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The Right Ventricle in Cardiac Surgery, a Perioperative Perspective: I. Anatomy, Physiology, and Assessment

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The importance of right ventricular (RV) function in cardiovascular disease and cardiac surgery has been recognized for several years. RV dysfunction has been shown to be a significant prognostic factor in heart failure, congenital heart disease, valvular disease, and cardiac surgery. In the first of our two articles, we will review key features of RV anatomy, physiology, and assessment. In the first article, the main discussion will be centered on the echographic assessment of RV structure and function. In the second review article, pathophysiology, clinical importance, and management of RV failure in cardiac surgery will be discussed.

This article has supplementary material on the Web site: www.anesthesia-analgesia.org.

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myocardium, and the outflow region (infundibulum) (Fig. 1). In hearts with congenital malformations, one or more of the three components may be rudimentary or absent. Table 1 summarizes key anatomical and physiological features of the RV and LV.

**RV PHYSIOLOGY**

The primary function of the RV is to receive systemic venous return and pump it into the pulmonary system. Under normal conditions the RV, in contrast to the LV, is coupled to a low pressure, highly distensible arterial system. The RV is normally connected in series with the LV. In the absence of shunt physiology or significant valvular regurgitation, the stroke volume of the RV will normally match that of the left. Because of the greater end-diastolic volume of the right ventricle, RV ejection fraction (RVEF) is lower than the left. The greater end-diastolic volume of the right ventricle, RV ejection fraction (RVEF) is lower than the LV, is coupled to a low pressure, highly distensible arterial system. In the presence of normal pulmonary circulation, the RV performs approximately one-fourth of the LV stroke work. Several factors modulate PVR, including hypoxia, hypercarbia, cardiac output, pulmonary volume and pressure, and specific molecular pathways, most prominent being the nitric oxide pathway (vasodilation), the prostaglandin pathway (vasodilation), and the endothelin pathway (vasoconstriction). Pulmonary vessels constrict with hypoxia (Euler-Liljestrand reflex) and relax in the presence of hyperoxia. In some instances, hypercarbia may also be a strong pulmonary vasoconstrictor.

Lung volumes have a differential effect on intrapulmonary vessels which accounts for the unique U-shaped relationship between lung volume and PVR. PVR is minimal at functional residual capacity and increased at large and small lung volumes alike (Fig. 4), this may be observed clinically when hyperinflation of the lungs greatly increases PVR. Application of high levels of positive end-expiratory pressure may narrow the capillaries in the well ventilated lung areas and divert flow to less well ventilated or nonventilated areas, potentially leading to hypoxia. An increase in cardiac output distends open vessels and may recruit previously closed vessels decreasing PVR. Regional blood flow to the lungs is also influenced by gravity, where pulmonary blood flow is greater in the dependant areas of the lung.

Ventricular interdependence refers to the concept that through direct mechanical interactions the size, shape, and compliance of one ventricle may affect the size, shape, and pressure-volume relationship of the other. The main anatomical determinants for the RV is also sequential, starting with the trabeculated myocardium and ending with the contraction of the infundibulum (normally separated by approximately 25–50 ms). To better understand the complex relationship between RV contractility, preload, and afterload, many investigators have studied the pressure-volume relationship of the RV (Fig. 2). One of their major findings was that the RV follows a time-varying elastance model in which ventricular elastance is described by the relationship between systolic pressure and volume under variable loading conditions. Many studies have shown that RV elastance may also be approximated by a linear relationship. RV maximal elastance is considered by many investigators to be the most reliable index of RV contractility. RV systolic elastance is lower than that of the LV. This arrangement implies that the RV is far more sensitive to increases in afterload. This can be illustrated in the acute setting, where RV stroke volume decreases significantly after an increase in pulmonary arterial pressure (Fig. 3). The pulmonary circulation is an important determinant of RV afterload. The pulmonary vascular bed is a highly compliant, low-pressure, low-resistance system. In hearts with congenital malformations, one or more of the three components may be rudimentary or absent. RV maximal elastance is considered by many investigators to be the most reliable index of RV contractility. RV systolic elastance is lower than that of the LV. This arrangement implies that the RV is far more sensitive to increases in afterload. This can be illustrated in the acute setting, where RV stroke volume decreases significantly after an increase in pulmonary arterial pressure (Fig. 3). The pulmonary circulation is an important determinant of RV afterload. The pulmonary vascular bed is a highly compliant, low-pressure, low-resistance system. In the presence of normal pulmonary circulation, the RV performs approximately one-fourth of the LV stroke work. Several factors modulate PVR, including hypoxia, hypercarbia, cardiac output, pulmonary volume and pressure, and specific molecular pathways, most prominent being the nitric oxide pathway (vasodilation), the prostaglandin pathway (vasodilation), and the endothelin pathway (vasoconstriction). Pulmonary vessels constrict with hypoxia (Euler-Liljestrand reflex) and relax in the presence of hyperoxia. In some instances, hypercarbia may also be a strong pulmonary vasoconstrictor.
Ventricular interdependence include the ventricular septum, the pericardium, and continuity between myocardial fibers of the RV and LV. Ventricular interdependence may occur in both systole and diastole. Although always present, ventricular interdependence is most evident with changes in loading conditions, such as those observed during respiration or sudden postural changes. Ventricular interdependence also plays an important part in the pathophysiology of RV dysfunction.

