

# **REVIEW PAPER**

# Echocardiographic Evaluation of Right Ventricular Function

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## Introduction

To allow optimal patient management an ideal measure of right ventricular performance should give an accurate objective assessment of function, be practicable in everyday clinical use and provide accurate prognostic information. This review will provide the practising clinician with an understanding of the advantages and disadvantages of the available echocardiographic techniques and aims to give some guidance on the most useful methods in specific clinical circumstances. The use of non-echocardiographic methods will also be discussed.

# Relevance of Right Ventricular Function

The right ventricle is a structurally and functionally complex chamber whose importance has been neglected previously. It propels systemic venous blood returning from the right atrium through the pulmonary vascular bed and maintains haemodynamic stability. In clinical practice right ventricular dysfunction is relevant in a variety of conditions.

# Chronic Lung Disease

To the practising clinician this is a common presentation of chronic right ventricular dysfunction. In both chronic obstructive and parenchymal pulmonary disease right ventricular dysfunction is associated with limited peripheral oxygen delivery and exercise capacity<sup>[1–3]</sup>. It has an important bearing on prognosis<sup>[4]</sup> that is independent of

other factors such as severity of airflow obstruction<sup>[5]</sup> and probably reflects the inter-relationship between it and deranged pulmonary haemodynamics. This group of patients usually have mild to moderate pulmonary hypertension that may increase considerably during exercise or pulmonary exacerbations. The term right ventricular dysfunction is probably a misnomer here. It is important to understand that the right ventricular hypertrophy and dilation that occurs in these patients is a beneficial adaptation allowing the ventricle to cope with an increased afterload and maintain a normal cardiac output<sup>[6]</sup>. Studies have shown right ventricular contractility to be preserved or even increased in these patients<sup>[7-9,10]</sup> and 'true' right ventricular failure only occurs during times of worsening hypoxia and hence increased afterload stress. The only treatment shown to be of be of prognostic benefit in these patients is longterm oxygen therapy which acts to reduce hypoxia and hence pulmonary artery pressure and afterload on the right ventricle.

### Primary Pulmonary Vascular Disease

In primary pulmonary hypertension the clinical features and mortality reflect the associated cardiac dysfunction manifest by progressive right ventricular pressure overload with hypertrophy and chamber dilatation. In the National Prospective Registry, pulmonary artery pressure, right atrial pressure and cardiac index measured at baseline were the best predictors of mortality confirming the important role of right ventricular performance in prognostic assessment<sup>[11]</sup>. The vasodilator prostacyclin improves echocardiographic indices of right ventricular function which correlate with the improvement in quality of life and survival<sup>[12]</sup>.

#### Pulmonary Thrombo-embolic Disease

In the case of acute massive pulmonary embolism, right ventricular dilation reflects the acute rise in pulmonary

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vascular resistance. Right ventricular failure causes a decrease in left ventricular pre-load which contributes to the fall in cardiac output. The increase in right ventricular afterload leads to higher right ventricular oxygen demand and ischaemia, which may even culminate in biochemically detectable infarction<sup>[13]</sup>. There is a relationship between the degree of right ventricular overload and the proportion of the pulmonary vascular bed occluded as measured by the extent of perfusion defects<sup>[14]</sup> and assessment of right ventricular function in patients with acute pulmonary embolism allows risk stratification with right ventricular dysfunction being a marker for both in-hospital and mediumterm mortality<sup>[15-17]</sup>. If a patent foramen ovale is demonstrated further information is gained<sup>[18]</sup>. Both thrombolytic treatment<sup>[19]</sup> and surgical thromboendarterectomy<sup>[20]</sup> are associated with a significant reduction in right ventricular pressure overload with a fall in right ventricular size and improvement in systolic function.

