Echocardiographic assessment of Right Ventricular (RV) Function

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Introduction:

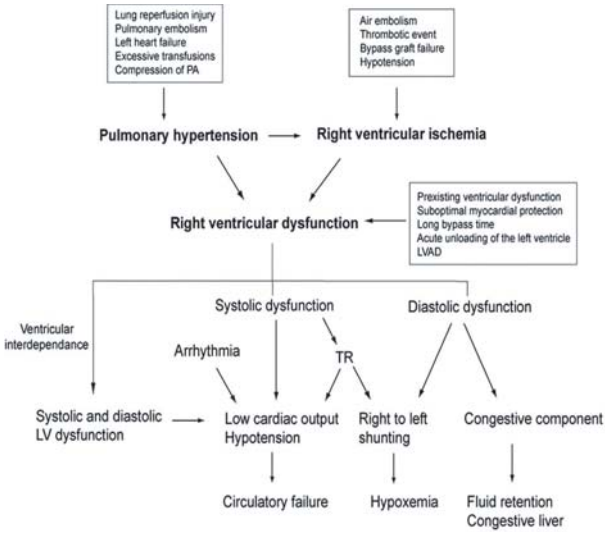
The importance of right ventricular function has been underestimated in the past, especially its role as a determinant of cardiac symptoms, exercise tolerance and survival in patients with valvular disease of the left heart. The pump function of the right ventricle has been thought not to be relevant for the overall function of the heart.[1, 2] and to improve if the cause for its dysfunction is removed.[3] It has been shown that right ventricular function is a major determinant of cardiac symptoms and exercise capacity in chronic heart failure. [4, 5] An increase in pulmonary wedge pressure as a result of mitral or aortic valve disease is associated with a rise in mean pulmonary artery pressure. Thus, right ventricular afterload increases, as dilatation of the right ventricle develops with a resultant drop in the right ventricular ejection fraction. As a consequence, the tricuspid valve annulus dilates and may induce tricuspid regurgitation with secondary right ventricular volume overload.[6] Echocardiography relies on geometrical assumptions or a three-dimensional analysis of the right ventricle which is not always sufficiently visualized.[7] Radionuclide angiography requires injection of radioactive markers and has a low spatial resolution.[8-10]

Right ventricular functions cannot reliably be evaluated by conventional echocardiography techniques because of asymmetrical shape (crescentic), narrow acoustic window, irregular endocardial surface, complex contraction mechanism.[11] The problem is compounded by irregular trabeculations, a separate infundibulum, and variations in right ventricular shape with altered loading conditions. The substernal right ventricle is less accessible than the left ventricle and its dimensions more difficult to standardize .[12]

RV failure is a complex clinical syndrome that can result from any structural or functional cardiovascular disorder that impairs the ability of the RV to fill or to eject blood. The cardinal clinical manifestations of RV failure are (1) fluid retention, which may lead to peripheral edema, ascites, and anasarca; (2) decreased systolic reserve or low cardiac output, which may lead to exercise intolerance and fatigue; or (3) atrial or ventricular arrhythmias.[13]

Pathophysiology of RV failure:

The RV may be subject to pressure or volume overload, ischemia, intrinsic myocardial disease, or pericardial constraint. RV dysfunction begins with an initial injury or a stress on the myocardium and may progress in the absence of a new identifiable insult to the heart. The most common cause of RV dysfunction is chronic left-sided HF. Pulmonary hypertension (PH) is an important cause of RV dysfunction. RV dysfunction also is a prominent feature of various forms of congenital heart diseases (CHD) such as tetralogy of Fallot (TOF), transposition of the great arteries, Ebstein's anomaly, and Eisenmenger syndrome.[13]



Pathophysiology of RV failure.[13]

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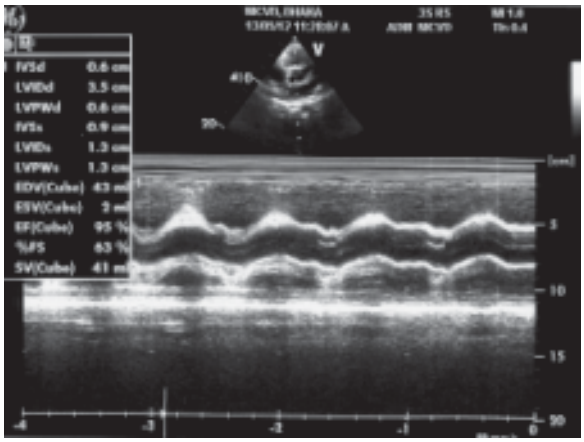
Echocardiographic parameters for right ventricular function assessment:

