Accurate Echo Assessment of Right Ventricular Dysfunction: Are We There Yet?

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At the conclusion of this lecture, the participant should be able to:
1. Discuss the complex anatomy and physiology of the right ventricle
2. List the reasons that RV dysfunction occurs perioperatively
3. Describe the approach to assessment of RV function by TEE

Assessment of right ventricular (RV) anatomy and function should always be performed during a comprehensive TEE examination. Preoperative RV dysfunction is a predictor of poor outcome. In CABG patients with poor left ventricular (LV) systolic function (LV ejection fraction [LVEF] <25%) undergoing elective revascularization, a pre-CPB RVEF <35% was associated with longer mechanical ventilation and length of stay, 71% in-hospital mortality and 0% 2-year survival. (Maslow, Anesth Analg 2002) In patients with non-ischemic mitral regurgitation, RVEF <30% was the only significant predictor of postoperative death. (Wencker, Cardiol 2000) In post-cardiac surgery patients with systemic arterial hypotension (mean arterial pressure <60 mmHg), the mortality rates were 82% in severe biventricular failure and 90% in isolated RV systolic failure. (Reichert, J Cardioth Vasc Anesth 1992) In addition, RV performance is an important determinant of clinical status and long-term outcome in patients with pulmonary hypertension, cardiomyopathies and congenital cardiac disease. However, there is still uncertainty on how to evaluate the RV in the daily clinical practice.

RV anatomy

The RV is located anterior and to the right of the left ventricle (LV). Its distance from the esophagus makes surface ultrasound (TTE) and CT and CMR more suitable for evaluation of its anatomy. Its complex geometric shape appears triangular when viewed from the side (ME 4C), as a half-circle (TG SAX) or crescent in cross-section (ME inflow-outflow and TG RV LAX). It is divided in three anatomic regions: the inflow tract (sinus) which is bordered by the tricuspid valve (TV) and annulus (TA), the outflow tract ([RVOT] or infudibulum) which terminates at the pulmonary annulus, and the heavily trabeculated apex between them. Contrary to the LV, the inflow and outflow parts are not adjacent (compare the mitral and aortic valve) and lie in different planes (and that
makes it difficult to image both of them simultaneously with 2D echocardiography). The RV wall is thin (3-5 mm), difficult to differentiate from the adjacent pericardium and is heavily trabeculated so that mass and volume calculations are difficult to make. The RV myocardial precursor cells have a different origin than the LV precursor cells, and the expression of genes that are involved in adaptive remodeling is different; both may explain the different response of the RV myocardium to abnormal loading conditions. (Friedberg MK, Mertens LL. Nature Rev Cardiol 2010) The architecture of the myocardial fibers is different in the RV: there are longitudinal epi- and endocardial and circumferential mid-myocardial fibers in the LV, while in the RV most myocardial fibers are longitudinally oriented and mid-wall radially oriented myocardial fibers are seen only in RV hypertrophy. RV fibrosis is present in adult patients after tetralogy of Fallot repair in the surgically patched RV outflow tract and the surrounding myocardial segments as well as the anterior wall. The extent of fibrosis correlates well with regional and global RV dysfunction and is associated with an increased risk of ventricular arrhythmia. (Wald. Circulation 2009)

**RV function**

While the RV stroke volume is equal to that of LV, it is ejected by a different mechanism. The RV contraction starts in the apex and propagates to the RVOT. It is produced by: i) contraction of the longitudinal fibers of the free wall, which draw the inflow part (TA) apically, ii) the bellows-like inward motion of RV free wall, and iii) traction of RV attachment sites to LV wall and bulging of the interventricular septum towards the RV cavity. The RV cavity contains more blood than LV, has a lower EF: 40-45% (compared to LVEF of 50-55%) and ejects its stroke volume to low vascular resistance bed. Analysis of pressure-volume loops showed that the RV has a brief isovolumic contraction period and RV ejections continues while the RV systolic pressure declines. This is represented by a triangular or trapezoidal shaped pressure-volume loop that is associated with a lower energy requirement, compared to the square shape of the LV pressure-volume loop. (Redington. Cardiol Clin 2002) The RV has much less torsional motion than the LV.