### Myocardial Infarction

Right ventricular dysfunction is present in at least one third of patients with inferior myocardial infarction and is associated with a significant increase in mortality<sup>[21]</sup>. Cardiogenic shock and the requirement for temporary transvenous cardiac pacing are more common in patients with right ventricular dilatation. Furthermore, by implying multivessel coronary artery disease, the presence of right ventricular dysfunction carries an adverse prognosis irrespective of infarct location. The demonstration of right ventricular dysfunction is important because it is often associated with a distinct clinical syndrome requiring specific management. In the presence of low cardiac output volume loading to restore left ventricular filling pressure is required. Inappropriate vasodilator and/or diuretic therapy may prove fatal. Information about right ventricular function can be applied in thrombolysis decision making when a relative contraindication is present. In fact, the findings of one study suggested that patients with inferior myocardial infarction derived no benefit from thrombolysis in the absence of right ventricular involvement<sup>[22]</sup>. Cardiogenic shock commonly develops when ventricular septal rupture complicates myocardial infarction. In these circumstances shock appears to be more related to impairment of right ventricular function than left ventricular function, territory of myocardial infarction or the extent of coronary disease<sup>[23]</sup>.

#### Congenital Heart Disease

Congenital heart disease is assuming greater importance in view of the increasing number of children surviving into adulthood, leading to a cohort of adult patients with complex and challenging cardiac morphology. Assessment of right ventricular morphology, function and connections is important in the diagnosis and management of this group of patients. The right ventricle is frequently subjected to the effects of both pressure and volume overload, which is relevant to both the native lesion and following corrective procedures. In pulmonary stenosis and tetralogy of Fallot the degree of the right ventricular outflow tract obstruction partly determines the severity and onset of symptoms. While tricuspid atresia and tricuspid stenosis result in a rudimentary right ventricle assessment of right ventricular area in Ebstein's anomaly has been shown to provide prognostic information<sup>[24]</sup>. It is widely accepted that closure of an atrial septal defect will prevent progressive right ventricular dilatation and dysfunction.

# Cardiopulmonary Transplantation

Assessment of right ventricular function is also relevant to the orthotopic heart transplant recipient. In the early postoperative period an elevated pulmonary vascular resistance may produce right ventricular dilatation. Although this pattern tends to improve progressively<sup>[25]</sup> right ventricular failure is responsible for some early deaths and many survivors are left with residual right ventricular dilatation. The importance of recognizing early right ventricular dysfunction has increased with the recent development of more advanced treatment strategies such as inhaled nitric oxide<sup>[26]</sup>. Recent work has highlighted the possibility that donor brain deathrelated myocardial injury could contribute to early postoperative right ventricular dysfunction<sup>[27]</sup>. Tricuspid regurgitation is the most common valvular abnormality after cardiac transplantation and the interaction with right ventricular function is complex, the effects on afterload making an accurate assessment difficult. Measurements of right ventricular function in the setting of single lung transplantation have focused mainly on evaluating the haemodynamic effects of therapy. Echocardiography can be used to follow the progress of the right ventricle after lung transplantation<sup>[28]</sup> and a number of studies have confirmed reversal of the preoperative progressive right ventricular dilatation and dysfunction. The absence of an improvement in right ventricular function following single lung transplantation is likely to be an important predictor of an adverse prognosis<sup>[29]</sup>.

# Important Considerations

## Right Ventricular Diastolic Function

Diastole is the period of ventricular relaxation incorporating periods of isovolumetric relaxation and early and late diastolic filling. Impaired diastolic function with relative preservation of systolic function is an early feature of right and left ventricular disease. The relevant echocardiographic methods will be described in the section on Doppler. Right ventricular diastolic dysfunction has been found to occur in patients with chronic pulmonary disease and pulmonary thromboembolism<sup>[30,31]</sup>, systolic left ventricular failure<sup>[32]</sup> and systemic sclerosis<sup>[33]</sup>.

### Ventricular Interdependence

The term ventricular interdependence has been used to describe dysfunction of one ventricle due to an effect on ventricular septal motion secondary to structural and/or functional abnormalities of the opposite ventricle. Ventricular interdependence may occur in systole or diastole and the timing of differences in pressure gradient between the ventricles is probably at least as important as the mechanical effects on the septum. Right ventricular diastolic dysfunction has been shown to occur in patients with left ventricular dysfunction without other causes of right ventricular disease or concurrent pulmonary hypertension implicating ventricular interdependence as a mechanism<sup>[32,34–36]</sup>. Conversely, it has long been recognized that when there is right-sided volume or pressure overload there may be a mechanical effect on the interventricular septum that can alter left ventricular systolic or diastolic function<sup>[37]</sup>. This has been observed in chronic obstructive pulmonary disease<sup>[36]</sup>, primary pulmonary hypertension<sup>[38]</sup> and atrial septal defect<sup>[37]</sup> and right ventricular infarction. The effect on LV function and clinical consequences may vary between individuals with similar degrees of right ventricular volume or pressure overload.

