Tissue Doppler in Everyday Diastolic Assessment

Claude Tousignant MD FRCP
Department of Anesthesia, St Michael’s Hospital
University of Toronto

A diastolic dysfunction (DD) assessment algorithm may seem simple to apply however, it is not always straightforward. Flow-derived parameters define the behavior of the chamber in its entirety. These parameters are unfortunately preload sensitive. Furthermore, in the perioperative period patients are not under ambulatory conditions. Examinations under anesthesia and positive pressure ventilation may significantly colour the assessment. In the post-operative period the effects of surgery, CPB as well as intervening factors such as pericardial collections, right ventricular failure, pacing, arrhythmias, tachycardia or inotropic support may significantly influence the assessment. These conditions may change rapidly as the patient improves or worsens and may not be permanent.

Initial examination:
It is unusual for DD to be present without structural abnormalities. Increased LA size, a thickened interventricular septum and a depressed EF are predictors of DD. Although DD is assumed in systolic failure, a normal systolic function does not exclude DD. Changes in some of these parameters however, will not be present following acute changes in the perioperative setting.

Physiology and Doppler parameters:
1) Increased LAP:
If all other factors remain constant (relaxation, compliance and systolic function), an increase in LAP will result in a pulling up of the early filling portion of the PV loop (ie: earlier filling) and a move to the right of the end diastolic portion (larger volume). Please note that the diastolic compliance curve remains normal. An increase in LAP will result in:

1) Increased E velocity.
2) Shortened IVRT (isovolumic relaxation time).
3) Decreased deceleration time (DT).

2) Ventricular relaxation:
Ventricular relaxation is an energy dependent process which results in the rapid decline of LV pressure following the closure of the aortic valve thereby increasing the LA-LV pressure gradient. Tau (\(_\)) (time constant of decay) is measured from the slope of pressure decay on LV catheterization from aortic valve closure to 5mmHg above LVEDP. A larger\(_\) value means it takes longer for the pressure in the LV to drop. A prolongation of\(_\) results in:

1) A prolongation of the IVRT.
2) An increase in DT (prolongation of the LA-LV pressure gradient).
3) A decrease in E velocity (decreased LA-LV pressure gradient).
The patient will become A dominant and dependent on atrial contraction for proper filling. Eventually, the LAP may rise and restore the E/A to a normal value.

3) **Myocardial compliance:**
Myocardial compliance is the sum of multiple factors affecting the distensibility of the myocardium excluding relaxation. LV compliance may be expressed as a volume constant; the volume required to increase the LV pressure by a factor of \( \_ \); the lower the number, the stiffer the ventricle. Reduced compliance results in a steeper EDPV relationship *ie*: a leftward and upward shift of the diastolic (EDPV) curve on the PV loop. The changes in mitral inflow resulting from a decrease in compliance are:

1) A decrease in E velocity.
2) A decrease in DT.

4) **Systolic function:**
With deteriorating systolic function, a larger amount of blood remains in the LV at the end of systole meaning that the heart begins to fill on top of this increased residual volume. This results in a distended LV which operates on the steeper (right shifted) portion of the EDPVR curve. The associated changes in mitral inflow are:

1) A reduced E velocity.
2) A shortened DT.

5) **Atrial function:**
As we use the E/A ratio to determine diastolic function we must remember the role of atrial function. In healthy young patients, the atrium behaves mainly as a conduit and atrial contraction contributes little to LV preload. As DD marches on however, the atrial contribution to LV filling increases. As the LA volume increases, so does its contractility. Further increases in LA size however, result in a loss of mechanical efficiency and function.

**Preload assessment:**
Doppler methods have been used to estimate LAP. For example, a difference of 30msec or more in PV AR duration compared to mitral A duration is suggestive of LVEDP >15. Depressed S/D ratios are also associated with elevated LVEDP. Unfortunately they cannot be applied to normal, healthy patients. For example, an athlete with excellent relaxation will have large E velocities and consequently an S/D ratio <1. None of this is a result of increased LAP. Other markers of ventricular relaxation (_*) such as E’ and Vp will be normal. E/E’ has been used to estimate filling pressures. E’ is relatively independent of preload in patients with abnormal relaxation. An E/E’ ratio <8 predicts a normal LVEDP, a ratio >15 predicts an elevated LVEDP and 8-15 is indeterminate.

**Diastolic Dysfunction Patterns:**
**Stage I:** When relaxation is delayed, the E/A is < 1 and atrial contribution is relatively increased. The IVRT increases to > 100msec and DT is increased. The S/D ratio is relatively increased as the decrease in E velocity is reflected in the D wave. Vp and E’ now reflect an impaired __. As the pattern is A dominant, the occurrence of atrial fibrillation in this group may have significant haemodynamic consequences.

**Stage II:** Indicators of impaired relaxation remain however; LAP has increased restoring E velocity, E/A ratio and DT to normal values. A Valsalva maneuver will help elucidate this pattern.

**Stage III:** In this restrictive stage, the LV stiffness has now increased such that the LAP is markedly increased. There is a characteristic increase in E/A which is superior to 1.5, a markedly reduced DT and IVRT all resulting from increased LAP. Indicators of relaxation such as Vp and E’ are severely depressed. At this point, the patients will demonstrate overt heart failure.