**RV volumes**

RV dilatation is often the first sign of RV pressure and volume overload and reflects the underlying RV pathology. The anterior position of the RV makes CMR the ideal “gold standard” technique for quantification of RV volumes (normal: RV end-diastolic volume
index [RVEDVi] <110 ml/m² for men and <100 ml/m² for women). In patients with corrected tetralogy of Fallot and pulmonic regurgitation, RV reverse remodeling is unlikely if RVEDVi >150-170 ml/m². (Oosterhof. Circulation 2007) 3D echocardiography is validated against CMR for RV volume assessment, but measurements are not always successful and volumes are underestimated by about 20-40%. (van der Zwaan HB. J Am Soc Echocardiogr 2010) 2D echocardiography is the most frequent clinical technique used to assess the RV size, but it correlates poorly with the CMR measurements due to the complex RV geometry. Instead, RV volume is estimated from various linear measurements.

**RV linear, structural measurements**

There are multiple linear measurements performed in the ME or TG views, which if followed through time, can be used to monitor changes in RV size. All measurements should be done at end-exhalation using second harmonic imaging and by adjusting gain and compression to improve border definition. The 2D linear measurements are:

- RV diameter at the TA annulus (ME 4C): 2-2.8 cm
- RV long axis diameter (ME 4C): 7.1-7.9 cm
- RVOT diameter at the hinge points of the pulmonic valve cusps (ME RV inflow-outflow): 1.7-2.3 cm
- RV free wall thickness (TG SAX): <5 mm

The RVOT shortening fraction (TTE: parasternal SAX; TEE: ME RV inflow-outflow) is the % difference of the end-diastolic and end-systolic diameters at the level of RVOT, measured with M-mode. The measurement correlates well with longitudinal function, pulmonary pressure gradient and RV-RA pressure gradient. (Lindqvist P. Eur J Echocardiogr 2003)

The RV %EF (TTE: Apical 4C; TEE: ME 4C view) is measured using the method of discs or area-length methods (as when evaluating LV function; normal values are 40-45%). Either of these methods will not calculate the true stroke volume of the RV due to its complex geometric shape.

The RV fractional area change (RV %FAC, TTE: apical 4C; TEE: ME 4C) is a more reliable measurement than RVEF, but requires good endocardial border delineation. Values are: normal 32-60%, mildly abnormal 25-31%, moderately abnormal 18-24%, severely abnormal <17%.

In normal ventricles, the longitudinal excursion of the RV base accounts for the greater proportion of the total RV volume changes in comparison with radial. The **Tricuspid Annulus Plane Systolic Excursion** (TAPSE) is a measurement of the systolic excursion of the lateral TA and is used to evaluate the RV longitudinal function. TAPSE is measured with an M-mode cursor positioned alongside the lateral side of the TA in a view that aligns best the motion plane with the M-mode cursor (any of the ME 4C, ME RV inflow-outflow or TG RV LAX) as the distance between end-diastolic and end-systolic excursions (normal values are >1.5-2 cm). In patients following myocardial infarction, TAPSE <1.5 cm had increased mortality (45% at 2 years) compared to those
with TAPSE >2 cm (4%). (Samad. Am J Cardiol 2002) However TAPSE ignores the outflow tract and the septal contribution to the RV ejection, which may be important in pathology involving the longitudinal fibers. TAPSE is one-dimensional, angle-dependent and load-dependent and may be influenced by LV systolic performance via interventricular interdependence.

RV distention caused by volume or pressure overload is assessed with the eccentricity index (EI). EI describes the changes in LV dimensions: it is the ratio of LV minor axis (inferior-to-anterior) to its perpendicular axis (septal-to-lateral) when measured in the TG mid SAX view (normal =1, i.e., LV is round in systole and diastole). EI >1 (LV is shaped as the letter D) is found in RV pressure overload at end-systole and in RV volume overload at end-diastole. A high EI is an important echocardiographic predictor of mortality in pulmonary arterial hypertension. (Ryan. J Am Coll Cardiol 1985)

**RV functional measurements**

The **RV myocardial performance index** is a physiological or functional measurement. It is the ratio of the time of no flow (ICT, isovolumic contraction and ICT, isovolumic relaxation) and flow through the pulmonic valve (ET, ejection time), or MPI (or “Tei” index) = (ICT + IRT) / ET. All intervals are measured with pulsed wave Doppler (PWD) from sequential recordings in the tricuspid and pulmonic valves. Normal values are 0.28±0.08%. It was shown to correlate well with the MRI-derived RVEF. (Karnati PK. Echocardiography 2008) The RV MPI increases with RV dysfunction, but its use is limited by the absence of isovolumic periods in the normal RV and “pseudonormalization” in cases of increased right atrial pressure, when IRT is shortened. A regional RV Tei index can be measured from the DTI signal (see below); it was found to correlate well with the RV MPI. (Harada. Am J Cardiol 2002)

The **RV dp/dt** is the first derivative of maximum RV pressure increase over time; it is considered an afterload-independent index of RV contractility. It is measured from the continuous wave Doppler (CWD) signal of tricuspid regurgitation (TR), and is based upon the time required for the RV velocity to increase from 1 m/s to 2 m/s, which corresponds to a pressure gradient of 12 mmHg. It is preload dependent and requires the presence of a measurable TR jet. However, changes within patients may direct therapeutic measurements, provided that loading conditions remain stable.