# Problems With Non-invasive Assessment of the Right Ventricle

A number of factors contribute to the complexities of estimating right ventricular function. Whilst the left ventricular cavity approximates to an ellipsoid model in healthy individuals the right ventricle is considerably more complex. The main body of the chamber is crescentic and truncated with separate inflow and outflow portions. The outflow portion or infundibulum may account for up to 25% of the total right ventricular volume. The fact that the chamber poorly approximates to any convenient geometric model means that volume calculated with these models only crudely represents true volume<sup>[39-41]</sup>. Marked regional differences exist in the extent of fibre shortening and contribution to stroke volume between different components of the right ventricular with the contraction-relaxation sequence of the inflow portion preceding that of the infundibulum<sup>[42]</sup>. The inaccessibility of the right ventricle behind the sternum often leads to inadequate image quality by conventional imaging modalities and this is particularly pertinent in patients with chronic pulmonary disease, a group in whom right ventricular dysfunction may be present. In addition, the problem of accurately locating

the endocardial boundary of the anterior wall of the chamber is compounded by a variable trabeculation pattern with the apical component having much coarser trabeculations than the corresponding zone of the left ventricle. Chamber orientation varies considerably between patients, particularly in those with right ventricular pressure or volume overload. In addition to myocardial function the shape and performance of the right ventricle depends on extrinsic factors such as pre-load, afterload and left ventricular performance. A limitation of conventional imaging methods in clinical practice is that these factors are frequently disregarded.

# Echocardiography of the Right Ventricle

### Early Methods

Attempts to measure right ventricular dimensions echocardiographically were made soon after the development of ultrasound<sup>[43]</sup>. Louridas<sup>[44]</sup> showed that M-mode echocardiography of right ventricular internal dimensions was significantly different between normal individuals and patients with cor pulmonale. M-mode measurements of right ventricular dimensions are, however, frequently not feasible<sup>[28]</sup> and limitations became apparent, in particular the fact that the dimensions were so heavily dependent on the angle traversed by the ultrasound beam across the ventricle.

#### Two-dimensional Imaging

Measurement of long and short-axis dimensions of right heart casts by two-dimensional imaging was quickly shown to be more accurate than M-mode techniques<sup>[45]</sup>. Two-dimensional imaging allows estimation of right ventricular dimensions, shape and wall thickness. In normal subjects, feasibility for assessment of right ventricular dimensions is high, with low inter- and intraobserver variability<sup>[46]</sup>. In patients with pulmonary disease, feasibility of two-dimensional imaging has been variable between studies<sup>[47–49]</sup> but developments in ultrasound technology, such as harmonic imaging and improved endocardial border definition, have further improved feasibility. An eccentricity index, which is a measure of the degree of septal displacement, has been described<sup>[50]</sup>. It is calculated as the ratio of the minor axis diameter of the left ventricle parallel to the septum to that perpendicular to it. Measuring this index at end-systole and end-diastole can provide a means of differentiating between right ventricular pressure and volume overload. Its clinical validity has been verified<sup>[12]</sup>. Calculation of right ventricular area based on single plane echocardiographic methods correlate with right ventricular ejection fraction<sup>[28,51]</sup> but assume a constant relationship between the dimensions of the

|                                  | Parameter  |  |
|----------------------------------|--|--|
| Chronic pulmonary disease        | RV dimensions<br>TR sPAP<br>RV time intervals (incl. IMP)*   |  |
| Pulmonary thromboembolic disease | Trans-tricuspid Doppler for diastolic function<br>RV/PA/IVC dimensions*<br>TR sPAP<br>RV time intervals  |  |
| Primary pulmonary hypertension   | Thrombus in transit*<br>Ecc. Ind.<br>RV dimensions*<br>TR sPAP<br>IMP*   |  |
| RV involvement in infarction     | Ecc. Ind.<br>+ve dp/dt<br>RV dimensions*<br>LVEF*<br>TR  |  |
| Left heart failure               | TR sPAP*   |  |
| Congenital heart disease†        | Trans-tricuspid Doppler for diastolic function<br>Doppler tissue imaging of tricuspid annulus*<br>RV dimensions<br>RA/RV/PA/TV/PV morphology and relations<br>Assessment of shunts |  |
| Heart transplantation            | IMP<br>RV dimensions‡*<br>TR§  |  |

Table 1. Relevant echocardiographic parameters in specific clinical conditions.

