Restrictive Filling

When there is poor LV compliance (or increased LV stiffness), with LV filling there is a marked and early increase in LVFP (and thus LAP); this results in a restrictive LV filling pattern. The increased LAP results in an increased transmitral E velocity. Due to poor LV compliance, there is a rapid rise in LV pressure with early diastolic filling with a rapid equalization of LV and LA pressures. This results in significant shortening of the DT. With atrial contraction, due to the very high LV pressures, there is only a small transmitral A velocity. In addition, the A duration is shortened as the LVEDP is elevated so there is an early crossover between the LV and LA pressures which terminates mitral inflow prematurely. When there is poor LA contractile function, the A velocity may be totally absent even though the patient is in sinus rhythm. This restrictive filling pattern is characterised by E/A ratio ≥ 2 and a DT less than 160 ms (Fig. 3.10, D).

The restrictive filling profile can be further classified as reversible restrictive filling or irreversible (fixed) restrictive filling. A reversible restrictive filling can be differentiated from an irreversible restrictive filling by decreasing preload; for example, by asking the patient to perform the Valsalva manoeuvre. When there is a reversible restrictive filling, preload reduction will result in a reversal of this filling profile to either a pseudonormal filling profile or even to an impaired relaxation filling profile (Fig. 3.15, top). In an irreversible restrictive filling, preload reduction will not change the pattern of this profile; that is, it maintains the characteristic features of a restrictive profile (E/A ratio ≥ 2 and a DT <160 ms) (Fig. 3.15, bottom).

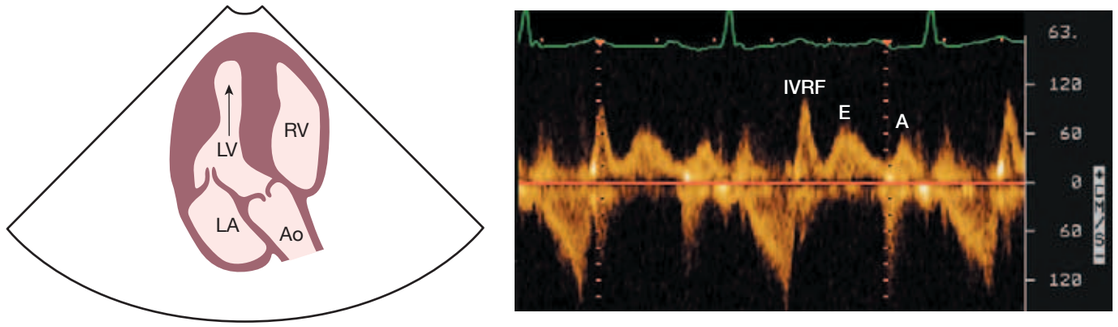

Figure 3.14 Isovolumic relaxation flow *(IVRF)* occurs between two regions of the left ventricle *(LV)* due to marked asynchrony in ventricular relaxation (*left*). This flow is most commonly directed from the base of the heart to the apex during the isovolumic relaxation flow period (*right*). IVRF may be seen in patients with intracavity gradients caused by LV hypertrophy and vigorous LV systolic function with near cavity obliteration in systole. It is important to recognise IVRF and not to confuse this flow pattern with that of a triphasic mitral flow profile.
Ao = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.