**Stage IV:** The pattern is similar to stage III however, it cannot be returned to stage III using preload reduction techniques.

**Other flow derived methods:**

**Propagation velocity (Vp):**

The propagation velocity (Vp) is a measure of the velocity of a column of blood as it is accelerated from MV to apex. There are 3 phases: columnar flow (I), vortex flow (II) and atrial contraction (III). An isovolumic phase may occasionally be seen. Although measured during the filling phase, the Vp has correlated well with __ and is relatively independent of preload. Its value is normally >45cm/s in older patients and over 55cm/s in young healthy patients. The Vp decreases with increasing __ even when the E velocity is high. This is a result of a larger influence from vortex flow. There is still controversy regarding load dependence. Ventricular geometry can have a significant impact as well; dilated ventricles display mostly vortex flow whereas patients with a small LV size and LVH may have delayed relaxation yet fast Vp. Significantly however, there remains a high intra and inter observer variability in the measurement of the slope. Vp may be a more complex measure that that of simple ventricular relaxation.

**Tissue based assessment of DD:**

**Mitral annular E velocity (E’):**

E’ can be measured using tissue Doppler. It is generally assumed that velocities measured at single or multiple sites will represent global relaxation. However, relaxation vectors are complex and are not limited to the longitudinal ascent velocity of the mitral annulus. Indeed, a large number of abnormal myocardial segments (12/18) must be present before flow-based abnormalities are detected. E’ has been shown to be inversely related to __. E’ may be helpful in discriminating pseudonormal from normal patterns or restrictive from constrictive disease. As E’ occurs during the early phase of LV filling, it is influenced by relaxation and filling pressures. In patients with dilated cardiomyopathies, E’ may fall in the normal range where normalizing to LV dimension may be necessary. It can also be affected by mitral annular calcification, regional wall motion abnormalities, tethering, rotation and translation as well as Doppler angle.
Myocardial strain and strain rate:
Tissue Doppler and speckle tracking technology allow us to measure myocardial events independent of translation, tethering and rotation. Colour-encoded TDE has good temporal resolution however, spatial resolution is limited by the Doppler plane. Speckle tracking has the advantage of superior spatial resolution however more limited temporal resolution.

Myocardial deformation rate (strain rate) can be measured using normalized velocity gradients based on tissue Doppler or speckle tracking technology: \( \frac{(V_2 - V_1)}{L} \). Integrating SR yields the percentage of deformation; the strain (\( \varepsilon \)). Deformation occurs along 3 axes; longitudinal, radial or circumferential.

Similar to flow-derived \( V_p \), the propagation of ventricular thinning during the filling phase can also be assessed using SR measurements. It is a measure of LV chamber expansion or stretching which propagates from base to apex. It can be measuring using a curved M-mode in myocardial tissue using SR colour-encoded tissue Doppler. The slope of the colour edge determines the “thinning or stretching” propagation rate. It has been shown to be reduced in patients with delayed relaxation however, remains load dependent. It does not always correlate with \( V_p \). In patients with LVH and a small cavity for example, \( V_p \) was found to be fast where SR propagation was slow. Contrary to \( V_p \), the SR propagation is not affected by chamber geometry.

The rate of myocardial deformation during IVR (SR\(_{IVR}\)) is a good, unpolluted measurement of relaxation (\( \varepsilon \)) as it occurs before LV filling. When measured using speckle tracking, its more global nature may render it superior to \( E' \). The ratio \( E/SR_{IVR} \) has also been shown to be a good predictor of preload and may be particular useful in patient who fall within the indeterminate zone (8-15) using \( E/E' \).

Ventricular untwisting:
The architectural fibre construction of the left heart, a double helix results in a configuration of the LV which promotes torsion. The normal configuration, as viewed from the apex (in TTE) consists of a clockwise rotation at the base (- deflection) and a counter clockwise rotation at the apex (+ deflection). The difference between the apical and basal rotation is the measure of torsion. The dominant apical rotation relative to the base may allow for measurements based solely on apical rotation. During diastole, rapid untwisting (predominantly apical) is the result of a release of stored potential energy (elastic recoil) and active relaxation. The magnitude of untwisting may also be related to systolic function (end systolic volume). LV untwisting is up to 50% complete prior to mitral valve opening.

In athletes, Notomi et al (Circulation 2006); found peak early diastolic untwisting velocities of \(-2.0\pm0.7\) rad/s (115°/s) increasing to \(-5.6\pm2.3\) rad/s (321°/s) during exercise. Furthermore, the rapid untwisting occurred prior to filling (the E wave). This suggests that untwisting can be a load-independent measure of global relaxation (but HR and systolic function dependent). It was the main contributor to the development of pressure gradients in the LV. On the other hand, Park et al found that peak LV systolic torsion untwisting rate was increased in patients with early stage or mild DD. This normalized in
patients with stage II disease and was depressed in stage III DD. This brings up the question as to whether the mechanism underlying torsion and untwisting is dependent on myocardial relaxation as we routinely measure it through mitral flow or annular ascent and Vp. Furthermore, is the increased untwist rate part of a compensatory process palliating for the reduced longitudinal relaxation in DD? Clearly further research is required in this area to elucidate the clinical usefulness.

**Useful References:**


