatic excursion between diaphragmatic and chest wall breaths in each subject was correlated with the corresponding change in IVCCI. Diaphragmatic breathing resulted in more diaphragmatic excursion than chest wall breathing (median 3.4 cm, range 1.7–5.8 vs. 2.2 cm, range 1.0–5.2, P = 0.0003), and was universally associated with decreased popliteal venous return (19/19 vs. 9/19 subjects, P < 0.004). The difference in diaphragmatic excursion correlated with the difference in IVCCI (Spearman’s rho = 0.53, P = 0.024).

Conclusion

During inspiration of equivalent tidal volumes, the reduction in IVC diameter and lower extremity venous return was related to diaphragmatic excursion, suggesting that the IVC may be compressed through descent of the diaphragm.

Keywords

Inferior vena cava • Echocardiography • Central venous pressures

Introduction

Non-invasive or physical examination estimates of central venous pressure can have immediate bedside value in the assessment of unexplained hypotension and dyspnoea and in guiding fluid management. In echocardiography, a 50% or greater reduction in the diameter of the inferior vena cava (IVC) during inspiration has been considered a sign of normal central venous pressure, although studies have varied in methodology and thresholds.1–3 A blunted or absent response has been considered indicative of elevated right atrial pressures and has been associated with a worse prognosis in patients with congestive heart failure.4 Conventionally, caval diameter reduction during inspiration is felt to be due to falling intra-thoracic pressures and vessel ‘collapse’ due to the increase in the difference in transmural pressure. However, the IVC traverses the abdominal compartment and is therefore under the influences of hepatic weight, intra-abdominal pressure,6,7 and venous return of pooled splanchnic and lower extremity blood.8,9 During inspiration, not only is there a fall in intra-thoracic pressure, but also a simultaneous rise in intra-abdominal pressure due to descent of the diaphragm occurs. Few data exist on determinants of the ultrasonic response of the IVC diameter even under normal physiologic breathing conditions. Therefore, we sought to evaluate the response of the IVC diameter to different types of inspiratory efforts in normal subjects while monitoring the effect upon lower extremity venous return.

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Methods

Study subjects

Nineteen healthy non-obese subjects without symptoms or history of cardiovascular disease volunteered for this study. The study was approved by the Scripps Institutional Review Board for Scripps Mercy Hospital, San Diego.

Examination protocol

Subjects were first instructed to perform non-laboured, comfortable breathing with a 1–2 s inspiratory duration using two types of breathing methods. ‘Diaphragmatic’ breathing was explained as inhaling by expanding the abdomen outwards without raising the chest. ‘Chest wall’ breathing was explained as inhaling by expanding the chest with inward motion of the abdomen. Exhalation was not instructed, other than to return to a baseline state in 1–2 s without holding the breath. A comfortable tidal volume, reproducible with either type of breathing, was determined for each subject during subject instruction and practice. To ensure reproducible tidal volumes, a Wright’s respirometer (nSpire Health Inc., Longmont, CO, USA) was used during subject training and was directly observed during subject breathing at the time of data acquisition. The study coordinator observed and coached the subject in breathing to ensure consistent inspiratory efforts at a non-tachypneic, regular pace and approximately equal tidal volume.

After successful demonstration of the ability to perform comfortable chest and diaphragmatic breathing in the supine position, subjects were then instructed to repeat an assigned type of breathing while simultaneous ultrasonic measurements of the IVC diameter and popliteal venous return were recorded. While monitoring patient breathing, the study coordinator signalled when to record data from a typical inspiration to both the cardiac and vascular sonographers. Data were recorded during the inspiratory phase of both types of breathing on each subject while in the supine position, as follows (Figure 1). The IVC image was recorded in a standard echocardiographic subcostal longitudinal view using a conventional echocardiograph and 3 MHz cardiac transducer (iE33, Philips Healthcare, Andover, MA, USA). For measurement of IVC collapse, a fixed point on the vessel, 2–3 cm from the right atrium at the junction of the most proximal anterior hepatic vein, was used to measure a maximum IVC transverse distance (IVCmax) during end expiration and at its minimum during inspiration (IVCmin). The IVC ‘collapsibility’ index, IVCCI, was defined conventionally as the ratio of (IVCmax – IVCmin) to IVCmax, and ranged from 0 (non-collapsible) to 1 (fully collapsible). On the same image, the most cephalad point of the liver contacting the anterior wall of the IVC was identified, and diaphragmatic excursion was measured as the maximal caudal distance travelled by this point during inspiration. The difference in diaphragmatic excursion between chest wall and diaphragmatic inspirations was recorded in each individual subject for correlation with the change in IVCCI. Simultaneous with echo recordings, a 7 MHz linear vascular probe attached to a second ultrasound machine (iE22, Philips Healthcare, Andover, MA, USA) was placed in the patient’s right popliteal fossa to visualize the popliteal vein. Spectral Doppler was used to measure popliteal venous flow in the direction of the heart. An increase or decrease in flow velocity during the inspiratory period was determined on all subjects during both types of breathing. All interpretations and measurements were made on video-looped digital recordings by an echocardiographer who was blinded to the mode of respiration.

