How to Utilize Respiratory Manouvers in Echocardiography
Practical Applications in Daily Practice

Karthik Ananthasubramaniam, MD FACC FASE FASNC
Associate Professor of Medicine
Wayne State University
Director, Echocardiography and Nuclear Cardiology/PET Laboratory
Henry Ford Hospital, Detroit MI

Respiration: Effects
Physiologic effects of respiration are secondary to
1. Variations in intra-thoracic and intra-abdominal pressure
2. Changes in systemic and pulmonary venous return
3. Intra pericardial pressure
4. Pericardial constraint
5. Ventricular interdependence

Anatomic effects of respiration are due to
1. Inflation of lungs and interference
2. Drop out of lateral shadows
3. Cardiac translation
4. Descent of the diaphragm

Disclosures
Research grant support
American Society of Echocardiography
Astellas Pharma Global Development, Inc
CV Therapeutics
GE Healthcare
Molecular Insight Pharmaceuticals

Speakers Bureau/Honoraria: Astellas Pharma Global Development, Inc
Consultant: Lantheus Medical Imaging

No conflicts of interest for this talk

Respiration and Image Quality
With inspiration:
1. Increase in AP diameter
2. Lung inflation
3. Rotation and posterior motion of heart
4. Subxiphoid imaging is an exception; heart images clearer with inspiratory diaphragm descent

Less heart available for imaging

With expiration:
1. Lung deflation (particularly in steep left lateral position with left arm up)

Less heart available for imaging

Objectives
Outline effects of respiration on heart
Outline how respiration can affect 2d, doppler imaging
Tips for better acquisition
Understand role of Valsalva manoeuver in echo
Use of respiratory in different cardiac conditions
Physiology of Respiration

- **Expiration**
  - Flow: Pulmonary vein
  - Intrathoracic pressure

- **Inspiration**
  - Flow: Pulmonary vein
  - Intrathoracic pressure

Respiration and Doppler of Veins

- **Hepatic vein doppler**
  - Inspiratory increase of S and D
  - SVC flow mimics HV doppler changes

- **Pulmonary vein doppler**
  - Minimal resp changes in S
  - but d and VTid increase

Respiration and Echo Measurements

- Inspiratory decrease in LV end diastolic dimensions as measured by M-mode
  - This may sometimes explain discrepancies between centerline PSAX measurements and those obtained by M-mode
  - Possible reasons:
    1. Heart moves medially during inspiration = Tangential M-mode cut
    2. Decrease in LV volumes in inspiration (preload reduction and increase afterload or impedance to LV emptying)

  How to measure: suspended quiet respiration make sure to instruct patients to avoid Valsalva during held expiration as it degrades image quality

Respiration and Doppler of AV and Systemic Valves

- **Mitr**
  - Transmirt v. varies by <10%, increasing with exp.

- **Tricuspid**
  - Varies by <20%, increasing with insp.

- **Aortic**
  - Pulm

Respiration And Doppler Issues

- Fixed doppler sampling errors can occur during respiration due to change in heart position and diaphragmatic traction
  - Parallel positioning and avoidance of angle of incidence issues is key.
  - Less than 10% error is most preferable given the quadratic relationship of pressure and velocity

- End expiration apnea most preferred for doppler sampling
  - Similar sampling issues affect tissue doppler
Respiration and Tissue Doppler

During normal respiration
During end expiratory apnea
Sample volume can move with respiration. Best obtained with held expiration

Respiration and IVC

Minimal size observed in end inspiration
Influenced by patient position:
Largest in right lateral position
Intermediate in supine position
Smallest in left lateral position
Normal ranges: 1.2 cm (upper normal range ASE guidelines 1.7 cm)
Can be large and minimally reactive in young patients, athletes can have large IVC and normal collapsibility index
Sniff test captured in real time and m-mode 3-5 loop rhythm capture will be helpful. M-mode cursor to be placed away from IVC-RA junction