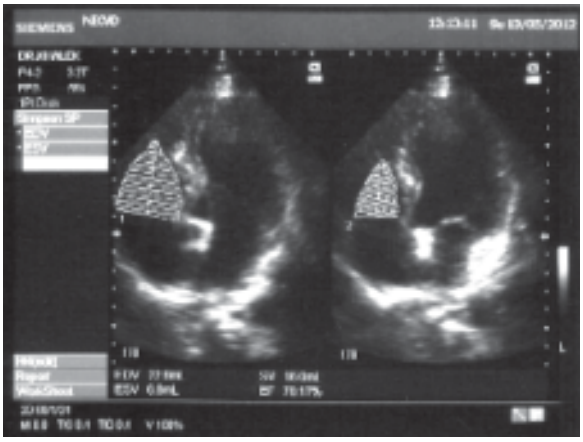
- RV free wall thickness (mm)
- RV diastolic diameter (mm)
- RVOTfs % (right ventricular outflow tract fractional shortening)
- RVEF % (right ventricular ejection fraction)
- RVTei (right ventricular total ejection isovolume) index
- RV IVA (m/s²) (right ventricular myocardial acceleration during isovolumic contraction)
- TAPSE (tricuspid annular plane systolic excursion)
- RVFAC % (right ventricular fractional area change

RVOTfs (%) From the parasternal short axis view at the level of the aortic root, the RV outflow tract diameters at end-diastole and end-systole calculated and RVOTfs (%) calculated by (RVEDD - RVESD / RVEDD) X 100. RVOTfs reflects RV infundibular function.[67, 68] Control group value 56.5 ± 4.4 %. [53]



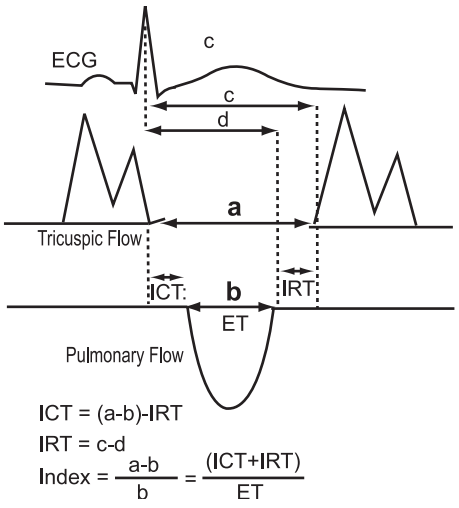
Measurement of RVOT fs (%)

RVEF (%): In Apical 4 chamber view, calculated by using Simpson's (single plane) Rule (tracing around the endocardial border going from one side of the tricuspid valve annulus to the other and joining the two ends with a straight line both in systole and diastole). [67,68] Normal value of RVEF 55.1± 3.7 (48-63) in male and in female 59.8 ± 5.0 (50-70).[69] another study showed normal RVEF 50-55%. [41]



Measurement of RVEF (%)

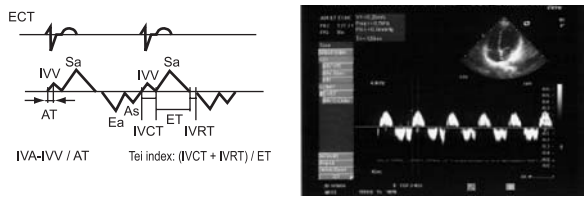
RV Tei Index: In apical 4 chamber view a sample volume (Pulsed wave Doppler) placed at the tricuspid valve. RVTei Index calculated as the sum of isovolumic contraction time (IVCT) and isovolumic relaxation time (IVRT) divided by ejection time (ET). Normal Tei index d" 0.40. The Tei index reflects both RV systolic and diastolic function. [68, 67, 70]



Measurement of RVTei (right ventricular total ejection isovolume) index.