TR sPAP, systolic pulmonary artery pressure derived from Doppler assessment of tricuspid regurgitant jet velocity; IMP, index of myocardial performance; PA, pulmonary artery; IVC, inferior vena cava; Ecc. Ind., eccentricity index; +ve dp/dt, rate of right ventricular pressure rise; LVEF, left ventricular ejection fraction; TR, presence of tricuspid regurgitation on Doppler; RA, right atrium; TV, tricuspid valve; PV, pulmonary valve.

\* indicates variable has been shown to be of prognostic value.

† role of echocardiography in congenital heart disease heavily related to specific defect.

*t* early post-operative period.

§ late post-operative period.

|| transoesophageal imaging of incremental benefit.

ventricle in two planes. Estimation of right ventricular volume is the theoretical ideal but is based on the biplane approach requiring perpendicular echocardiographic planes, and standardization of views is difficult<sup>[41,52,53]</sup>. A combination of apical four-chamber and subcostal right ventricular outflow tract views is the most used<sup>[41]</sup>. Many geometric models have been applied to the different ventricular views to estimate right ventricular volume and a variety of these formulae have been directly compared in patients with cor pulmonale<sup>[49]</sup>. Simpson's method, which calculates volume based on a summation of the volume of individual slices, appears to be the best approach.

The value of two-dimensional echocardiography has perhaps been most clearly demonstrated in patients with pulmonary embolism<sup>[17,54]</sup>. In this setting the feasibility is high. The echocardiographic features may be nonspecific or demonstrate dilatation of the proximal pulmonary arteries or disturbance of flow velocity in pulmonary Doppler recordings. Presence of thrombus in transit is required before other causes of right ventricular dilatation can be excluded, but normal right ventricular morphology is highly unlikely with a haemodynamically significant pulmonary embolism<sup>[16]</sup>. Transoesophageal echocardiography has improved sensitivity and specificity compared with the transthoracic approach<sup>[55]</sup>. Specific echocardiographic features in other conditions are shown in Tables 1 and 2.

# Doppler Echocardiography

With the introduction of Doppler echocardiography other parameters have been examined as measures of right ventricular performance. The widest application of Doppler to the right heart has been to estimate pulmonary artery systolic pressure by measuring the peak velocity of a tricuspid regurgitant jet and applying the modified Bernoulli equation<sup>[56]</sup>. Although the relationship between pulmonary artery pressure and right ventricular function is complex and remains to be adequately elucidated, Doppler ultrasound is reliable in the detection of tricuspid regurgitation and a clearly defined continuous wave Doppler profile can be recorded in most patients. The application of

