

### Exclusion of LV and/or RV thrombus

Thrombus occurs due to stasis of blood flow. The recognition of LV thrombus in patients with DCM is especially important as LV thrombus is a potential source for systemic embolisation. The 2D echocardiographic features of thrombus include the appearance of echo-dense material within the chamber cavity; the echogenicity of thrombus is usually distinct from underlying endocardium (Fig. 6.13). Importantly, 2D image optimisation is fundamental for the detection of LV thrombus. Therefore, the sonographer should:

- Use the highest possible transducer frequency (to improve spatial resolution),
- Use harmonic imaging (to improve contrast resolution),
- Use a shallow field of view (thrombus usually at the apex),
- Reposition the focal zone (to improve lateral resolution),
- Slowly and carefully pan through the ventricle (anterior to posterior) from all possible imaging planes,
- Confirm the presence of thrombus in more than one imaging plane.

It is also important to be aware that poor image quality may mask LV thrombus (especially at the apex) while normal anatomic structures and imaging artefacts may mimic LV thrombus. Anatomic structures mimicking LV thrombus include: (1) aberrant fibrous cords or accessory bands, (2) ruptured papillary muscle and/or cords, and (3) prominent LV trabeculations. Colour flow Doppler imaging or the injection of intravenous contrast agents may be very useful in the determining the presence or absence of thrombus in these situations (see Fig. 5.26 and 5.27).

### Role of Echocardiography in Cardiac Resynchronization Therapy (CRT)

LV dilatation and impaired systolic function can result in desynchronised ventricular contraction which effectively reduces the LVEF and increases the severity of MR. Cardiac resynchronization therapy (CRT) aims to resynchronize ventricular contractility thereby improving the LVEF and reducing MR severity. This is achieved by biventricular pacing whereby the RV is paced in the traditional manner and the lateral wall of the LV is paced by placing a pacing wire down the coronary sinus and into a coronary vein on that LV wall.

A positive response to CRT includes a decrease in NYHA class by one class, an increase in exercise capacity, improved quality of life, less hospital admissions, evidence of reversed LV remodelling, improved systolic function, and a decrease in MR severity. However, in approximately 20-30% of patients receiving CRT there is no apparent clinical improvement; these patients are referred to as non-responders. Various echocardiographic techniques have been investigated in an attempt to distinguish between CRT responders and non-responders. Echocardiography can also be utilised to determine the degree of LV reverse remodelling and the reduction in MR severity, and to optimise atrioventricular and interventricular intervals following CRT.

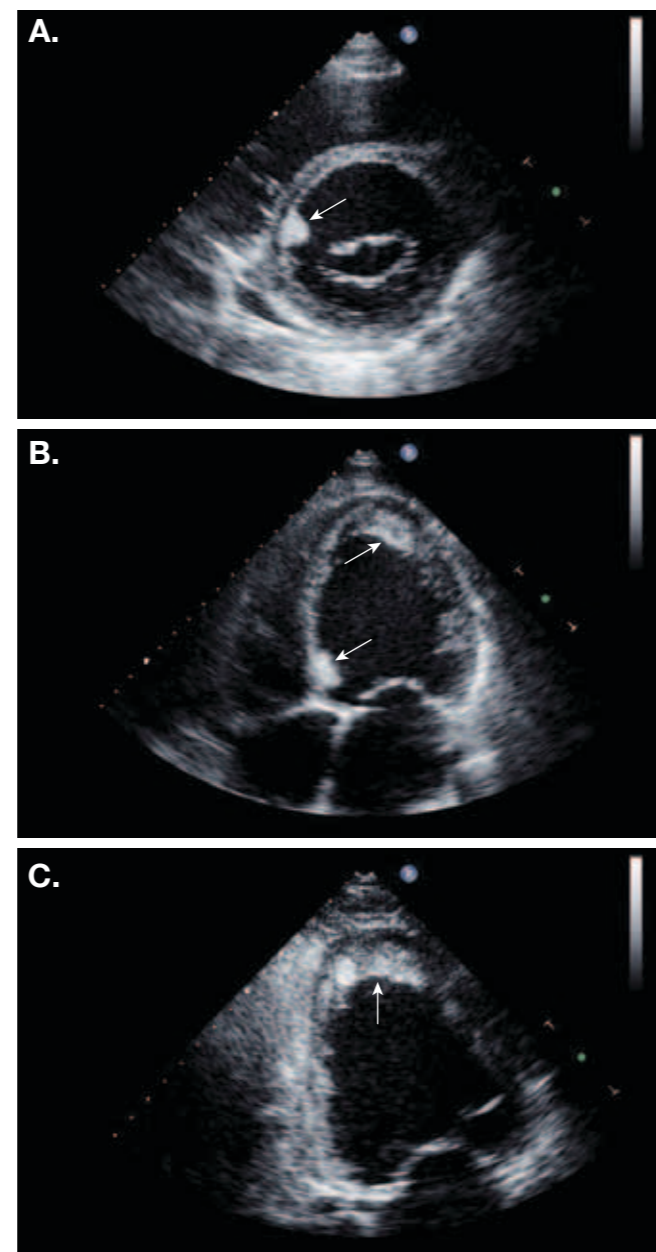
### Echocardiographic Measures of Dyssynchrony