**Doppler tissue imaging** (DTI) of the RV inflow velocities; the systolic velocity of the RV free wall (S’) is measured with a DTI sample volume placed lateral to the TA in the TG RV LAX view. Normal values are >12 cm/s (TTE, awake subjects). Recordings are also feasible in the deep TG LAX view with a sample volume placed lateral to the TA. An S’ <11.5 cm/s was predictive of RVEF <45%. (Meluzin. Eur Heart J 2001) The myocardial isovolumic acceleration time (IVA) is supposed to be less dependent of loading conditions than the DTI velocities. An IVA >1.1 m/s² correlates well with an MRI RVEF >45%. (Lyssegen E. Circulation 2005)

**RV free wall strain** by DTI or speckle-tracking echocardiography represents the longitudinal deformation during the cardiac cycle (shortening in systole, lengthening in diastole) and requires echocardiographic systems with appropriate preset modes. The
rate of deformation (strain rate) is less load dependent than strain; both strain and strain rate are higher in the apical regions than the basal ones. Normal values are reported for TTE (in the apical four chamber view); normal values: DTI-strain < -18.2%, speckle-strain < -26--29 (±5.2-4.3)%.(Teske AJ. J Am Soc Echocardiogr 2008)

Evaluation of RV function is not complete unless the proximal (RA pressure) and distal (pulmonary artery) pressures as well as, pulmonary vascular resistance are determined.

**RA pressure** can be reliably differentiated between normal or elevated, by combining 2D, PWD and DTI findings. Elevated RA pressure is associated with:
i) inferior vena cava dilation (see above).
ii) enlargement of hepatic veins, coronary sinus, and RA and bowing of the interatrial septum towards the RA.
iii) Hepatic Vein Flow (HVF) profile demonstrating diastolic predominance (S/D <1; caveat: this is valid only if the rhythm is sinus; patients in atrial fibrillation will have a ratio S/D <1 irrespective of RA pressure!). As RA pressure increases, the pressure gradient between hepatic veins and RA decreases and so does the S wave velocity. Another HVF sign of increased RAP is when rA velocity > S velocity. TR will obviously decrease the HVF S velocity and complicate determination of RA pressure.
iv) a ratio of transtricuspid flow-E to DTI-E’ (E/E’) >6, was associated with mean RAP>10 mmHg in spontaneously breathing subjects.

The inferior vena cava (IVC) diameter and its change with spontaneous inspiration are used in the ambulatory patient to estimate the RA pressure. A baseline diameter <2 cm that decreases >50% with inspiration is associated with a RA pressure <5 mmHg, while a dilated IVC which changes <10% with inspiration is associated with a RA pressure >20 mmHg. Although there are no accepted normal values for mechanically ventilated patients, an IVC size <12 mm predicts a RAP <10 mmHg.

The **PA systolic pressure** (PASP) is equivalent to RV systolic pressure (excluding obstruction between RV and PA). PASP is calculated from the peak velocity of TR jet using the simplified Bernoulli equation: PASP = 4 × TRjet² + estimated RAP. The obvious limitations of this method are: i) underestimation of TR maximum velocity (multiple windows should be used), ii) undetectable TR jet (use of agitated saline and blood can improve imaging), and iii) erroneous assumptions of RA pressure (unless a central venous catheter is in situ). Pulmonary hypertension is diagnosed if PASP >35 mmHg or mean PA >25 mmHg (at rest) or mean PA >30 mmHg with exercise.

The PA diastolic pressure can be estimated using the simplified Bernoulli equation if there is a recordable pulmonary insufficiency (PI) jet, measured with CWD (ME ascending aorta SAX or UE arch SAX views): PADP = 4 × PI jet end-diastolic² + estimated RAP. Alternatively, mean PAP = 4 × PI jetpeak² + estimated RAP.

The estimation of **Pulmonary Vascular Resistance (PVR)** is done using the TR jet (measured with CWD) and the VTI of the RVOT velocity (measured with PWD in the deep TG LAX view): PVR (WU) = 10 × TRvelocity/RVOTVTI. A ratio TRvelocity/RVOTVTI >0.2 predicts PVR >2 WU. The physiologic background of this ratio is that a rise in
PASP due to increased flow will result in lower ratio than a rise in PASP secondary to increased PVR.

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