PERIOPERATIVE ASSESSMENT OF THE RV
Overview

In cardiac surgery, right heart catheterization and echocardiography play an essential and complementary role in the assessment of RV structure and function. Both techniques provide useful information that may help tailor the anesthetic and surgical approach and provide guidance in the management of hemodynamically unstable patients. Hemodynamically, RV dysfunction or failure is usually recognized in the presence of a right atrial pressure (RAP) ≥8–10 mm Hg or a RAP to pulmonary capillary wedge pressure ≥0.8 (isolated RV failure) and/or a low cardiac index (≤2.2 L · min⁻¹ · m⁻²). Increasing RAP may also

Table 1. Characteristics of Right and Left Ventricle

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Right ventricle</th>
<th>Left ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structure</td>
<td>Inflow region, trabected myocardium, infundibulum</td>
<td>No infundibulum mitro-aortic continuity</td>
</tr>
<tr>
<td>Shape</td>
<td>From the side: triangular cross-section: crescentic</td>
<td>Elliptic</td>
</tr>
<tr>
<td>Volume (end-diastolic)</td>
<td>49–101 mL/m²</td>
<td>44–89 mL/m²</td>
</tr>
<tr>
<td>Mass (g/m²)</td>
<td>&lt;35 g/m² ≈ 1/6 LV mass</td>
<td>&lt;130 g/m² (men) &lt;100 g/m² (women)</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>40%–68% &gt; 45%</td>
<td>57%–74% &gt; 50%</td>
</tr>
<tr>
<td>Ventricular elastance</td>
<td>1.30 ± 0.84</td>
<td>5.48 ± 1.23</td>
</tr>
<tr>
<td>Ventricular compliance</td>
<td>Higher compliance than LV</td>
<td>5.0 ± 0.52 × 10⁻²</td>
</tr>
<tr>
<td>Adaptation to disease</td>
<td>Better adaptation to volume overload states</td>
<td>Better adaptation to pressure overload states</td>
</tr>
</tbody>
</table>

Normal variables of the right (RV) compared with the left ventricle (LV). Range of normal values depends on method of acquisition.

a Lower value of normal RV and LV ejection fraction used in clinical practice. Adapted from Haddad et al.²³

Figure 2. Pressure-volume loops of the right ventricle under different loading conditions. The slope of maximum time-varying elastance ($E_{\text{max}}$) is displayed on the graph. Adapted from Dell'Italia et al.¹⁶

Figure 3. The response of the right and left ventricle to experimental increase in pressure or afterload. Adapted from MacNee et al.¹⁸

Figure 4. Relationship between lung volume and pulmonary vascular resistance (PVR). As lung volume is reduced or increased, the increase in PVR result from compression of the alveolar and extraalveolar vessels. RV = residual volume; FRC = functional residual capacity; TLC = total lung capacity. Adapted from Fischer et al.²⁰
be a sign of impeding RV failure. Significant RV outflow tract (RVOT) obstruction may also be suspected in the presence of RV-pulmonary artery gradient more than 25 mm Hg. As will be reviewed in this section, echocardiography also provides useful information on RV and pulmonary structure, valvular function and pericardial physiology. Four recently published guidelines may help to guide the echocardiographic assessment of the RV: 1) ASE/SCA (American Society of Echocardiography/Society of Cardiovascular Anesthesiologists) guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography (TEE) examination; 2) ASE/SCA guidelines for a comprehensive epicardial echocardiography examination; 3) the ASE and European Association of Echocardiography guidelines on