|                                       | Normal range                      | In disease                                  | Pathology  |
|---------------------------------------|-----------------------------------|---|------------|
|                                       |                                   |   |            |
| RVEDD (mm)                            | $27.1 \pm 4.1^{[31]}$             | $38.0 \pm 8.7^{[31]}$                       | COPD+PH    |
|                                       |                                   | $35.6 \pm 6.2^{[102]}$                      | PE         |
| RVEDA                                 | $10.0 \pm 0.4 \text{ cm}^{2[12]}$ | $21.2 \pm 0.9 \text{ cm}^2/\text{m}^{[12]}$ | PPH        |
|                                       |                                   | $22 \pm 4 \text{ cm}^{2[103]}$              | RLD        |
|                                       |                                   | $24 \pm 10 \text{ cm}^2/\text{m}^{[29]}$    | Various PH |
|                                       |                                   | $31.3 \pm 7.3 \text{ cm}^{2[104]}$          | PPH        |
| RVESA                                 |                                   | $18 \pm 8 \text{ cm}^2/\text{m}^{[29]}$     | Various PH |
|                                       |                                   | $25.4 \pm 7.0 \text{ cm}^{2[104]}$          | PPH        |
| Change RVA (%)                        | $41.5 \pm 1.2^{[12]}$             | $18.1 \pm 1.4^{[12]}$                       | PPH        |
|                                       |                                   | $24 \pm 14^{[29]}$                          | Various PH |
|                                       |                                   | $19.7 \pm 6.5^{[104]}$                      | PPH        |
|                                       |                                   | $24 \pm 7^{[105]}$ ‡                        | RVMI       |
|                                       |                                   | $32 \pm 5^{[105]}$                          | RVMI       |
| RVEF (%)                              | $67 \pm 5^{[106]}$                | $28.3 \pm 8.1^{[104]}$                      | PPH        |
|                                       |                                   | $42 \pm 12^{[80]}$                          | CHD        |
| PEP (msecs)                           | $110 \pm 18^{[31]}$               | $109 \pm 19^{[107]}$                        | PPH        |
|                                       |                                   | $129 \pm 27^{[31]}$                         | COPD+PH    |
| AT (msecs)                            | $154 \pm 19^{[31]}$               | $93 \pm 15^{[31]}$                          | COPD+PH    |
|                                       | $121 \pm 34^{[91]}$               | $90 \pm 22^{[91]}$                          | Adult CF   |
|                                       |                                   | $56 \pm 15^{[108]}$                         | PE         |
|                                       |                                   | $68.1 \pm 26.9^{[104]}$                     | PPH        |
| Systolic Ecc. Ind.                    | $1.01 \pm 0.01^{[12]}$            | $1.70 \pm 0.07^{[12]}$                      | PPH        |
| Diastolic Ecc. Ind.                   | $1.00 \pm 0.01^{[12]}$            | $1.91 \pm 0.07^{[12]}$                      | PPH        |
| E:A ratio                             | $1.5 \pm 0.3^{[68]}$              | $1.3 \pm 0.6^{[68]}$                        | Amyloid    |
|                                       |                                   | $1.4 \pm 1.1^{[104]}$                       | PPH        |
| Edt (msecs)                           | $188 \pm 22^{[70]*}$              |   |            |
|                                       | $198 \pm 23^{[70]}$ †             |   |            |
|                                       | $225 \pm 28^{[68]}$               | $163 \pm 41^{[68]}$                         | Amyloid    |
|                                       | $139.8 \pm 13.8^{[30]}$           | $154.4 \pm 19.6^{[30]}$                     | COPD/PTE   |
| IVRT (msecs)                          | $34.1 \pm 25.0^{[31]}$            | $65.0 \pm 20.8^{[31]}$                      | COPD+PH    |
| TYTET (Insees)                        | $38 \pm 7^{[74]}$                 | $74 \pm 33^{74}$                            | PPH        |
|                                       | $49 \pm 9^{[107]}$                | 71200                                       |            |
| HVF (%)                               |                                   |   |            |
| Inspiration                           | $14 \pm 7^{[68]}$                 | $36 \pm 27^{[68]}$                          | Amyloid    |
| Expiration                            | $21 \pm 9^{[68]}$                 | $50 \pm 27$<br>$59 \pm 38^{[68]}$           | Amyloid    |
| TLA excursion (cm)                    | $2.2 \pm 0.4^{[91]}$              | $1.6 \pm 0.4[^{91}]$                        | Adult CF   |
| Sa (cm/sec)                           | $15.5 \pm 2.6^{[92]}$             | $10.3 \pm 2.6^{[92]}$                       | HF         |
| Ea (cm/sec)                           | $15.6 \pm 3.9^{[92]}$             | $10.9 \pm 2.0$<br>$10.8 \pm 2.5^{[92]}$     | HF         |
| Aa (cm/sec)                           | $15.4 \pm 4.5^{[92]}$             | $10.0 \pm 2.9$<br>$15.0 \pm 4.9^{92}$       | HF         |
| IMP                                   | $0.28 \pm 0.04^{[107]}$           | $0.89 \pm 0.25^{[107]}$                     | PPH        |
|                                       | 0 20 2 0 0 0                      | $0.49 \pm 0.12^{[72]}$                      | Ebstein's  |
|                                       |                                   | $0.49 \pm 0.12$<br>$0.51 \pm 0.23^{[4]}$    | COPD+RLD   |
| +ve dp/dt (mmHg/sec)                  | $255 \pm 17.5^{[60]}$             | $728 \pm 265^{[109]}$                       | PPH        |
| · · · · · · · · · · · · · · · · · · · | 255 ± 17 5                        | 120 ± 200                                   | 1111       |