Doppler Tissue Imaging (DTI) derived myocardial acceleration during isovolumic contraction (IVA) of tricuspid lateral annulus: In apical 4 chamber view, a sample volume placed at the tricuspid

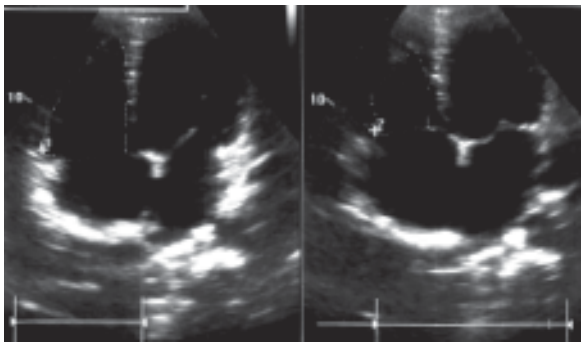
lateral annulus. IVA measured by dividing myocardial velocity during isovolumic contraction by the time interval from onset of the myocardial velocity during isovolumetric contraction to the time at peak velocity of this wave The ratio of IVV (myocardial velocity during isovolumic contraction) divided by the acceleration time (AT). IVA (m/s^2) = IVV / AT. [67, 68] IVA is a measurement of RV contractile function that is unaffected by preload and afterload change in a physiological range. This novel index (IVA) may be ideally suited to the assessment of acute changes of RV function in clinical studies and could be used in early detection of RV systolic dysfunction in patient with MS, before the signs of systemic venous congestion occur. Control group value 3.2 ± 0.3 (m/s^2). [68]



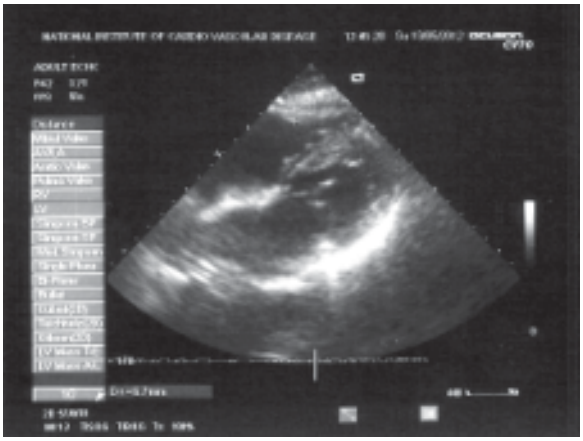
Measurement of RV IVA (m/s^2). [68]

TAPSE (tricuspid annular plane systolic excursion): The tricuspid annular plane systolic excursion was determined by the difference in the displacement of the RV base during systole and diastole.[67, 68] (Normal motion is around 1.5 – 2.0 cm).[71]

RVFAC % (right ventricular fractional area change: RV end diastolic and end-systolic areas were measured from the apical four chamber view to calculate RV fractional area change (RVFAC).[67] Fractional area change = Change in area / area in diastole × 100%. Normal range 32- 60 %. [71]

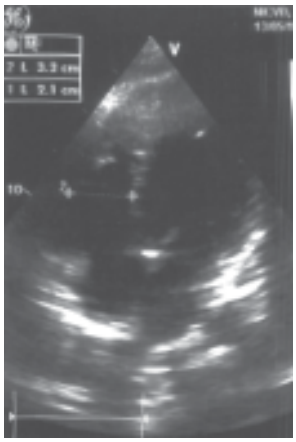


RV free wall thickness (mm): this can be assessed in parasternal long axis and apical 4 chamber views but most consistent measures are from the subcostal view (normal value < 5 mm).[71]



Measurement of RV free wall thickness.[71]

RV diastolic diameter (mm): in apical four chamber view (mid RV diameter 27- 33mm) and at RVOT RV diameter 25- 29 mm (parasternal short axis view). [71]



Measurement of RV diastolic diameter

Importance of the right ventricular function assessment:

As a prognostic factor in cardiac surgery:
The importance of the right ventricle for peri-operative morbidity and mortality was recognized several years ago. Thus, the right ventricle plays an important role not only for survival but also for the postoperative course and functional recovery of the patient with valve disease of the left heart. [14]

RV dysfunction has been shown to be a significant prognostic factor in cardiac surgery and heart transplantation. [13]

Postcardiotomy RV failure is often precipitated by an element of ischemia and myocardial depression after cardiopulmonary bypass (CPB).[15]

Myocardial dysfunction and pulmonary hypertension (PH) after CPB are usually mild and do not lead to postoperative circulatory failure.