Figure 1  Composite figure of example subject data. The IVC diameter reduction is larger with diaphragmatic inspiration, right panels (IVCCI = 1.0), than with chest wall inspiration, left panels (IVCCI = 0.2). Corresponding popliteal vein flow is arrested with diaphragmatic inspiration (blue arrow marks inspiration) but slightly increases with chest wall breathing.

Statistical analysis

Descriptive data for the 19 subjects are reported as means with SDs. Paired sample Wilcoxon–Mann–Whitney tests were used to examine differences in IVCCI and diaphragmatic excursion between chest and diaphragmatic breathing. McNemar’s test of paired proportions was used to test whether a significant difference exists between the proportion of subjects with arrest of venous flow during chest when compared with diaphragmatic breathing and the proportion of subjects with IVCCI < 0.5 during chest when compared with diaphragmatic breathing. Spearman’s correlation was used to assess the relationship between changes in IVCCI and changes in diaphragmatic excursion between chest and diaphragmatic breathing within each patient.

Results

Nineteen subjects, 12 men and 7 women, aged 35 ± 12 years (range 25–60) with a body mass index of 25 ± 3.1 (range 19–29), were studied. The subject tidal volume was 910 ± 318 ccs (range 450–1850). In the setting of equivalent tidal volumes for both types of inspiration per subject, diaphragmatic breathing resulted in significantly more diaphragmatic excursion than chest wall breathing (median 3.4 cm, range 1.5–5.8 vs. 2.2 cm, range 1.0–5.2, P < 0.01) and a trend for a larger IVCCI (median 0.80, range 0.48–1.00 vs. 0.57, range 0.13–1.00, P = 0.053) (Figure 2). Using non-collapsibility defined by an IVCCI < 0.5 as the threshold for abnormally elevated central venous pressures, 8/19 subjects erroneously reached this threshold with chest breathing while only 1/19 subjects reached this threshold with diaphragmatic breathing (P = 0.023). Popliteal vein flow velocity decreased in 19/19 subjects with diaphragmatic inspiration. Conversely, during chest wall inspiration, a decrease in popliteal blood flow velocity was observed in only 9 patients, whereas in the remaining 10 an increase was actually recorded (P < 0.004). When diaphragmatic and chest wall breathing were compared in each patient, the difference in diaphragmatic excursion correlated with the difference in IVCCI (Spearman’s rho = 0.53, P = 0.024) (see Figure 3).
Discussion

This study found that reduction of the IVC diameter and lower extremity venous return during different types of quiet inspiratory efforts of the same tidal volume was related to the difference in diaphragmatic excursion. These findings suggest that during inspiration the IVC, in addition to responding to falling intra-thoracic pressure, may also be compressed with diaphragmatic descent and have implications regarding the use of IVC diameters to estimate the central venous pressure without knowing the manner of breathing, intra-abdominal pressure, or magnitude of diaphragmatic excursion.

Well-accepted echocardiography texts and seminal publications have described the inspiratory reduction in IVC diameter as ‘collapse,’ which implies a mechanism of an inspiratory fall in luminal pressure and ignores the confounding effects of the breathing method, intra-abdominal pressure, or hepatic venous return. The ‘collapsibility index,’ IVCCI, has been used in multiple studies as a measure of this phenomenon without mention of an alternate mechanism of vessel compression as a limitation to the stated findings. Intra-abdominal pressure averages 6.5 mmHg (range 0.2–16.2 mmHg) in hospitalized patients, increases during inspiration when it can exceed the central venous pressure, and is notably higher in the obese. In the current study, the fact that popliteal venous flow was arrested in all cases during inspiration using diaphragmatic breathing is evidence that the intra-abdominal compartment pressure rose above the pressure within the distal deep venous system of the lower extremity (Figure 1) and is the predominant physiology during normal breathing. Conversely, an increase in the lower extremity venous flow velocity was only seen in some patients, all of whom were performing chest wall inspirations at the time. The heterogeneity in the flow response during chest breathing may be due to contamination of the chest breath with diaphragmatic breathing and variability in each subject’s intra-abdominal pressures.

The possibility that the IVC can be compressed during inspiration has been suggested by prior animal, human, and radiographic studies. Physiologic experiments in 12 open-chested canines using phrenic nerve stimulation have demonstrated a decrease in IVC flow and an increase in femoral vein pressures when intra-abdominal pressure exceeded IVC pressure during inspiration. In a study of 10 normal human volunteers, the inspiratory increase in the abdominal pressure measured with a gastric balloon was associated with a fall in femoral venous return. The phenomenon was more marked with ‘pure diaphragmatic’ breathing when compared with ‘rib-cage’ breathing. Clinical observations in patients have similarly related raised intra-abdominal pressure to narrowing of the IVC and reduced lower extremity venous return in those with ascites and during abdominal insufflation at laparoscopic surgery.