Table 2. Echocardiographic measures of RV function used in previous studies

RVEDD, right ventricular end-diastolic diameter; RVEDA, right ventricular end-diastolic area; RVESA, right ventricular end-systolic area; RVA, right ventricular area; RVEF, right ventricular ejection fraction; PEP, pre-ejection period; AT, acceleration time; E:A ratio, ratio of early to late diastolic filling velocity; Edt, deceleration time of early diastolic filling; IVRT, isovolumic relaxation time; HVF, reversal of flow as a proportion of total forward flow in hepatic vein; TLA, tricuspid long axis; Sa, peak systolic tricuspid annular velocity; Ea, peak early diastolic tricuspid annular velocity; Aa, peak late diastolic tricuspid annular velocity; COPD, chronic obstructive pulmonary disease; PH, pulmonary hypertension; RLD; restrictive lung defect; PPH, primary pulmonary hypertension; Various PH, pulmonary hypertension of various aetiologies; RVMI, right ventricular involvement in myocardial infarction; CHD, congenital heart disease; PE, acute pulmonary embolism; PTE, chronic pulmonary thromboembolism; CF, cystic fibrosis; Ebsteins, Ebsteins anomaly.

\* subjects <50yrs.

† subjects >50yrs.

‡ failed reperfusion.

§ successful reperfusion.

|| study using radionuclide vetriculography.

echocardiographic contrast agents has further improved feasibility<sup>[57]</sup>. The necessary estimation of right atrial pressure can be facilitated by a variety of means includ-

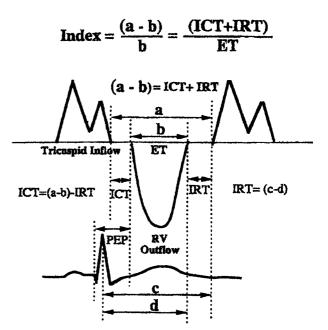
ing clinical examination<sup>[56]</sup>, tricuspid inflow parameters<sup>[58]</sup> and inferior vena caval measurements<sup>[59]</sup>. The tricuspid regurgitant profile on continuous wave Doppler can also be used to derive the rate of right ventricular pressure rise in early systole which is a measure of right ventricular systolic function although measurements are influenced by pulmonary artery pressure<sup>[60]</sup>.

Systolic time intervals such as duration of the right ventricular pre-ejection period and ratio of the preejection period to the right ventricular ejection time have been investigated and found to correlate with similar intervals derived from two-dimensional images and right ventricular ejection fraction<sup>[61-63]</sup>. More recently pulmonary artery flow acceleration time has been investigated. Acceleration time is shortened in patients with pulmonary artery hypertension who may have a characteristic flow morphology with a steep acceleration time and a slower deceleration phase with mid-systolic notching<sup>[64,65]</sup>. Correlation between acceleration time and pulmonary artery pressure varies between studies but while acceleration time may not be fully reliable for accurate pulmonary artery pressure predictions it has been found to be of prognostic value in patients with chronic lung disease<sup>[66]</sup>.

Diastolic Function: Evaluation of right ventricular diastolic function is conventionally based on the Doppler transtricuspid flow velocity profile. Variables measured include peak velocity of early filling (E velocity), peak velocity of late filling due to atrial contraction (A velocity), E/A ratio and deceleration time of early filling (Edt). The velocities across the tricuspid valve are significantly lower than across the mitral valve and tricuspid Edt is longer than mitral Edt. The tricuspid flow parameters do not appear to be as affected by age but respiration causes pronounced variability<sup>[67]</sup> and measurement should be made at end-tidal or endexpiratory apnoea. The normal filling pattern is affected by pre-load, afterload and compliance of the ventricular myocardium. In patients with pulmonary hypertension hypertrophic changes in the right ventricle may lead to an alteration in compliance with slowing of early right ventricular filling and augmentation of filling at atrial contraction. Edt is prolonged<sup>[34]</sup>. Although abnormal relaxation may be observed in patients with infiltrative/ restrictive myocardial disease, restrictive right ventricular filling with a short tricuspid Edt may occur if disease is advanced<sup>[68,69]</sup>.