Dyssynchrony can be classified as interventricular dyssynchrony and/or intraventricular dyssynchrony. Interventricular dyssynchrony refers to dyssynchronous contraction between the LV and RV while intraventricular

dyssynchrony refers to dyssynchronous contraction within the LV. Comparisons of selected measures of dyssynchrony including methods, normal values, cut-off values for identifying dyssynchrony, advantages and limitations are summarized in Table 6.9.

### Interventricular dyssynchrony

Interventricular dyssynchrony or interventricular mechanical delay (IVMD) can be measured using PW Doppler as well as DTI. A normal interventricular conduction time is < 20 ms; a delay  $\geq 40$  ms is an indicator of interventricular dyssynchrony. Using PW Doppler, IVMD is derived from two measurements: (1) the RV pre-ejection period (RVPEP) and (2) the LV pre-ejection period (LVPEP) (Fig. 6.14). The IVMD is then derived as the difference between the LVPEP and the RVPEP.



**Figure 6.13** These images were recorded from a patient with a dilated cardiomyopathy and left ventricular (LV) thrombus. Observe that there are two prominent echogenic masses within the LV (arrows). An echogenic mass on the inferior septum is seen in the parasternal short axis view (A) and in the apical 4-chamber view (B); an echogenic mass at the LV apex is seen in the apical 4-chamber view and in the apical 2-chamber view (C). The appearance of these masses is consistent with a laminar thrombus.

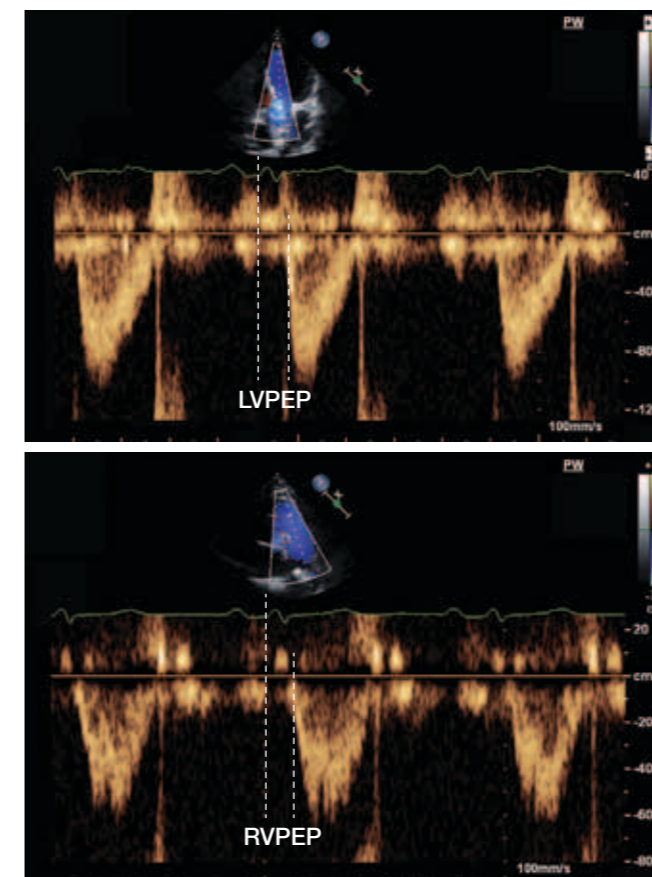
Using DTI, IVMD is derived from two measurements: (1) the time to systolic peak from the RV lateral annulus (Ts [RV]) and (2) the time to systolic peak from the LV lateral annulus (Ts [LV]) (Fig. 6.15). The IVMD is then derived as the difference between the Ts [LV] and the Ts [RV].

### Intraventricular dyssynchrony

Several methods have been described for measuring intraventricular dyssynchrony. They include the septal to posterior wall delay (SPWD), the opposing wall delay (OWD), and by calculating the standard deviation of the time-to-peak systolic velocity from 12 sites (Yu index).

The SPWD is a measure of radial intraventricular dyssynchrony. This measurement can be derived from two M-mode measurements: (1) the time to the first posterior peak of the IVS after the QRS (interval A) and (2) the time to the first anterior peak of the posterior wall following the QRS (interval B) (Fig. 6.16). The SPWD is then derived as the difference between interval B and interval A. Alternatively, the SPWD can be measured as the interval between the peak posterior deflection of the IVS and the peak anterior deflection of the PW. The normal SPWD is < 50 ms; a SPWD  $\geq 130$  ms is an indicator of intraventricular radial dyssynchrony.