Respiration and IVC

<table>
<thead>
<tr>
<th>IVC Diameter</th>
<th>% Collapse with Sniff</th>
<th>Estimated CVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1.2 cm</td>
<td>Full collapse</td>
<td>0 mmHg</td>
</tr>
<tr>
<td>1.2-1.7 cm</td>
<td>&gt;50% collapse</td>
<td>1-5 mmHg</td>
</tr>
<tr>
<td>&gt;1.7 cm</td>
<td>&gt;50% collapse</td>
<td>6-10 mmHg</td>
</tr>
<tr>
<td>&gt;1.7 cm</td>
<td>&lt;50% collapse</td>
<td>11-15 mmHg</td>
</tr>
<tr>
<td>&gt;1.7 cm</td>
<td>0% collapse</td>
<td>&gt;16 mmHg</td>
</tr>
</tbody>
</table>

Dilated IVC in vented patients does not mean elevated RAP. Collapsed IVC (>50%) indicates dehydration in vented patients.

Specificity for predicting RAP increases when IVC size is measured at end expiration by M-mode AND at end diastole

Respiration , IVC and Echo RAP: ? Time for Reappraisal

Brennan et al: JASE 2007
RAP measured from IVC in subcostal view and compared to RHC. This study questions using tight cutoffs of 5 mm to define RAP based on IVC
Overall IVC size 2cm served as the most optimal cutoff for RAP: > or < 10 (sens 73% spec 85%)
IVC collapsibility index of 40% was the best predictor for response (sens 74% spec 84%)

Respiration and Svc

Normal: Systolic forward flow = RA relaxation during RV contraction
Diastolic forward wave = rapid RV filling wave
Vary with respiration with both waves higher during inspiration than expiration

A New Classification for RAP

(1) High collapsibility with a small or normal-sized IVC; RAP is very likely low (<5 mm Hg).
(2) High collapsibility with a large IVC or normal collapsibility with a small/normalsized IVC; RAP is probably between 0 and 10 mm Hg
(3) Normal collapsibility with large IVC; RAP is 10 to 15 mm Hg
(4) Low collapsibility with a large IVC; RAP is clearly high (10-20 mm Hg)
(5) RAP in patients with low collapsibility and a normal-sized or small IVC should be interpreted as indeterminate.

Brennan et al: JASE 2007
**Mechanism of Flow Changes in SVC**

Possible clue to Pul HTN in COPD

SVC is an intra-thoracic structure: Interrogation is from right supraclavicular fossa with doppler

- Changes in RA pressure following change in pleural pressure cause SVC flow variations

Expiratory systolic flow in SVC is a measure of RA flow reserve

- In Pul HTN: RV filling is restricted—RA pressure is elevated and—— SVC forward variation is blunted

Hence SVC interrogation would be a useful adjunct in patients with significant lung disease

---

**Valsalva in Diastolic Function**

a. Need to instruct patients correct how to do it. To avoid forceful deep inspiration prior to the forced expiration.

b. Forced expiration against closed nose and mouth.

c. Useful to clarify pseudonormal mitral inflow patterns.

d. In normal patients if the E wave drops by 20 cm/sec that is a good effort useful to interpretation. E/A ratio remains above 1 despite the change in patients with normal diastolic function.

e. A E wave drop > 50 cm/sec is highly specific (100% spec) for elevated filling pressures accompanied by reversal to impaired relaxation pattern to confirm pseudonormal pattern although lower levels may also indicate the same diagnosis.

---

**SVC Doppler Index and Pul HTN**

![SVC Doppler Index and Pul HTN](image)

- **With PH**: EXP/INS ratio = 0.5
- **Without PH**: EXP/INS ratio = 0.35

---

**The Valsalva Manouever: Its Effects on Intracardiac Volumes**

![The Valsalva Manouever: Its Effects on Intracardiac Volumes](image)

---

**The Valsalva Manouever**

- **Phase 1** start strain: increased BP, transient increase venous return
- **Phase 2** strain maintained: decrease BP, pulse pressure and sinus tachycardia
- **Phase 3** strain release: progressive fall in BP
- **Phase 4** post release phase: overshoot of BP and reflex bradycardia

---

**Echocardiographic Classification of Diastolic Dysfunction**

![Echocardiographic Classification of Diastolic Dysfunction](image)
Correlations between LVEDP and the baseline E/A ratio (A), the change of the E/A ratio during Valsalva (B), and the change of the A wave during Valsalva (C).