Relevant information can also be derived by measuring the right ventricular isovolumic relaxation time or from pulsed wave Doppler assessment of superior vena caval and hepatic venous flow. Right ventricular isovolumic relaxation time is the period from pulmonary valve closure to tricuspid valve opening and is prolonged with abnormal relaxation and shortened with rapid relaxation or increased filling pressures. As could be predicted, changes in superior vena caval and hepatic venous flow also occur during the respiratory cycle, both in health<sup>[70]</sup> and disease<sup>[68]</sup>, with significant variations as a result of changes in heart rate and loading conditions.

Doppler Index of Myocardial Performance: There has been considerable recent interest in the application of this index initially described by Tei<sup>[71]</sup> to the right



**Figure 1.** Schematic representation of Doppler intervals for derivation of IMP. Index ([a-b]/b) is calculated by measuring two intervals: (1) a is interval between cessation and onset of tricuspid inflow and (2) b is ejection time (ET) of right ventricular outflow.

ventricle. The index of myocardial performance encompasses important energy dependent periods of systolic contraction, ejection and diastolic relaxation. The use of Doppler offers the possibility of high feasibility in patients with poor image quality and accurate characterisation of function in the context of complex chamber geometry. Calculation of the index of myocardial performance is based on measurement of Dopplerderived time intervals. In the case of the right ventricle relevant intervals are measured from the tricuspid inflow and pulmonary ejection profiles in the apical four chamber and parasternal short axis views respectively. The duration of the time intervals is identified by recording the time between the onset and cessation of blood flow on the Doppler recording. Figure 1 demonstrates how the index of myocardial performance is calculated. The isovolumic contraction time increases in systolic dysfunction and the right ventricular ejection time decreases. Most abnormalities of diastolic function are manifested in an abnormally slow rate of pressure decline. These changes are reflected in a prolongation of the isovolumic relaxation time. As global myocardial dysfunction progresses, therefore, the value for the index of myocardial performance increases, due to changes in all three time interval components used for its calculation. The clinical utility of the index of myocardial performance has been validated in congenital heart disease<sup>[72,73]</sup>, primary pulmonary hypertension<sup>[74]</sup> and chronic respiratory disease<sup>[4,75,76]</sup>. It has been shown to have prognostic relevance<sup>[4,74]</sup>. The potential problems with the index of myocardial performance are that it is invalidated by heart block and arrhythmias. Primary

**Table 3.** Echocardiographic developments facilitatingimproved assessment of right ventricular function.

| Systolic function                     | Three dimensional echocardiography<br>Automated border detection <sup>[42,82–85]</sup><br>Colour kinesis <sup>[86,87]</sup><br>Use of contrast agents <sup>[88]</sup><br>Doppler tissue imaging <sup>[89–92]</sup> |
|---------------------------------------|--|
| Diastolic function<br>Global function | Intracardiac echocardiography <sup>[93]</sup><br>Automated border detection <sup>[110]</sup><br>Doppler tissue imaging <sup>[111]</sup><br>Tei index <sup>[4,72–74]</sup>  |

valvular diseases also lead to problems in interpretation. The effect of changes in loading conditions on the index of myocardial performance are still the subject of debate. It is particularly important to validate local values for the index of myocardial performance before applying them in clinical practice.

### Three Dimensional Echocardiography

The advent of three-dimensional echocardiographic reconstruction removes the longstanding limitation imposed by standard imaging planes. Effectively, it eliminates the need for geometric assumptions of the complex right ventricular architecture and allows improved endocardial detection. The feasibility and reliability of three-dimensional echocardiographic imaging has been examined by in vitro<sup>[77,78]</sup> and in vivo<sup>[39,79]</sup> studies, and modern data collection and gating systems minimize artefactual errors due to subject motion or respiration. Three-dimensional echocardiographic measurements of right ventricular volumes correlate closely with those of magnetic resonance imaging and permit reliable serial measurements<sup>[80]</sup>. The accuracy and reproducibility of the technique improve the evaluation of right ventricular hypertrophy<sup>[81]</sup>. While three-dimensional echocardiography appears to be excellent for objective and accurate measurements of right ventricular geometry there is a paucity of information on its prognostic value, it is currently not widely available for routine use and time is required for post-acquisition reconstruction of the images.