The OWD is a measure of longitudinal intraventricular dyssynchrony. This measurement can be derived from pulsed

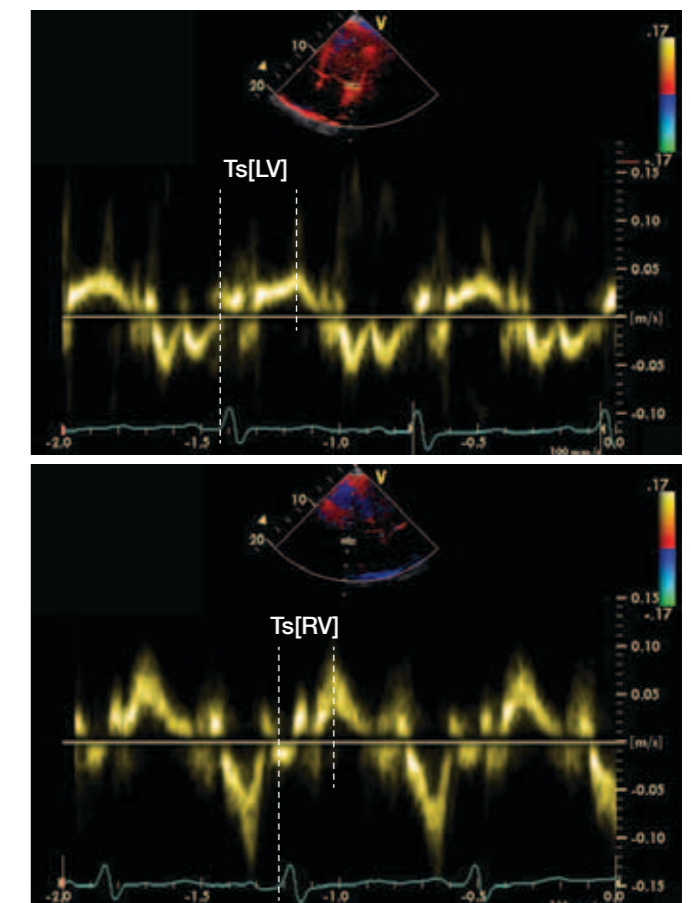


**Figure 6.14** Interventricular mechanical delay (IVMD) via pulsed-wave (PW) Doppler is derived from the left ventricular pre-ejection period (LVPEP) and the right ventricular pre-ejection period (RVPEP). From the PW Doppler signal recorded from the left ventricular outflow tract, the LVPEP is measured from the onset of the QRS complex of the ECG to the onset of systolic flow (top). From the PW Doppler signal recorded from the right ventricular outflow tract, the RVPEP is measured from the onset of the QRS complex of the ECG to the onset of systolic flow (bottom). The IVMD is calculated as the difference between the LVPEP and the RVPEP:  $IVMD = LVPEP - RVPEP$ .

and colour DTI from either an apical 4-chamber view or apical long axis view. For example, from the apical 4-chamber view, two measurements are required: (1) the time to systolic peak from the septal mitral annulus (Ts [S]) and (2) the time to systolic peak from the lateral mitral annulus (Ts [L]) (Fig. 6.17). In this instance, the opposing wall delay corresponds to the septal to lateral delay (SLD) which is derived as the difference between (Ts [S]) and (Ts [L]). The normal SLD is < 50 ms; a SLD  $\geq 65$  ms is an indicator of intraventricular longitudinal dyssynchrony.

The Yu index is also a measure of longitudinal intraventricular dyssynchrony. This index is derived from colour DTI by measuring the time to systolic peak (Ts) from 12 sites of the LV myocardium: Ts is measured from the basal and mid segments of the apical 4-chamber view, the apical 2-chamber view, and the apical long axis view. The Yu index is then calculated as the standard deviation (SD) of these 12 Ts measurements. The normal Yu index is < 30 ms; a SD  $\geq 33$  ms is an indicator of intraventricular longitudinal dyssynchrony.

This section on CRT provides a brief overview of the role of transthoracic echocardiography in this therapy. It should be noted that advanced techniques such as strain, speckle tracking and 3D echocardiography have also been used in guiding CRT (see the Further Reading section of this Chapter for selected articles on this topic).



**Figure 6.15** Interventricular mechanical delay (IVMD) via Doppler tissue imaging (DTI) is derived from the onset of the QRS complex of the ECG to the systolic myocardial peak (s') at the left ventricular lateral annulus (Ts [LV]) (top) and at the right ventricular lateral annulus (Ts [RV]) (bottom). The IVMD is calculated as the difference between the Ts [LV] and Ts [RV]:  $IVMD = Ts [LV] - Ts [RV]$ .