Figure 5. Upper esophageal (UE), mid-esophageal (ME), low esophageal (LE), transgastric (TG), and deep TG views useful in the evaluation of right ventricular (RV) function. Asc = ascending; Ao = aorta; AoV = aortic valve; CS = coronary sinus; IVC = inferior vena cava; LA = left atrium; LAX = long axis, LPA = left pulmonary artery; LV = left ventricle; MPA = main pulmonary artery; PV = pulmonic valve; RA = right atrium; RPA = right pulmonary artery; RV = right ventricle; RVOT = right ventricular outflow tract; SAX = short axis; SVC = superior vena cava; TV = tricuspid valve. Adapted from Shanewise et al. and Denault et al.

410 Right Ventricular Function ANESTHESIA & ANALGESIA
two-dimensional (2D) chamber quantification; and 4) the ASE recommendations for evaluation of the severity of native valvular regurgitation with 2D and Doppler echocardiography.12,24–26

Echocardiographic Views of the RV

In the operating room, both TEE and epicardial echocardiography are helpful in obtaining images of the RV. In the intensive care unit, TEE and trans-thoracic echocardiography (TTE) constitute useful modalities.

The most useful transesophageal views are illustrated in Figure 5.24 The midesophageal four-chamber view is ideal for visualizing the RV lateral wall and measuring RV internal dimensions and RV fractional area change (RVFAC) (Videos 1 and 2; please see video clips available at www.anesthesia-analgesia.org). The midesophageal views are also useful in visualizing the coronary sinus (Fig. 6), assessing tricuspid regurgitation (usually achieved at an angle of 30°–60°), and assessing potential atrial or ventricular septal defects. The transgastric views allow short-axis views (SAX) of the RV and septum, and views of the RV inflow tract and RVOT, inferior vena cava (IVC) as well as hepatic veins. The anterior and inferior walls of the RV are best visualized in the transgastric views. The great vessels are best studied in the upper esophageal views, whereas tricuspid annular tissue Doppler signals are best assessed in the deep transgastric RV views.

Epicardial echocardiography may be very helpful in the presence of a contraindication to TEE such as an esophageal stenosis, if the images obtained by TEE are suboptimal, for diagnosis of pulmonic valve pathology or for detection of intraoperative thromboembolism.27,28 Most of the imaging planes from TEE can be obtained using epicardial echocardiography (Fig. 7). However, the four recommended ASE views which provide helpful images of the RV include 1) the epicardial aortic valve (AoV) SAX view (TTE parasternal AoV SAX equivalent) (Fig. 7B, Video 3; please see video clips available at www.anesthesia-analgesia.org); 2) the epicardial LV long-axis (LAX) view (TTE parasternal LAX equivalent) (Fig. 7C, Video 4; please see video clips available at www.anesthesia-analgesia.org); and 3) the epicardial LV basal SAX view (TTE modified parasternal mitral valve basal SAX equivalent) (Fig. 7D); and 4) the epicardial RVOT view (TTE parasternal SAX equivalent) (Fig. 7E). Guidelines on this topic were published in 2007.26

Challenges in the Echocardiographic Study of the RV

The echocardiographic study of the RV is more challenging than that of the left. The main difficulties encountered may be explained by 1) the complex shape of the RV, 2) heavy apical trabeculations of the RV, which limits endocardial surface recognition, and 3) the marked load dependence of several indices of RV function.5 Despite these limitations, a comprehensive assessment of the RV may provide important insights into its contractility, preload, and afterload.