# Recent Advances in Echocardiography of the Right Ventricle

Recently, the advent of more sophisticated imaging techniques has been of great benefit in more accurately quantifying right ventricular performance (Table 3). Automated border detection methods have been employed in the assessment of global right ventricular function in patients with a variety of underlying pathologies.<sup>[42,82–84]</sup>. Estimation of changes in right ventricular dimensions based on this technique are in close agreement with contrast angiographic data<sup>[85]</sup>. Colour kinesis can be used to quantitatively assess segmental

right ventricular function and has been shown to be of value in a variety of pathologies affecting the right ventricle with excellent correlation between ventriculographic and colour kinesis measurements<sup>[86,87]</sup>. Contrast echocardiography has been applied to the right ventricle in an attempt to overcome difficulties with endocardial border definition. In healthy subjects the correlation between echocardiographic and radionuclide derived right ventricular ejection fraction significantly increased and inter-observer variability improved with the addition of contrast<sup>[88]</sup>. Tricuspid annular motion can be assessed easily using Doppler tissue imaging<sup>[89,90]</sup>. An advantage is that measurement is independent of geometric assumptions and endocardial border tracing. This method has been accepted as a convenient means of quantitatively evaluating right ventricular systolic function and has been used as a diagnostic tool by a number of groups<sup>[89,91]</sup>. The sensitivity of tricuspid annular motion for the detection of early right ventricular dysfunction may be superior to more conventional imaging techniques and a recent study has suggested that reduced systolic annular velocity is highly predictive of right ventricular dysfunction as measured by radionuclide ventriculography<sup>[92]</sup>. Intra-cardiac echocardiography is able to accurately delineate the entire right ventricular architecture in the invasive catheterization laboratory setting. Using a sequential catheter pull-back technique it has been demonstrated that right ventricular volume and function could be accurately assessed compared to a directly measured standard model<sup>[93]</sup>.

# Alternatives to Echocardiographic Evaluation of the Right Ventricle

All non-invasive methods of evaluating right ventricular structure and function have inherent limitations. Studies evaluating the usefulness of electrocardiography have been directed predominantly towards the detection of right ventricular hypertrophy and had low sensitivity and specificity<sup>[94–96]</sup>. Angiocardiographic estimation of right ventricular volume is invasive and is not widely performed. Accurate measurements are reliant on the selection of appropriate orthogonal planes<sup>[97]</sup> and accurate border tracing. A large variety of geometric figures have been used and many overestimate true volume probably due to problems with inclusion of right ventricular myocardium in heavily trabeculated regions of the ventricle. The use of radionuclide techniques to evaluate right ventricular function has been reviewed elsewhere<sup>[98]</sup>. Both first pass and equilibrium techniques have been applied. Each have limitations and it is unlikely that the favourable mean inter-observer and inter-study variabilities for the estimation of left ventricular ejection fraction are applicable to the right ventricle<sup>[99]</sup>. Magnetic resonance imaging provides a method of accurately visualizing the complex internal architecture of the right ventricular cavity<sup>[100]</sup> and offers fast data acquisition. Time-volume curves of the right

ventricle can be used to assess both systolic and diastolic function<sup>[101]</sup>. It is now generally accepted that magnetic resonance imaging measurements are the gold standard for non-invasive estimation of right ventricular volume and wall mass but, unfortunately, availability is still limited and data is lacking on its prognostic value and use for serial assessment of right ventricular function.

#### Conclusion

Echocardiography provides a readily accessible tool for the evaluation of right ventricular function. It remains the first line investigation because of its ability to provide comprehensive information on right ventricular size, structure and function. Recent developments in ultrasound technology have overcome the limitations of simple M-mode and two-dimensional imaging and facilitated more accurate monitoring of disease progression. Competition from quantitative, highly reproducible techniques is emerging. Magnetic resonance imaging has become the gold standard technique for evaluation of right ventricular volume and function but a balance needs to be struck between techniques which are available, accurately reflect right ventricular performance and are of proven prognostic value.

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