Basic Diastology:
Left Atrial Inflow and Outflow
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I have no conflicts of interest and I have no disclosures

Objectives: At the conclusion of this session, the participant should be able to:
1 – Understand left atrial blood flow patterns
2 – Understand and interpret pulmonary vein flow spectral profiles as assessed by Doppler echocardiography
3 – Understand normal trans mitral pressure gradients and flow patterns as assessed by Doppler color flow and spectral displays
4 – Be introduced to the basics of diastolic dysfunction and it’s assessment

Introduction: Systolic performance of the left ventricle (LV) primarily assessed by measurement of blood pressure is the pillar of hemodynamic monitoring in the perioperative period. It is well recognized that blood pressure serves as a readily measured proxy of cardiac output - the resultant product of stroke volume time heart rate. Determinants of stroke volume are similarly well characterized and consist of contractility, pre-load and after-load. The relationships between chamber pressures and the cardiac cycle as well as left ventricular pressure volume relationships (“PV-loops”) are presented in most chapters on “left ventricular function” with assessment methods and interventions primarily focused on the systolic-ejection phase of ventricular function. Receiving less print and attention is the filling phase of the cardiac cycle: diastole. It is now well recognized that this is not a simple passive phase of filling, but rather a complex period of relaxation, compliance and interaction between adjoining and communicating chambers.

Advanced diastology encompasses measurements of tissue Doppler imaging, transmitral flow propagation and strain and strain rate. Essential to one’s understanding of these sophisticated modalities is a fundamental understanding of trans atrial blood flow patterns, timings and gradients. These can be measured directly by high fidelity catheters but can be approximated with considerable accuracy (given certain assumptions) by Doppler derived echocardiography measurements of blood flow.

Diastole: Isovolumic contraction (IVC) during which the LV contracts without volume change until intracavitary pressure exceeds that in the aorta precedes the ejection phase of systolic function. An interval of rapid LV pressure change occurs at the onset of diastole as the ventricle relaxes until the pressure falls below that of the left atrium. This period is called the Isovolumetric Relaxation Time (IVRT) and can be measured as the interval from the dichrotic notch of aortic valve closure and the opening of the mitral valve and represents LV relaxation. High fidelity pressure measurements permit
quantification of this by measuring the first derivative of the LV pressure with respect to time and further by solving for the time constant of the pressure decline (τ) from the equation below.

\[ \tau = \frac{-1}{\text{d}P/\text{d}t} \]

LV relaxation is recognized to be a complex active process and impaired by conditions such as hypertrophy, ischemia and negative inotropic drugs. It involves the resequestration of calcium into the sarcoplasmic reticulum, and an energy requiring uncoupling of the contractile elements of the myofibrils. Normal IVRT is 70-90 ms and when prolonged usually represents impaired relaxation. However, since this represents the time from aortic valve closure to mitral valve opening, it may be influenced by many variables.

Transmitral Flow Profiles: Whenever left atrial (LA) pressure exceeds LV pressure flow will occur across the mitral valve. Examination of the pressure-time curves of the LV and LA reveals that there are tow periods of large gradients and therefore flow volumes during diastole. These are the early rapid filling phase and a second phase occurring later in diastole and resulting from the increase in LA pressure from atrial contraction. Doppler echocardiography permits quantification of the blood flow velocities and may be displayed as either color flow patterns or spectral: velocity/time plots. Pulsed wave Doppler is typically used as velocities across a non-stenotic mitral valve should not exceed 1.5 m/sec. Two dagger chapped flow profiles below the baseline (away from the probe when using TEE) result and are labeled “E” and “A” waves corresponding to “early” and “atrial contraction.” A period of relatively little pressure gradient and therefore flow occurs in between these two waves and is called diastasis: a period of little transmitral flow since LA volume has just been translocated into the LV during the “E” wave and LV pressure has risen along a flat LV compliance relationship-curve. Absolute velocities of the “E” and “A” waves are listed in tables but typically in the range of 60-70 cm/sec and 40-50 cm /sec respectively. If one images with Doppler color flow, two blue pulses of flow can be imaged...
**Diastolic Dysfunction as detected by Echocardiography:** Diastolic dysfunction usually involves impaired relaxation and/or impaired compliance of the ventricle. Initial assessment of diastolic dysfunction can be made by careful observation of 2-D imaging of the ventricle. This qualitative method is largely supplanted by more qualitative characterization of LV diastole and relies upon Doppler flow profiles of the trans-mitral diastolic flow, systolic and diastolic pulmonary vein profiles and in more advanced settings, tissue Doppler mapping and mitral inflow Doppler flow propagation slopes.