Identifying the Anatomic RV

Although the RV is usually on the right side of the heart and connects with the pulmonary artery, the anatomic RV is defined by its structure not by its position or connections. Features which help differentiate the anatomic RV from anatomic LV include 1) the more apical insertion point of the septal leaflet of the tricuspid valve relative to the anterior leaflet of the mitral valve, 2) the presence of a moderator band, 3) the presence of more than 2 papillary muscles, and 4) the trileaflet configuration of the tricuspid valve.1,29,30 This is especially important in CHD, where the anatomic RV may be positioned on the left side of the heart or connect to the aorta.3,30 In a corrected transposition of the great vessels (l-TGA), the anatomic RV is positioned on the left side of the heart and connects to the aorta (systemic ventricle). In a d-transposition of great arteries (d-TGA), the anatomic RV is positioned...
to the right side of the heart and connects to the aorta. Alternatively, the LV may show more pronounced trabeculations, which may mimic the structure of the anatomic RV (noncompaction of the LV).

RV Size and Shape

Because the complex shape of the RV does not lend itself to simple mathematical modeling, the assessment of RV size using 2D echocardiography remains challenging. The best correlations between 2D echocardiography and RV volumes have been obtained using the maximal SAX dimension and the RV area measured in the four-chamber view (Fig. 8).\(^5\)\(^,\)\(^12\)\(^,\)\(^31\) It is, however, important to note that there is significant overlap between normal patients and patients with RV volume overload.\(^12\) Furthermore, normal 2D values have not been well established in patients requiring mechanical ventilation. The availability of 3D TEE

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**Figure 7.** Epicardial views of the right ventricle. (A) Main, left and right pulmonary artery views; (B) Aortic valve (AoV) short axis (SAX) view (Video 3; please see video clips available at www.anesthesia-analgesia.org); (C) Left ventricular long-axis (LAX) view (Video 4; please see video clips available at www.anesthesia-analgesia.org); and (D) Left ventricular basal SAX view E. Right ventricular outflow tract view F. Right atrial and ventricular views. Figure B–E are the recommended views\(^26\) but views (A) and (E) can be used to complete the evaluation of the right ventricle. AL = anterolateral; Ao = aorta; AoV = aortic valve; ATV = anterior tricuspid leaflet; CS = coronary sinus; IVC = inferior vena cava; LA = left atrium; LPA = left pulmonary artery; LV = left ventricle; MPA = main pulmonary artery; MV: mitral valve; PM = posteromedial; PTV = posterior tricuspid valve; RA = right atrium; RPA = right pulmonary artery; RV = right ventricle; RVOT = right ventricular outflow tract; SVC = superior vena cava; TV = tricuspid valve.
technology may in the future allow better intraoperative assessment of RV volumes. At this time, however, software used for RV volume quantification is not routinely available. Figure 9 illustrates normal values of RVOT measurements.

RV hypertrophy is echocardiographically defined as a ventricular wall thickness more than 5 mm at end-diastole. The inferior or lateral walls of the RV are the preferred locations for measurement since, in contrast to the anterior wall, they are not invested with as much epicardial fat. The inferior wall of the RV is best assessed in transgastric views performed at 0°, whereas the RV lateral wall is best measured in the four-chamber view. Because the RV wall is thinner and more trabeculated than the LV wall, precise measurements are more difficult to obtain.

The RV shape cannot be described by one simple geometric shape. Under normal conditions, the RV appears triangular, when viewed from the side and crescentic in cross-section. The analysis of septal curvature may provide useful insights into RV pathology. The interventricular septum is usually curved (convex) toward the RV (Fig. 10). The eccentricity index, a measure of septal curvature, represents the ratio of the LV minor axis diameter (parallel to the septum) to its perpendicular axis. In normal subjects, the index is essentially one at both end-diastole and end-systole. In the adult with acquired pressure overload, the RV dilates early and the ventricular septum is displaced toward the LV cavity especially at end-systole. This will distort both RV and LV geometry (D-shaped LV; eccentricity index >1). In volume overloaded states, the RV is dilated and rotated clockwise (apical reference). The RV initial crescentic shape is transformed into a more cylindrical configuration, and the ventricular septum is displaced toward the LV cavity (D-shape LV; eccentricity index >1), mainly at end-diastole. In patients with congenital pulmonary stenosis, the RV has a greater hypertrophic response, and its shape is more elliptic; dilation occurs late in the course of the disease or in the presence of a critical stenosis. When aneurysms are seen in the RV, the possibility of arrhythmogenic RV dysplasia must be considered. In this condition, the aneurysms occur most commonly in the anterior infundibulum, basal inferior wall, and apex. Other causes of RV aneurysms include myocardial infarction and, in rare cases, absence of a right pericardium.