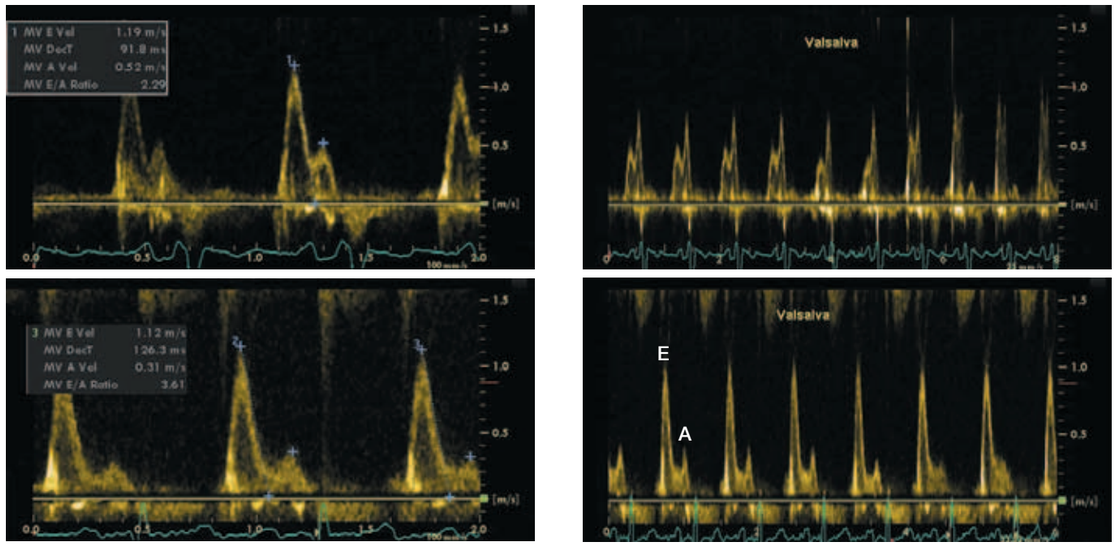

Figure 3.15 The two resting transmitral inflow profiles displayed to the left show characteristic features of a restrictive filling profile: E/A ratio > 2.0, a DT (DecT) < 160 ms. Following the Valsalva manoeuvre the transmitral inflow pattern on the top reverts to an impaired relaxation pattern *(top right)*; this is characteristic of reversible restrictive filling. Following the Valsalva manoeuvre the transmitral inflow pattern on the bottom does not change *(bottom right)*; that is, the profile maintains the characteristic features of a restrictive filling profile (high E/A ratio and short DT). This is characteristic of irreversible restrictive filling.

Isovolumic Relaxation Time

As mentioned, the IVRT is the interval between aortic valve closure and mitral valve opening. The IVRT can therefore be measured by displaying both the transmitral signal and the left ventricular outflow tract (LVOT) signal on the same spectral trace. This is accomplished by aligning either the PW or continuous-wave (CW) Doppler beam so that it intercepts flow between these two regions (Fig. 3.16). The IVRT is optimally displayed when the closing click of the aortic valve is depicted both above and below the zero baseline. Measurements should be performed at a sweep speed of 100 mm/s, at end-expiration, and averaged over 3 consecutive cardiac cycles.

In the normal heart, the IVRT is between 50-100 ms. However, as for the transmitral measurements, the normal value for the IVRT is dependent on the patient's age (Table 3.3). For example, with advancing age there is a decline in elastic recoil and myocardial relaxation; this leads to prolongation of the IVRT.

Factors Affecting IVRT

The IVRT is influenced by a number of factors including LV relaxation and LAP (Fig. 3.17). When there is impaired LV relaxation, the LV pressure decline during IVR is slowed so there is a longer time before the LV pressure falls below (crosses over) the LAP which means that mitral valve opening is delayed; this results in prolongation of the IVRT. An IVRT ≥ 100 ms is consistent with impaired LV relaxation (Fig. 3.17, B). IVRT is also dependent on preload or LAP. Therefore, as for the transmitral inflow profile, pseudonormalisation of the IVRT can also occur. Recall that pseudonormalisation occurs when there is impaired relaxation with a moderate increase in LAP. So even though the LV pressure decline is slower because the LAP is elevated the crossover between the LA and LV pressures now occurs within a normal timeframe and, thus, the IVRT is within the normal range (Fig. 3.17, C). When there is a marked increase in LAP, as seen with restrictive filling, the crossover between the LV and LA pressures occurs earlier than normal; this results in earlier than normal mitral valve closure and shortening of the IVRT. An IVRT ≤ 60 ms is consistent with restrictive filling (Fig. 3.17, D).