As mentioned above, impaired relaxation will delay the fall in LV pressure and result in a prolongation of the time interval between the end of LV contraction and when the LV pressure falls below LAP and the mitral valve opens (IVRT). Not only will mitral valve opening be delayed when compared to normal, but since LV pressures are declining more slowly, there is less pressure difference from the LA to LV during the early phase of filling and the resultant “E” wave is blunted and flatter. Since less blood will be emptied from the LA to the LV, subsequent atrial contraction boosts the LA pressure and an increase in “A” wave velocity occurs. Qualitatively this results in “E” wave / “A” wave velocities of < 1. The flatter profile can be quantitated as the deceleration time (DT). This later may become shorter and represents reduced LV compliance as well.

As mentioned above, the LA and LV are in continuity hence pressure gradients and hence flow between them will be interrelated. As dysfunction progresses and LA emptying becomes impaired, LA pressures will increase. The consequence of such is that LV pressures do not need to fall as far in order for mitral valve opening to occur. IVRT can be seen to shorten (<60ms) and early restoration of a gradient occurs with a return towards a more normal “E” wave velocity and E/A ratio. This represents pseudonormalization. Finally, the degree of dysfunction progresses to profound restrictive filling dynamics and in this case, all gradients are of short duration, the markedly elevated LAP results in a brief high gradient and “E” wave but little “A” wave and a ratio of E/A > 2.0.

Thus what occurs is a continuum or E/A ratio reversal, pseudo-normalization and then restrictive pattern. The location along this continuum depends to a large degree on left atrial filling and pressure. Insight into LA compliance can be gained by examining gradients for inflow of blood from the lungs or pulmonary vein flow profiles (PVF).

Placement of a pulsed wave Doppler sample gate in the left upper pulmonary vein permits assessment of the normal phasic velocities of pulmonary vein to left atrial blood flow. Under normal conditions, the onset of ventricular systole results in the decent of the mitral apparatus and a “suction” on
the LA. The LA pressure is already low as it had just emptied into the LV following atrial contraction and further, pulmonary vein pressures are high (from impeded emptying from atrial contraction) and increasing from right ventricular ejection into the lungs. These conditions all favor a high systolic PVF velocity. As the LA fills, its pressure increases, the MV bulges with end systole and the pulmonary venous pressure falls hence the systolic PVF wave approaches zero. The onset of diastole and LV relaxation lowers LV pressure below LA pressure and the mitral valve opens. This lowers LAP and hence a gradient for vein-LA blood flow is reestablished. The end of diastole is punctuated by atrial contraction, raising LAP and impeding pulmonary vein flow and in fact resulting in a pressure gradient reversal and transient flow retrograde in the pulmonary veins (“a” reversal wave- below the baseline on TEE).

Advancing diastolic dysfunction alters these LA and pulmonary vein gradients. As with mitral E and A waves, early impaired LV relaxation diminishes diastolic E wave flow and hence LAP. This will lead to a parallel loss of the gradient for pulmonary vein flow during diastole and a smaller D wave. Further progression of dysfunction and pseudo-normalization of transmitral flow ratios now results in a systolic blunting of PVF. The LAP is high from impaired LV compliance thus the gradient less. Finally, severe impediment to filling the ventricle in turn means that LAP will only decline during the early phase of diastole. Superimposing the two profiles then allows the distinction between a normal E/A ratio and one that is pseudo normal.

![Graph showing diastolic function](image)

**Summary:** Assessing diastolic performance is assuming increasing importance in the quantitation of LV function. Analysis of transmitral and pulmonary vein flow profiles is the cornerstone of this analysis and understanding the phenomena that lead to the magnitude and time course of LA pressure gradients is essential to making these interpretations.

**References:**