### Indices of RV Function

The study of RV function comprises indices that reflect RV systolic function, RV diastolic function, global and regional RV function, (systole and diastole) and valvular function. The study of RV dysynchrony is a new field of research and could play a larger role in the future. Indices of RV systolic function may describe the extent of RV contraction or reflect RV contractility (i.e., the intrinsic ability of the ventricle to contract). An ideal index of contractility would be independent of afterload and preload, sensitive to change in inotropic state, independent of...

<table>
<thead>
<tr>
<th>Reference range</th>
<th>Mildly abnormal</th>
<th>Moderately abnormal</th>
<th>Severely abnormal</th>
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<tr>
<td>RVD1, cm</td>
<td>2.0 - 2.8</td>
<td>2.9 - 3.3</td>
<td>3.4 - 3.8</td>
</tr>
<tr>
<td>RVD2, cm</td>
<td>2.7 - 3.3</td>
<td>3.4 - 3.7</td>
<td>3.8 - 4.1</td>
</tr>
<tr>
<td>RVD3, cm</td>
<td>7.1 - 7.9</td>
<td>8.0 - 8.5</td>
<td>8.6 - 9.1</td>
</tr>
</tbody>
</table>

Figure 8. Mid-esophageal four-chamber view (A, B) with the specific recommended measurements (C). The right ventricular diameter (RVD) 1 correspond to the tricuspid annulus, RVD2 to the minor axis and RVD3 to the major axis. BSA = body surface area; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; RVD = right ventricular diameter. Adapted from Lang et al.
The most commonly used echocardiographic indices of RV systolic function are summarized in Tables 2 and 3 and include:

1. Geometric indices, such as RVFAC, RVEF, and tricuspid annular plane systolic excursion (TAPSE).
2. Myocardial velocity indices, such as the tricuspid annular plane maximal systolic velocity and the isovolumic acceleration (IVA).
3. Hemodynamic indices, such as the RV first derivative of pressure and time (RV dP/dt).
4. Time interval indices, such as the RV myocardial performance index (RVMPI) or Tei index which reflect both systolic and diastolic parameters.

Indices of RV Systolic Function

RVEF represents the ratio of stroke volume to end-diastolic RV volume ([RVEDV-RVESV]/RVEDV). RVEF has the advantage of being a widely accepted and validated index of RV function. Its prognostic value has been proven in heart failure, valvular heart disease, and CHD. RVEF has, however, the disadvantage of being highly load dependent and may not always reflect ventricular contractility in volume or mass.
of RV ejection fraction (RVEF) using 2D echocardiography remain elusive. In patients with heart failure, a moderate correlation was noted between St velocity and RVEF \((r = 0.65, P < 0.001)\).\(^{46}\) Currently, its predictive value in cardiac surgery is not well established.

The IVA is a recently described index of systolic performance that is relatively load independent. It is calculated by dividing the maximal isovolumic myocardial velocity by the time to peak velocity using spectral pulsed wave or color tissue Doppler (Fig. 13). In 2002, Vogel et al. used color tissue Doppler to study tricuspid annular IVA in a closed chest animal model during modulation of preload, afterload, contractility, and heart rate. Their study demonstrated that, of all myocardial velocity parameters, IVA was the most reliable noninvasive index of contractility. Three clinical studies confirmed its value in CHD, i.e., postrepair of tetralogy of Fallot (TOF), TGA, and after cardiac surgery.\(^{47–50}\) Further validation of this promising new index in cardiac surgery is, however, required.

The maximum first derivative of RV pressure development \((dP/dt)_{\text{max}}\) has also been used as an index of RV contractility. This index may be calculated using continuous-wave Doppler of the tricuspid valve and the Bernoulli equation to calculate the pressure difference from 1 m/s to 2 m/s. Obtaining a reliable signal of tricuspid regurgitation in TEE, however, may be more difficult than in TTE. Furthermore, it has been demonstrated, in numerous studies, that RV \((dP/dt)_{\text{max}}\) is significantly affected by loading conditions and cannot be used as a reliable index of contractility.\(^{51}\) It may, however, be useful in assessing directional changes in response to therapy, assuming stable loading conditions.