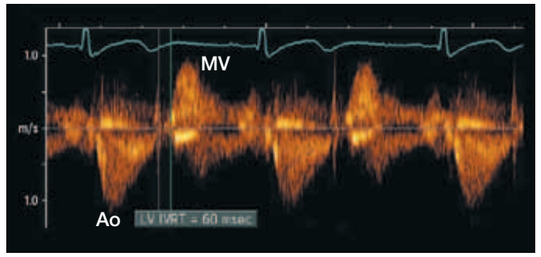

Figure 3.16 The isovolumic relaxation time (*IVRT*) is measured as the time interval between aortic valve closure and mitral valve opening. This is achieved by aligning the Doppler beam so that is intercepts flow between the aorta (*Ao*) and mitral valve (*MV*).

Pulmonary Venous Flow

The pulmonary venous signal reflects the pressure gradient between the pulmonary veins and the LA over the cardiac cycle (Fig. 3.18).

The pulmonary venous signal is acquired via PW Doppler from the apical 4-chamber view. A 3-5 mm sample volume is positioned approximately 1 cm into the right upper pulmonary vein (Fig. 3.19). From the spectral Doppler trace, several measurements are performed (Fig. 3.20).

Systolic forward flow reflects the systolic pressure gradient between the pulmonary vein and the LA and is identified as the peak systolic (S) velocity on the pulmonary venous trace (Fig. 3.20, A). The S velocity may appear biphasic with S1 and S2 components. S1 occurs in early systole and represents the increase in pulmonary venous flow secondary to LA relaxation. S2 occurs in mid to late systole and is produced predominately by the increase in pulmonary venous pressure that occurs with transpulmonary propagation of flow after RV systole.

Diastolic forward flow reflects the diastolic pressure gradient between the pulmonary vein and the LA and is identified as the peak diastolic (D) velocity on the pulmonary venous

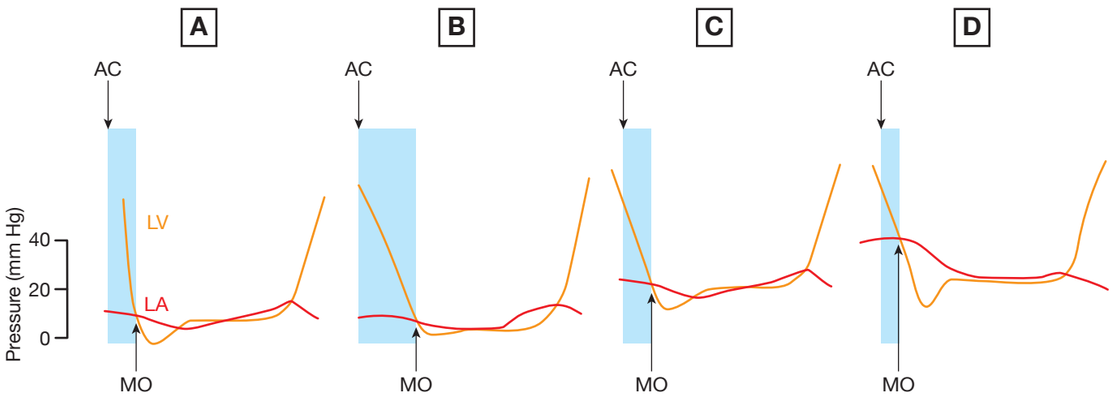

Figure 3.17 This schematic shows the left ventricular (LV) and left atrial (LA) pressure traces during diastole. The IVRT (shaded area) is measured from aortic valve closure (AC) to mitral valve opening (MO); MO occurs when the LV pressure falls below the LA pressure. **A** = an example of a normal IVRT. **B** = an example of an IVRT seen with impaired LV relaxation; observe that due to delayed LV relaxation, the LV pressure decline is slower than normal and crosses over the LA pressure later resulting in prolongation of the IVRT. **C** = an example of a pseudonormal profile; observe that the IVRT is normal. Pseudonormalisation of the IVRT occurs when there is impaired LV relaxation plus an increase in the LA pressure; therefore, LV pressure crosses over the elevated LA pressure within a normal timeframe. **D** = an example of an IVRT seen with restrictive filling; observe that the IVRT is very short even though there is impaired LV relaxation. Shortening of the IVRT occurs due to a marked increase in the LA pressure so that crossover between the LV and LA pressures occurs earlier than normal.