Commonly Used Respiratory and Pharmacologic Interventions in the Echocardiography Laboratory

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(ECHOCARDIOGRAPHY, Volume 22, May 2005)

Valsalva, *Müller*, *postural*, *amyl nitrite*, *echocardiography*

Respiratory and pharmacologic interventions are used during auscultation of heart sounds. These same principles may be utilized in the echocardiography laboratory to aid diagnosis. Four physical maneuvers and one pharmacologic intervention performed in the echocardiography laboratory are described. A brief discussion of applications in specific clinical situations is then presented.

The Valsalva maneuver involves having the patient inspire deeply followed by a forceful exhalation against a closed glottis for approximately 10 seconds. Placing the hand on the patient's abdomen by the examiner provides a force for the patient to strain against and ensures that the abdominal muscles are tightening.[1] Some patients who have difficulty with this may find it best to insert their index finger in the mouth and attempt to blow out with lips sealed. Alternatively, the patient may blow into a manometer to maintain 40 mmHg or more.[2] The Valsalva maneuver involves the following four phases:

Phase 1—marked with beginning of strain causing a transient rise in systemic blood pressure.

Phase 2—marked by a decrease in blood pressure and pulse pressure with a reflex sinus tachycardia.

Phase 3—begins with the cessation of straining and exhibits an abrupt transient decrease in blood pressure.

Phase 4—exhibits overshoot of systemic arterial pressure and reflex bradycardia.[3]

The Müller maneuver, the converse of the Valsalva, is performed by forcibly inspiring while the nose is held closed and the mouth is sealed for about 10 seconds.[4] Because the maneuver exaggerates inspiratory effort, right-sided filling is augmented as are right-sided murmurs—especially tricuspid regurgitation and tricuspid stenosis.[1]

The next two maneuvers involve postural changes. The first is a change from standing to squatting which increases systemic venous return and systemic arterial pressure. The increase in systemic venous return causes the systolic murmurs of aortic and pulmonic stenosis to intensify and the diastolic murmurs of tricuspid and mitral stenosis to intensify.[4] The increase in systemic arterial pressure is valuable in augmenting the murmur created by aortic regurgitation.[3] Because it increases the size of the left ventricle, squatting also reduces the intensity of the systolic murmur created by hypertrophic obstructive cardiomyopathy and delays

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the mid-systolic click and late systolic murmur of mitral valve prolapse.[4]

The second postural change is passive leg-raising. This maneuver increases venous return; therefore, many of its effects are similar to squatting. The increase in stroke volume created by the augmented venous return increases the murmurs of aortic and pulmonic stenosis, mitral and tricuspid regurgitation, and ventricular septal defect.[4] Passive leg-raising also increases left ventricular end-diastolic volume; therefore, it has the same effect on hypertrophic obstructive cardiomyopathy and mitral valve prolapse as squatting.[4] The murmur of aortic regurgitation is not increased as leg-raising does not increase systemic pressure as squatting does.

Amyl nitrite, the volatile ester of nitrous acid, is administered by crushing an ampule in gauze near the patient's nose and then having the patient take three to four deep breaths over a 10–15-second period.[4] Within 15 seconds (effect lasts for about 30 seconds), the drug creates marked vasodilation with a rapid drop in systemic blood pressure and reflex tachycardia. This is followed by a reflex increase in cardiac output (primarily due to increased heart rate) and velocity of blood flow.

Amyl nitrite should only be given while with the patient in the supine position. It is contraindicated in severe aortic stenosis or severe angina pectoris. Patients with severe peripheral vascular disease or autonomic dysfunction may not respond, and patients with congestive heart failure will have a diminished response.[5] It should not be administered to pregnant women.[6] Presently, amyl nitrite is used infrequently, as it is difficult to obtain and has uncomfortable effects on the patient.