The RVMPI has been described as a nongeometric index of global ventricular function (Fig. 14).\(^{52}\) It represents the ratio of isovolumic time intervals to ventricular ejection time (ET) and is calculated as 

\[
\text{RVMPI} = \frac{\text{IVCT} + \text{IVRT}}{\text{ET}},
\]

where IVCT is the isovolumic contraction time, and IVRT is isovolumic relaxation time.\(^{52}\) RVMPI increases in the presence of systolic or diastolic dysfunction. RVMPI has been validated in several disease states, including CHD, primary pulmonary hypertension, myocardial infarction, and chronic respiratory disease.\(^{53–56}\) A small prospective study has suggested that RVMPI may be useful in stratifying patients undergoing high-risk valvular surgery.\(^{38}\) It is important to remember that RVMPI is less reliable in the presence of arrhythmias.
or high-grade atrioventricular block. Pseudonormalization of the RVMPI has also been reported in acute, severe RV myocardial infarction.

**Regional RV Systolic Function**

The pattern of regional RV dysfunction may also be helpful in differentiating causes of RV dysfunction. For example, in pulmonary embolism, McConnell et al. described a distinct pattern of RV dysfunction characterized by severe hypokinesis of the RV mid-free wall associated with normal contraction of the apical segment (best seen in the four-chamber view). In contrast, in other causes of pulmonary hypertension, apical contraction is often depressed.

**Variables of RV Diastolic Function**

RV diastolic function has not been as extensively studied as that of the LV. Clinically useful diastolic parameters include:

- **IVC dimension (cm), collapse index**: ≤1.7 cm, CI >50%
- **Tricuspid early (E) to late (A) filling velocity ratio**: E/A: 1.5 ± 3.9
- **Hepatic vein profile (S: systolic, D: diastolic)**: S/D velocity ratio >1, no S reversal, atrial reversal <50% S
- **IVRT (<60 ms)**
- **Rapid myocardial filling velocity (E_t) (cm/s)**: E_t: 15.6 ± 3.9
- **Late diastolic myocardial filling velocity, A_t (cm/s)**: A_t: 15.4 ± 4.5

**Combined systolic and diastolic parameter**

**RVMPI**

The severity of RV systolic dysfunction may be graded using RVFAC and RVEF. Using RVFAC, mild dysfunction: 25%–31%, moderate dysfunction: 18%–24%, severe dysfunction <17%; using RVEF, mild dysfunction: 35%–44%, moderate dysfunction: 26%–34%, severe dysfunction <25%. Almost all normal values have been established in nonventilated patients.

<table>
<thead>
<tr>
<th>Functional parameters</th>
<th>Normal value</th>
<th>Load dependency*</th>
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<tbody>
<tr>
<td><strong>Systolic performance variables</strong></td>
<td></td>
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</tr>
<tr>
<td>RVFAC (%)</td>
<td>32%–60%</td>
<td>++ +</td>
</tr>
<tr>
<td>RVEF (%)</td>
<td>45%–68%</td>
<td>++ +</td>
</tr>
<tr>
<td>TAPSE</td>
<td>&gt;15 mm</td>
<td>++</td>
</tr>
<tr>
<td>Tricuspid annular plane maximal systolic velocity (using spectral pulsed wave tissue Doppler)</td>
<td>&gt;12 cm/s</td>
<td>++</td>
</tr>
<tr>
<td>IVA (using tissue pulsed wave Doppler)</td>
<td>1.4 ± 0.5 m/s²</td>
<td>+</td>
</tr>
<tr>
<td><strong>Diastolic parameters</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVC dimension (cm), collapse index</td>
<td>&lt;1.7 cm, CI &gt;50%</td>
<td>+++</td>
</tr>
<tr>
<td>Tricuspid early (E) to late (A) filling velocity ratio</td>
<td>1.5 ± 0.3</td>
<td>+++</td>
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<tr>
<td>Hepatic vein profile (S: systolic, D: diastolic)</td>
<td>S/D velocity ratio &gt;1, no S reversal, atrial reversal &lt;50% S</td>
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</tr>
<tr>
<td>IVRT</td>
<td>&lt;60 ms</td>
<td>+++</td>
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<tr>
<td>Rapid myocardial filling velocity (E_t) (cm/s)</td>
<td>E_t: 15.6 ± 3.9</td>
<td>+++</td>
</tr>
<tr>
<td>Late diastolic myocardial filling velocity, A_t (cm/s)</td>
<td>A_t: 15.4 ± 4.5</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Combined systolic and diastolic parameter</strong></td>
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<td></td>
</tr>
<tr>
<td>RVMPI</td>
<td>0.28 ± 0.04</td>
<td>++</td>
</tr>
</tbody>
</table>