Patients with a baseline E/A ratio of <1 (impaired relaxation pattern) despite substantially elevated LVEDP are represented by the grey circles.

Relaxation and Compliance

Tug of War

As diastolic dysfunction progresses, 2 scenarios can play out or co-exist:

- Impaired relaxation gives way to increased LV stiffness (decreased compliance)
- Severely impaired relaxation persist despite progressive worsening of compliance

Estimation of Filling Pressures in Patients with Depressed EF

Is It This or That?

Beat to beat variation of E/A with respiration
- E/A < 1 in Insp
- E/A > 1 in Exp
- DT shorter in Exp than Insp
- No effects of resp on A wave

This pattern associated with impaired tissue doppler
Could be a clue to diastolic dysfunction
**Respiration and PFO Detection**

TEE considered highly sensitive even more than TCD

<table>
<thead>
<tr>
<th>No shunt</th>
<th>Small shunt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate shunt</td>
<td>Large shunt</td>
</tr>
</tbody>
</table>

**Detection of PFO**

**SPARC Study Protocol**

- 2 rest injections
- 2 injections with cough
- 2 injections with Valsalva

**TEE PFO and Respiration**

Cough or Valsalva is widely employed
Both false negatives and false positives can occur related to the respiratory effort

- **False negative**: poor effort in combination with sedation or lack of understanding of how the Valsalva is to be done
  
  Solution: instruct pt prior to TEE. A good idea would be to perform a bubble study with Valsalva prior to sedation and intubation both as a practice run and as check to pt compliance and understanding of what is expected

- **False positive**: Spurious contrast misinterpreted as “shunt” small pulm AV malformation causing bubble appearance

**Detection of PFO**

**Methodologic Issues**

- Number of injections
- Use of maneuvers
- Femoral approach
- Use of color Doppler
- Harmonic imaging (TTE)
- Transmitral Doppler

**Spurious contrast: Snow Storm**

Has been described with cough and Valsalva maneuver
Has been described with TTE but most often TEE

Valsalva —— blood pooling in pulmonary veins —— rouleaux formation
Release of Valsalva ———— drops LA pressure ———— rush of pulmonary vein blood into LA

<table>
<thead>
<tr>
<th>Saline Bubbles</th>
<th>Spurious contrast</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bright and localized onset</td>
<td>Faint and weak intensity</td>
</tr>
<tr>
<td>Stronger echodensity</td>
<td>Weaker echodensity</td>
</tr>
<tr>
<td>Persistent</td>
<td>Evanescent</td>
</tr>
</tbody>
</table>

Spontaneous “spurious” contrast – likely due to the combination of rouleaux and cavitation as blood enters LA after Valsalva release
Spurious Contrast, SEC and Respiration

<table>
<thead>
<tr>
<th>SEC (Spontaneous echo contrast)</th>
<th>versus</th>
<th>Spurious contrast</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low flow states</td>
<td>No specific condition</td>
<td></td>
</tr>
<tr>
<td>Spontaneous occurrence</td>
<td>** Only with cough / valsala</td>
<td></td>
</tr>
<tr>
<td>Gain dependent</td>
<td>Gain dependent</td>
<td></td>
</tr>
</tbody>
</table>

Solution:
1. Check for exit of bubbles with Valsalva release from septum or pulmonary vein. Make sure bubble study protocol allows for adequate cine loop capture focusing on septum/RUPV in bicaval view with definitive capture post Valsalva release.
2. If septum normal with no clear PFO tunnel and no definite exit documented, but apparent bubble appearance in LA noted suspect Spurious Contrast.
3. Allow saline to clear and do a Valsalva /release cine without any right heart bubbles to look for spurious contrast.