The conceptual change from IVC ‘collapse’ due solely to falling intra-thoracic pressures to the possibility of a component of ‘compression’ under rising intra-abdominal pressures has multiple clinical implications particularly in the supine, obese, and critically-ill patient subsets in whom the knowledge of the central venous pressure is important, yet is difficult to obtain non-invasively. Prior echocardiographic methods that employed various IVC parameters to estimate central venous pressure have demonstrated overall accuracies of 66–90% and r values of 0.6–0.9 compared with catheter-based measurements. However, these studies utilized non-standardized, variable breathing methodologies and were limited to stable, compliant patients. Mode of ventilation, hyperinflation, sedation and neuromuscular blockade, abdominal adiposity, bowel distension, patient positioning, and pleuritic pain could potentially influence the relative contributions

![Figure 2](https://example.com/figure2.png)

**Figure 2** Graph: IVCCI by breathing manner comparison of paired IVCCI data during diaphragmatic (median 0.80, range 0.48–1.00) and chest wall breathing (median 0.57, range 0.13–1.00) for each of the 19 subjects (P = 0.053, non-significant).

![Figure 3](https://example.com/figure3.png)

**Figure 3** Graph: difference in IVCCI vs. difference in diaphragmatic excursion. The difference in IVCCI is plotted on the y-axis as a function of the difference in diaphragmatic excursion in centimetres (diaphragmatic breathing minus chest wall breathing) for each of the 19 subjects. Diaphragmatic breathing resulted in more diaphragmatic excursion and more IVCCI. (Spearman’s rho = 0.53, P = 0.024)
of diaphragmatic and chest wall expansion and intra-abdominal pressures to breathing, thus confounding the IVCCI. It is possible that in clinical scenarios in which intra-abdominal pressures are elevated, the IVC will be more easily compressed to a smaller size, artificially suggesting lower central venous pressures. As evidence to the potential clinical impact of this confounding mechanism, in the current study, 8 of 19 normal subjects could be misclassified as having elevated central venous pressures while chest breathing when compared with diaphragmatic breathing of the same approximate tidal volume. The actual magnitude of this effect in clinical medicine will need to be defined in various patient subgroups. Nonetheless, our data support the physiology that the IVC diameter is reduced during inspiration due to changes in overall vessel wall transmural pressure determined not only by transmission of falling intrathoracic pressures causing ‘collapse,’ but also by rising intra-abdominal pressures causing ‘compression.’

The current study has limitations primarily related to the attempted learning and isolated performance of two different breathing patterns. It should be noted that the two types of inspiratory efforts, although physiologic and encountered in clinical medicine, were primarily employed in this study as simple methods to vary the amount of diaphragmatic descent during isovolemic breaths in each individual subject. In normal physiology, diaphragmatic breathing is used at rest for small tidal volumes and the chest wall mechanism is a reserve. We did note a significant individual overlap in breathing methods, as have other investigators, as manifest by the presence of diaphragmatic excursion when subjects were attempting to breathe only with their chest wall. This effect may have been more prominent in larger tidal volumes or poorly trained subjects. However, this study accounted for ambiguous breathing by investigating the relationship between the amount of diaphragmatic excursion and IVC collapse within each subject’s different breathing attempts. As expected, more profound IVC effects were noted with larger net differences in diaphragmatic excursion (See Figure 3). However, the type and method of inspiration has been variable in the literature, including quick ‘sniffs,’ which likely represent forceful diaphragmatic contractions, to slow or mechanically ventilated breaths, which likely recruit the chest wall. Although the mean inspiratory force was not measured, we took efforts to regulate subject inhalation to avoid the formation of a vascular waterfall at the proximal junction of the right atrium due to rapid local pressure changes and specifically measured the IVC diameter a distance away from this junction. Although the specific location along the IVC used for measurement is not standardized in the literature, the site in which the IVC diameter is maximally reduced may have potential significance as it may reflect an interaction of proximal forces such as hepatic venous filling and transmission of intrathoracic suction with the more distal compressive forces of abdominal pressure.

The use of ultrasound at the bedside is becoming more widespread as a method to augment patient examination and has value in the assessment of central venous pressures, particularly in those patients in whom jugular venous assessment is difficult, such as the obese, intubated, or critically ill. This study proposes an alternate, co existing mechanism for diameter reduction of the IVC during inspiration that is dependent upon the amount of diaphragmatic excursion and intra-abdominal pressure and provides evidence for a larger, more complex mechanism of IVC behaviour during respiration than the one currently held. The observations of this investigation do not invalidate the current, established IVC-based methods of central venous pressure estimation when applied to stable patients in the echo laboratory, but may account for some of the imprecision encountered in untested clinical populations.

References

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