*Refers to the degree of load dependency from minimal to significant: ++ ++. IVA = isovolumic acceleration using Doppler tissue imaging; IVC = inferior vena cava; RVEF = right ventricular ejection fraction; RVFAC = right ventricular fractional area change; RVMPI = right ventricular myocardial performance index; TAPSE = tricuspid annular plane systolic excursion; St = maximal systolic tricuspid annular plane velocity; IVRT = isovolumic relaxation time.

Adapted from Dell'Italia, Leng, Lorenz et al., Huwitz et al., Pfisterer et al., Santamore et al., Weyman, Davlouros et al., Tei et al., Cohen et al.
Figure 12. (A) Deep transgastric right ventricular in/outflow long-axis view. This view allows the evaluation of both the pulsed wave Doppler interrogation of the tricuspid valve and tissue Doppler imaging of the tricuspid annulus along the dotted line. (B) The darker line on the right side of the triangular sketch is matched to the thicker line on the triangular slice of the three-dimensional (3D) icon. (C) Tissue Doppler signal obtained at the base of the tricuspid annulus (Video 6; please see video clips available at www.anesthesia-analgesia.org). $A_t =$ tricuspid late diastolic filling (during atrial contraction) tissue Doppler velocity; $E_t =$ tricuspid early diastolic filling tissue Doppler velocity; $IVC =$ inferior vena cava; $MPA =$ mean pulmonary artery; $PV =$ pulmonic valve; $RA =$ right atrium; $RV =$ right ventricle; $St =$ tricuspid systolic tissue Doppler velocity; $SVC =$ superior vena cava; $TV =$ tricuspid valve. Adapted with permission from Denault et al. 71

Figure 13. Mean tissue color Doppler velocities of the basal tricuspid annulus during a cardiac cycle. The dominant slope is used to measure the isovolumic acceleration (IVA). $A_t =$ tricuspid late diastolic filling (during atrial contraction) tissue Doppler velocity; $E_t =$ tricuspid early diastolic filling tissue Doppler velocity; $IVC =$ isovolumic contraction; $St =$ tricuspid systolic tissue Doppler velocity; $t =$ time; $V_t =$ tissue Doppler velocity during IVC.
variables of RV diastolic function include RAP, RV filling profiles, and hepatic vein profiles.\textsuperscript{58} Compared with LV filling, the velocities across the tricuspid valve are significantly lower than those of the mitral. The tricuspid deceleration time is also longer than mitral deceleration time. Tricuspid filling profiles are usually measured in the mid-esophageal view or the transgastric view with rightward rotation of the probe.

In nonventilated patients, the IVC size and collapse index correlate well with RAP. The collapse index refers to the relative decrease in IVC diameter with inspiration (as with sniffing). An IVC size <2 cm with a collapse index of more than 50% usually corresponds to a RAP <5 mm Hg. A dilated IVC with a collapse index of \( \leq 10\% \) usually corresponds to a RAP of 20 mm Hg. Although correlations in ventilated patients have not been as well validated, studies suggest that the collapse index percentage of the IVC correlates with fluid responsiveness.\textsuperscript{59}

Only a few studies have assessed the importance of RV diastolic filling profiles in cardiac surgery.\textsuperscript{58,60–62} In a study by Carricart et al.,\textsuperscript{61} abnormal hepatic venous flow velocities before cardiac surgery were associated with an increased need for vasoactive support after cardiopulmonary bypass. These flow velocities, however, were not shown to be an independent predictor of worse outcome on multivariate analysis. In a study by Denault et al.,\textsuperscript{62} abnormal preoperative RV diastolic profiles were associated with difficult separation from cardiopulmonary bypass. Further studies are needed to validate these findings and to assess the independent value of RV diastolic function in cardiac surgery.