Detection of PFO
Causes of False Positive TEE

- Transpulomonary microbubbles
- Extraneous background echoes/noise precipitated by Valsalva
- Pulmonary A-V malformation

Patients with Atrial Shunts Detected by Three Different Imaging Modalities

Kuhl et al. JACC 34:1823(1999)

Detection of PFO
Causes of False Negative TEE

- Inadequate visualization (uncommon)
- Insufficient number of injections
- Elevated LA pressures may prevent left-to-right passage of contrast
- IVC-directed flow along IAS prevents impingement of antecubital bubbles against IAS
- Improperly performed Valsalva maneuver

Common Provocations

- Valsalva

HOCM and Valsalva
Gradient in HCM with Valsalva

2D Echo Features
- Moderate to large pericardial effusion ≥ 2 cm or greater (loculated/circumferential)
- Swinging heart motion pattern (not always present)
- Right atrial collapse (late diastole or early systole) > 30% inversion
- Right ventricle collapse (early diastole) < 0.5 sec after MVO
- Left atrial collapse (late diastole or early systole)
- Left ventricle collapse (early diastole)
- Dilated inferior vena cava w/out collapse on inspiration
- Dilated hepatic veins > 1.1 cm
- Paradoxical septal motion of the IVS or inspiratory bounce (towards the LA)
- Inspiratory bounce of IAS (towards the LA)

Cardiac Tamponade-Respiratory Changes

Echo Changes in Cardiac Tamponade
1. RA and RV expand with leftward shift of ventricular and atrial septa
2. Reduced aortic and mitral valve opening time
3. Increased RV and reduced LV stroke volume
4. Decreased aortic and mitral flow velocities
5. Sharply increased tricuspid and pulmonary flow velocities
6. Decreased transmural Doppler E/A ratio and increased tricuspid E/A ratio.

20 normal patients
7 patients with tamponade

- Left Heart
  - Normal
    - IVRT, MV E vel, MV A vel < 10%
  - Tamponade
    - IVRT 1.85%
    - MV E vel 1.43%
    - MV A vel 1.25%
- Right Heart
  - Normal
    - TV E and A vel < 25%
  - Tamponade
    - TV E vel 1.85%
    - TV A vel 1.58%
Percentage change from first beat of expiration to first beat of inspiration.

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>IVRT (ms)</th>
<th>M1 (cm/s)</th>
<th>T1 (cm/s)</th>
<th>LVET (ms)</th>
<th>Ao (cm/s)</th>
<th>PA (cm/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal and post Tap (n=20)</td>
<td>2</td>
<td>-4</td>
<td>14</td>
<td>-3</td>
<td>-4</td>
<td>5</td>
</tr>
<tr>
<td>Tamponade (n=7)</td>
<td>85</td>
<td>-43</td>
<td>85</td>
<td>-21</td>
<td>-26</td>
<td>40</td>
</tr>
<tr>
<td>Eff – Variation (n=8)</td>
<td>32</td>
<td>-31</td>
<td>74</td>
<td>-9</td>
<td>-17</td>
<td>49</td>
</tr>
<tr>
<td>Eff – No Variation (n=7)</td>
<td>3</td>
<td>-5</td>
<td>32</td>
<td>-2</td>
<td>-4</td>
<td>6</td>
</tr>
</tbody>
</table>

Respiratory variation of pulmonary outflow > 30%

Respiratory variation of mitral inflow > 25 - 30%

Respiratory Variation

- Patients with Tamponade have marked variation in flow velocities.
- Some patients with large effusions without tamponade physiology have variation in flow velocities.
- These patients have increased pericardial pressures and an element of hemodynamic compromise
- Velocities return to normal following pericardiocentesis.

Differential Diagnosis of Respiratory Variation

- COPD
- Pericardial Constriction
- Severe Tricuspid Regurgitation
- RV infarction
Constrictive Pericarditis

- Restricted diastolic filling
- Normal systolic function

Thickened Pericardium acts as a shell that limits ventricular expansion.

Rapid filling occurs in the first 1/3 of diastole with little filling occurring in the last 2/3.