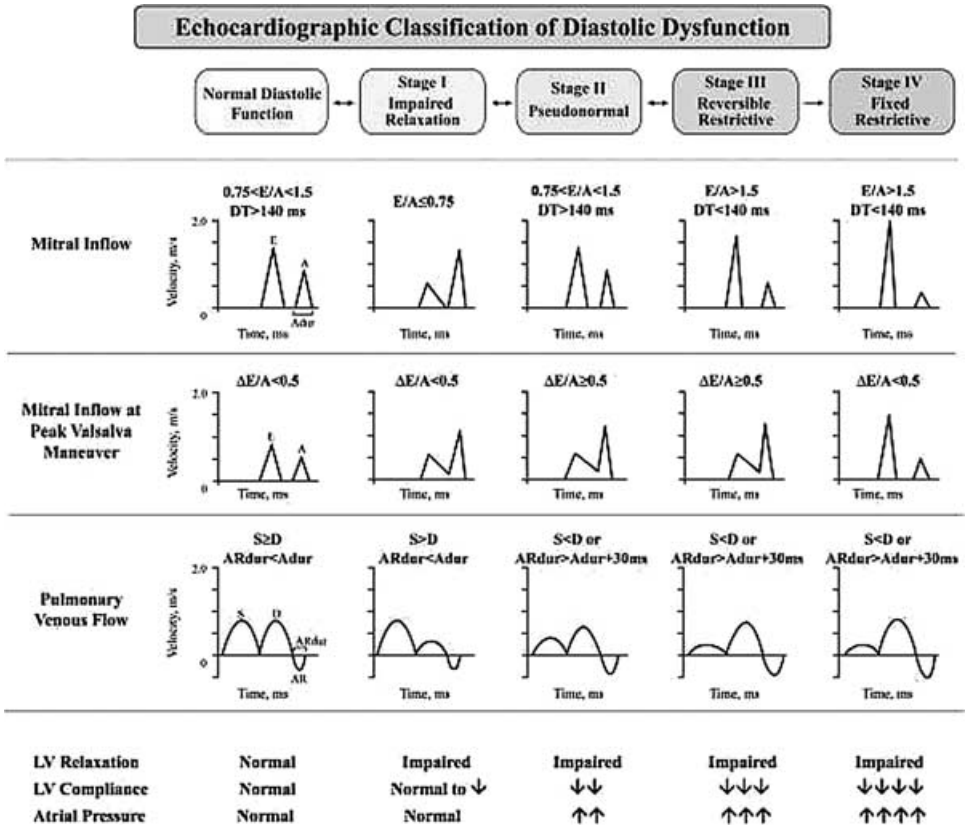

Figure 1. *Classification of diastolic function into five categories. Left atrial (LA) loading conditions affect diastolic parameters in patients with diastolic abnormality (see text). A = late diastolic transmitral inflow; Adur = A-wave duration; Ardur = peak pulmonary vein (PV) atrial reversal flow duration; D = diastolic PV flow velocity; DT = deceleration time; E = peak early diastolic mitral inflow velocity; LV = left ventricle; and S = peak systolic PV flow velocity (modified with permission from Appleton et al.[8]).*

Relatively common applications of several of the described maneuvers are used in the echocardiography laboratory to assess: (1) left heart diastolic function and filling pressures (preload), (2) diagnosis of patent foramen ovale (PFO) and its functional significance, and (3) the evaluation of dynamic obstruction in hypertrophic cardiomyopathy (HCM).

Left Heart Diastolic Function and Preload

Diastolic parameters based on mitral Doppler inflow patterns and the response to decreasing left heart preload during the strain phase of the Valsalva maneuver (Fig. 1) [7,8] may be prognostic in heart failure due to systolic dysfunction.[9,10] By increasing left heart preload with leg raising a more abnormal pattern predicts higher morbidity.[9] The reader is referred to several reviews of diastolic function and assessment of loading conditions.[7,8,11]

With normal diastolic function, the mitral peak E decreases by ≥20% and the peak A wave somewhat less than that during the Valsalva maneuver (Fig. 1), as this maneuver decreases left atrial filling and left atrial pressure.[11] The E/A ratio remains >1 at rest and during the Valsalva maneuver.