In TOF, a restrictive RV filling profile has been associated with worse outcome early after repair of TOF. A “restrictive RV physiology,” as described in TOF, is characterized by the presence of forward and laminar late diastolic pulmonary flow throughout respiration.\textsuperscript{30} In the presence of a noncompliant RV, atrial contraction results in an increase in RV pressures exceeding pulmonary pressures, thus resulting in late diastolic pulmonary flow. Early after TOF repair, a restrictive RV pattern, suggestive of a noncompliant ventricle, has been associated with a low cardiac output and longer intensive care stays.\textsuperscript{30,63} Late after TOF repair, however, restrictive RV physiology counteracts the effects of chronic pulmonary regurgitation and is associated with a smaller RV, shorter QRS duration and increased exercise tolerance.\textsuperscript{30,64}

**Ventricular Interdependence**

Ventricular interdependence may be exaggerated in patients with constrictive pericarditis or tamponade physiology, whereas it is usually in the normal range in restrictive disease. Other factors which may increase ventricular interdependence include loading conditions and increased intrathoracic pressures.\textsuperscript{21}
The two most useful features that suggest increased ventricular interdependence include the presence of increased reciprocal respiratory changes in tricuspid and mitral inflow maximal velocity and reciprocal respiratory changes in RV and LV size. These features have mainly been studied in nonventilated patients.

Under normal conditions and spontaneous respiration, the tricuspid inflow maximal velocity increase with inspiration is usually 15%, whereas that of the mitral valve is usually 10%. In constrictive pericarditis, the tricuspid velocity change is often greater than 40%, whereas mitral inflow velocity change with respiration is usually more than 25%. In tamponade, the tricuspid respiratory velocity change is often greater than 85% (increasing with inspiration), whereas that of the mitral valve is usually more than 40% (decreasing with inspiration). In contrast, in restrictive physiology, the respiratory changes in tricuspid and mitral velocities are usually not increased and the early filling to atrial contraction ratio is often greater than 2 with a mitral inflow deceleration time of <150 ms. There is usually no abnormal septal motion. It is, however, important to emphasize that restriction, constriction, and tamponade may vary in degree of severity and can also coexist, creating clinical pictures which may sometimes be difficult to sort out.

Valvular Function and RV Inflow Tract and RVOT Gradients

The study of RV function would be incomplete without the assessment of the tricuspid or pulmonary valves function. Tricuspid regurgitation may be primary or, more commonly, secondary to RV dilation or pulmonary hypertension. The most common causes of increased right atrioventricular gradient are tricuspid stenosis, tricuspid valvuloplasty, or a prosthetic tricuspid valve. Significant pulmonary regurgitation is usually seen after TOF repair or as a consequence of severe pulmonary hypertension. Increased transpulmonary (outflow tract) gradients may be caused by pulmonary stenosis, structural or dynamic RVOT obstructions or by increased RV cardiac output. Recently, dynamic RVOT obstruction has been described as a possible cause of hemodynamic instability in cardiac surgery (Fig. 15; Video 7; please see video clips available at www.anesthesia-analgesia.org).

The guidelines from the ASE for the evaluation of tricuspid and pulmonary regurgitation may be found in a review article published in 2003 by Zoghbi et al.

CONCLUSION

Acute RV failure after cardiac surgery remains a major cause of morbidity and mortality. A comprehensive assessment of RV function may improve risk stratification and lead to early management of RV failure. Echocardiography is becoming a mainstay in the assessment of perioperative RV function. Although RV assessment remains challenging, echocardiography offers useful information on RV size, shape, and function. Future advances in 3D echocardiography may further improve the assessment of complex

Figure 15. Dynamic right ventricular outflow tract obstruction in a 28-year-old man after aortic valve replacement. A 17 mm Hg gradient between the right ventricular pressure (Prv) and the pulmonary artery pressure (Ppa) is present. An M-mode view obtained in the mid-esophageal inflow-outflow view demonstrates the right ventricular outflow tract collapse (B). This is also shown in the same view during systole (C–E) and in a deep right ventricular outflow tract (D–F). EKG = electrocardiogram; LA = left atrium; LV = left ventricle; PA = arterial pressure; RV = right ventricle.
cogentinal defects and lead to better quantification of RV size and function.

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