

Figure 4.37 The image on the left is a mid-systolic frame recorded from an apical 4-chamber view. Observe the marked dilatation of the right atrium (RA) and right ventricle (RV) with a relatively smaller left ventricle (LV). Also observe that the tricuspid valve leaflets fail to coapt during systole; this is due to the marked dilatation of the tricuspid annulus. The interatrial septum also bows towards the left atrium (LA); this indicates a marked increase in the RAP during ventricular systole. The image on the right is the corresponding colour flow Doppler image. As expected, there is a large TR colour jet area into the RA. In addition, flow convergence on the RV side of the tricuspid valve is noted which is also consistent with significant TR.

Prognostic Variables

Several echocardiographic indices have been reported to offer prognostic value for patients with PAH (Table 4.18). Therefore, these indices should be assessed in the echocardiographic examination for all patients with PAH.

A frequent cause of death in PAH is RV failure (RVF). RVF occurs due to a combination of diastolic and systolic dysfunction. Systolic dysfunction occurs due to chronic pressure overload of the RV. Diastolic RV dysfunction is thought to be related to RV hypertrophy and/or chronic pressure overload. Therefore, RV dysfunction is an important variable in determining prognosis. Indices such as TAPSE and RV MPI reflect RV function and, thus, may be useful predictors of adverse outcome in patients with PAH. RV MPI is measured from the IVCT, IVRT and ET (see Chapter 2).

TAPSE has also been reported as a predictor of survival in PAH. TAPSE is measured via M-mode from the lateral tricuspid annulus (see Chapter 2). The presence of any degree of pericardial effusion has also been shown to be a predictor of mortality. Typically, patients with PAH and an associated pericardial effusion tend to have more severe PHTN with increased RAP. It is thought that pericardial effusions in patients with PAH occurs due to impaired venous and lymphatic drainage resulting from elevated RAP.

RA enlargement is another echocardiographic abnormality that reflects the severity of right heart failure and predicts adverse outcomes in patients with severe PAH. The RA area is traced from an apical 4-chamber view at end-systole; this area is then corrected (indexed) for height.

The left ventricular eccentricity index (EI) was originally described as a method of quantifying the abnormal IVS curvature in RV pressure and volume overload states. From the parasternal short axis view at the level of the papillary muscles, this index is derived from the LV cavity dimensions measured parallel and perpendicular to the IVS (Fig. 4.38). From these two measurements, the EI is derived:

Equation 4.29

$$EI = D_2 \div D_1$$

where EI = eccentricity index (unitless)

D₁ = LV cavity dimension perpendicular to the IVS (cm)

D₂ = LV cavity dimension parallel to the IVS (cm)

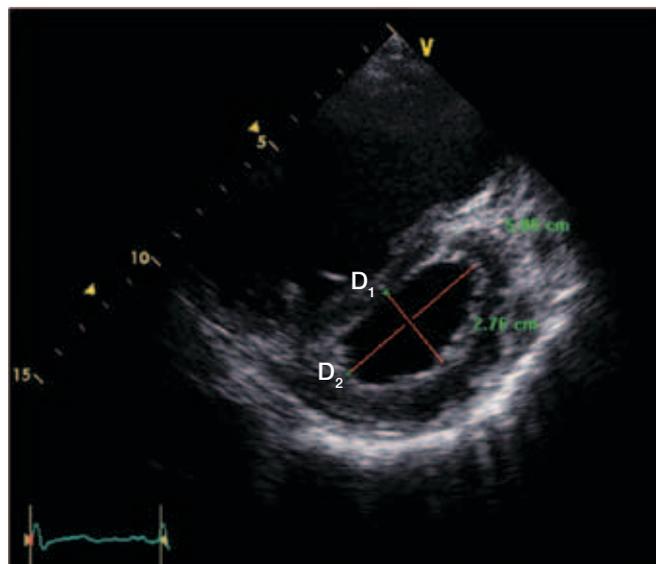


Figure 4.38 The LV diastolic eccentricity index (EI) is measured from the parasternal short axis view at the level of the papillary muscles at end-diastole. The LV cavity is measured parallel to the IVS (D₂) and perpendicular to the IVS (D₁). The diastolic EI in this example is:

$$EI = D_2 \div D_1 = 5.06 \div 2.76 = 1.83$$

Table 4.18 Echocardiographic Prognostic Variables in PAH

	Better prognosis	Worse prognosis	Ref.
RV MPI	< 0.83	≥ 0.83 [^]	1
	< 0.98	≥ 0.98*	2
TAPSE (cm) + EI	>1.8 + <1.7	≤1.5 + ≥1.7	3
Pericardial effusion	None	Present	2, 4, 5
RA Area index (cm ² /m)	< 5	> 5	4

[^] patients with idiopathic PAH; * patients with PAH due to any cause.

References: [1] Yeo TC, Dujardin KS, Tei C, et al. *Am J Cardiol* 1998;81: 1157–1161; [2] Brierre G, Blot-Souletie N, Degano B, et al. *Eur J Echocardiogr.* 2010 Jul;11(6):516-22; [3] Ghio S, Klersy C, Magrini G, et al. Prognostic relevance of the echocardiographic assessment of right ventricular function in patients with idiopathic pulmonary arterial hypertension. *Int J Cardiol.* 2010 Apr 30;140(3):272-8; [4] Raymond RJ, Hinderliter AL, Willis PW, et al. *J Am Coll Cardiol* 2002; 39:1214–1219; [5] Eysmann SB, Palevsky HI, Reichek N, et al. *Circulation* 1989; 80:353–360.