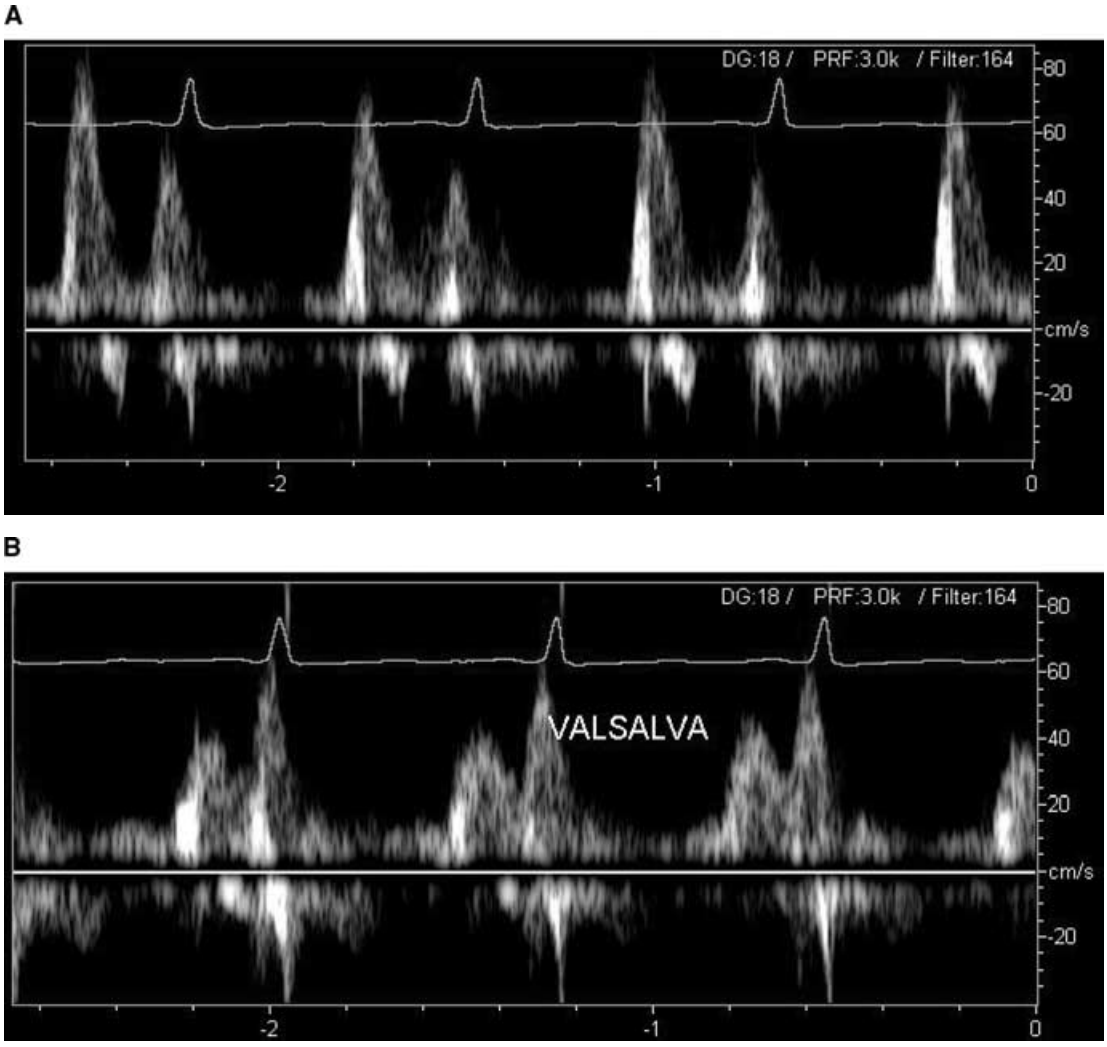

Figure 2. *A middle-aged male with long-standing hypertension and resultant systolic dysfunction presented with heart failure symptoms. Apically obtained mitral inflow pulsed wave Doppler patterns were then obtained.* **A.** *The mitral inflow pattern appears "normal," but* **(B)** *repeat mitral inflow patterns measured during the Valsalva maneuver become that of abnormal relaxation. This is consistent with "pseudonormal" mitral filling, secondary to abnormal relaxation with elevated left atrial filling pressures.*

Stage I diastolic abnormality is associated with a mitral E/A ratio ≤0.75 (impaired relaxation). During the strain phase of the Valsalva maneuver, the E/A ratio remains <1.0.

With Stage II (pseudonormal filling) diastolic filling, the "normal" E/A ratio (E/A ratio from 0.75 to 1.5) will become that of abnormal relaxation (E/A <1). This helps to differentiate normal from pseudonormal mitral filling, as normal diastolic function is associated with an E/A ratio >1 at rest and Valsalva.

With Stage III restrictive filling, the Valsalva maneuver will result in a pseudonormal or even abnormal relaxation pattern (E/A <1) (Fig. 2), but with Stage IV restrictive filling, there will be no change in mitral filling.[12] Patients with