---

Diastolic septal bounce with inspiration

- Venous return increases leading to increased RV volume
- Total cardiac volume constrained by pericardium
- Interventricular dependence leads to septal shift

With permission, Dunitz 2000

---

Respiration, Constriction and AV Valve Flows

Respiration, Constriction and SVC Flow

- PW Doppler mitral inflow: High E velocity, E/A ratio > 2, short E wave deceleration time (E/e' < 160 ms); inspiration: decrease E velocity >25%, prolonged IVRT >25% expiration; opposite changes (Figure 6A)
- PW Doppler tricuspid inflow: E>A; inspiration: increased tricuspid E velocity >15%; characteristic phenomenon increased TR velocity (Figure 6B)
- PW Doppler recordings of hepatic vein flow: inspiration-minimally increased S and D; expiration: decreased diastolic flow/exaggerated atrial reversal waves (Figure 7)
- PW Doppler recordings of pulmonary vein flow: S/D ratio = 1; inspiration: decreased PV S and D waves, expiration: opposite changes
- SVC Doppler usually shows a diastolic dominant pattern, minimal respiratory variation as right atrial pressure is constantly elevated throughout the respiratory cycle by the thickened, constraining pericardium
- Inspiration: aortic velocity decreases (-14 ± 5%), pulmonary artery velocity increases (16 ± 4%)
- Dilated IVC with reduced inspiratory change in diameter
When respiration doesn’t work

Abdalla et al., Echocardiography, 2002

- Normal
  - IPPV will produce opposite respirophasic changes in velocity
  - Hypovolemia increased these changes further

- Hypovolemia increased these changes further

- Tamponade
  - MV opposite direction and attenuated

- Constriction
  - E vel increases with inspiration 18%
  - Pulmonary vein D wave increased 28% with inspiration

Intermittent Positive Pressure Ventilation

- Normal
  - IPPV* will produce opposite respirophasic changes in velocity
  - Hypovolemia increased these changes further

- Hypovolemia increased these changes further

Respiration and Constriction Vs COPD

- More dramatic change in forward velocities with Insp in COPD than CP

Respiration: Constriction Vs COPD

- Systolic forward velocity varies > 20 cm/sec with inspiration. Approximately a 35% or greater increase with inspiration due to greater effects of negative intra-thoracic pressure on RA pressure. On contrary RA pressure doesn’t change with CP so SVC flow variations are minimal.

Intermittent Positive Pressure Ventilation

- Normal
  - IPPV will produce opposite respirophasic changes in velocity
  - Hypovolemia increased these changes further

Restrictive Cardiomyopathy

- Fluid Challenge

Constrictive Pericarditis

- Fluid Challenge
<table>
<thead>
<tr>
<th>Condition</th>
<th>RCH</th>
<th>COP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral Valve</td>
<td>No respiratory variation of mitral flow, low velocity, short DT, diastolic regurgitation</td>
<td>Inspiration: increased mitral flow velocity, prolonged MITT, expiration: opposite changes, short DT, diastolic regurgitation</td>
</tr>
<tr>
<td>Pulmonary vein</td>
<td>Increased S/D ratio, prominent atrial septum, no respiratory variation, D wave</td>
<td>S/D ratio &lt; 1, inspiration: decreased pulmonary with S and D waves, expiration: opposite changes</td>
</tr>
<tr>
<td>Tricuspid valve</td>
<td>Mild regurgitation with increased outflow tract area, low velocity, E/A ratio 1:3, peak velocity, no significant inspiration change, short DT with inspiration, diastolic regurgitation</td>
<td>Inspiration: increased tricuspid outflow tract area, increased TR peak velocity, expiration: opposite changes, short DT, diastolic regurgitation</td>
</tr>
<tr>
<td>Hepatic veins</td>
<td>Increased S/D ratio, increased respiratory variations</td>
<td>Inspiration: minimally increased hepatic veins, expiration: increased diastolic flow, increased diastolic regurgitation</td>
</tr>
</tbody>
</table>

Thank You