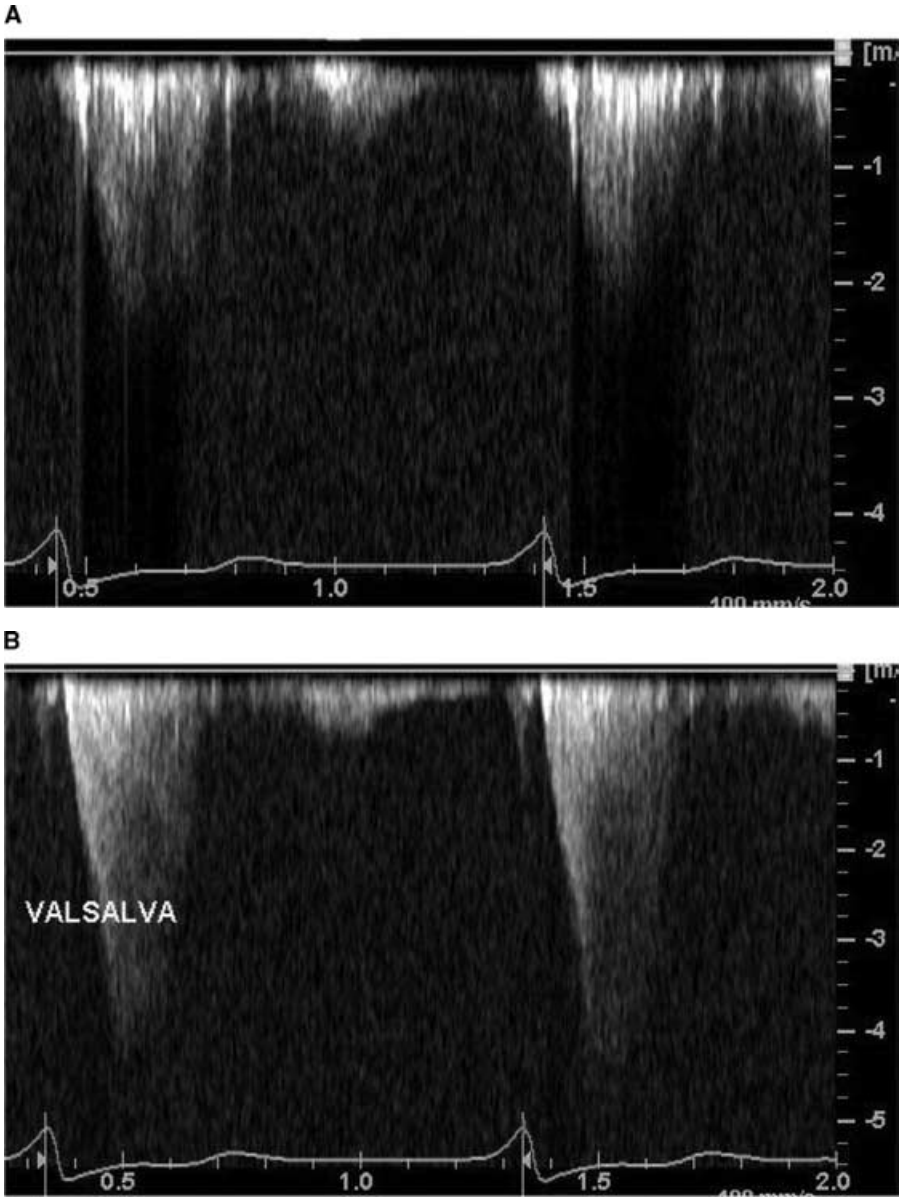

Figure 3. *A 35-year-old female with exertional near-syncope and a murmur was referred for an echocardiogram. The patient was found to have classic hypertrophic cardiomyopathy with mitral systolic anterior motion and aortic valve mid-systolic closure. Continuous wave Doppler (apical five-chamber) of the left ventricular outflow tract* **(A)** *during normal respirations revealed a mid-systolic peak velocity of 2.25 m/sec.* **B.** *With the Valsalva maneuver the gradient increased to 4.5 m/sec.*

left ventricular dysfunction and Stage IV mitral diastolic filling have a poorer prognosis than those with Stage III filling.[9,10]

Atrial Level Shunts

Patent foramen ovale is associated with several clinical disorders, including cryptogenic stroke,[13–20] platypnea orthodeoxia,[21–26] and decompression sickness in divers and astronauts.[27–38] Patent foramen ovale has been noted in up to approximately one-fourth to one-third of the general population by autopsy.[39,40] The autopsy size has been described as "probe" patent (0.2–0.5 cm maximum dimension) in 29% and "pencil" patent (0.6–1.0 cm) in 6%.[39] Not all PFOs are anatomically or physiologically similar, as there appears to be a "continuum" when evaluating PFO for its functional significance.[41] Studies for the evaluation of cryptogenic stroke[13–15,42,43] and decompression sickness[27–31] have used different procedural and diagnostic protocols using agitated saline injected either in an antecubital vein or femoral vein.[44,45] These have been performed both during normal respiration and with the Valsalva maneuver; to not only make a diagnosis of an atrial level right-to-left shunt, but also to attempt a correlation between "bubble load" in the left heart and PFO clinical significance. When evaluating for PFO, atrial shunting from right to left will begin during the release phase (Phase 3).

Dynamic Obstruction of Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is a primary disorder of myocardium manifested as hypertrophy of a nondilated ventricle. Hypertrophy nearly always involves some part of the ventricular septum, most often the anterior septum.[46–48] Obstruction of the left ventricular outflow tract (LVOT) is dynamic, and may be mild or nonexistent at rest.[49] M-mode echo features of dynamic obstruction include systolic anterior motion (SAM) of the mitral valve, and aortic valve mid-systolic closure.[50,51] Continuous wave Doppler flow acceleration at the level of the LVOT has a mid-to-late systolic peaking concave appearance. This dynamic gradient may increase after meals or alcohol ingestion,[52,53] after the compensatory pause of a premature ventricular beat,[54] or during the strain phase of the Valsalva maneuver[55] (Fig. 3).

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