Recognition of high-risk patients and early management of RV dysfunction may decrease the incidence of refractory postoperative RV failure. [13] Despite advances in the perioperative management of heart transplantation, acute RV failure still accounts for a significant number of early complications and early deaths in up to 20% of patients in some reports. [16, 17]

Mitral valve disease:

Generally, the prognosis of patients with mitral valve disease and depressed right ventricular function improves significantly after successful mitral valve replacement or repair. [18, 19]

Abnormalities of right ventricular function play an important role in the development of clinical symptoms and the over all prognosis of the patients with MS. The right ventricular function is an important determinant of clinical symptoms, exercise capacity, pre-operative survival and post-operative outcome in patients with mitral stenosis .[20] Impairment of right ventricular (RV) function by post rheumatic mitral stenosis due to passive increase of left atrial pressure and due to the reactive changes of pulmonary arteriolar vasculature, right ventricular after load may increase to values 25 to 30 fold above normal, leading to right ventricular overload and right ventricular failure. It is hypothesized that rheumatic heart disease may directly involve the myocardium, thus directly impairing right ventricular function.[21] Intramyocardial branches of coronary vessels were involved in a form of active rheumatic vasculitis or inactive lesions characterized by medial hypertrophy and replacement fibrosis.[22]

Congestive heart failure: [23]

The critical role of right ventricular function in patients with advanced congestive heart failure. The estimation of right ventricular function is nowadays warranted in the standard evaluation of patients with heart failure either due to ischemic heart disease or to primary dilated cardiomyopathy, since it is helpful in the clinical assessment and in the prognostic stratification of such patients.[24]

Congenital heart diseases: [25]

In congenital heart disease. The right ventricle may be subject to abnormal loading conditions both before and after surgery, or to the long-term effects of prolonged hypoxemia. Extensive data support concern about the long-term effects of these problems on right ventricular (RV) function.[26-30] The RV plays an important role in CHD, where it may support the pulmonary circulation (pulmonary RV) or the systemic circulation (systemic RV).[31, 35, 36] Recent studies have demonstrated that RV function is one of the most important predictors of survival and postoperative outcome in patients with CHD and RV pressure or volume overload.[31-34] In patients with repaired tetralogy of Fallot (TOF), severe pulmonary regurgitation is the most common cause of progressive RV dilation and failure and is associated with decreased exercise tolerance, atrial and ventricular arrhythmias, and sudden death. Severe RV dilation, especially when progressive, may be the first sign of a failing RV and should prompt consideration of pulmonary valve replacement. Pulmonary valve replacement generally results in a decrease in RV volume.[31]

In patients with isolated atrial septal defect (ASD) and normal pulmonary pressure or mild PH, closure of the defect usually results in progressive remodeling of the RV. Incomplete RV remodeling in ASD may, however, be seen in older patients (> 40 yr old) or in patients with abnormal preoperative RV myocardial relaxation.[37] Closure of an ASD in patients with severe pulmonary vascular disease usually precipitates RV failure.[38, 39]

In transposition of the great arteries (TGA) the anatomic RV supports the systemic circulation. Because the RV is not well suited to support the systemic circulation, RV failure occurs and is closely related to outcome.[31]

In Ebstein's anomaly malformation results in atrialization of a portion of the RV and moderate-to-severe tricuspid regurgitation. The size of the

functional RV and tricuspid valve morphology (attachment,commissures, surface) determines the best surgical approach.[31]

Right ventricular dysfunction after acute pulmonary embolism (PE):

Acute PE may lead to right ventricular dilatation and failure. [40, 41]

RV dysfunction after VVI pacing:

Right ventricular apical pacing has a major effect on systolic and diastolic ventricular functions. On serial evaluation, right ventricle dysfunction is the first abnormality that occurs in right ventricular apical paced patients, followed by LV dysfunction which appears later and on both sides, diastolic dysfunction precede the systolic dysfunction.[42]

RV dysfunction after 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. [43]

RV dysfunction in chronic lung diseases:

In chronic lung disease common presentation is 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. [44-46]

RV dysfunction in primary pulmonary hypertension:

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. [47]

Right/left ventricular interaction

Left-to-right interaction

Apart from the influences of the left ventricle on the right ventricle via the pulmonary circulation, the left ventricle acts also on right ventricular function through the interventricular septum. [48-50] The left ventricular contraction contributed 24% of its own stroke work to the generation of right ventricular stroke work via the interventricular septum. In pulmonary hypertension this contribution increased to 35%.[51]

Right-to-left ventricular interaction

The filling state of the right ventricle influences the motion of the interventricular septum and thus, left ventricular performance. Right ventricular volume overload with dilatation of the right ventricle and right atrium causes an increase in intrapericardial pressure (pericardial constraint), reducing venous return, cardiac output and, thus, left ventricular function. [52]

Right ventricular ejection fraction is dependent on right ventricular afterload and, thus, on left ventricular or left atrial filling pressures. [53-56] An increase in left ventricular afterload is compensated for by an increase in mass to reduce left ventricular fibre stress. Thus, a decrease in right ventricular ejection fraction with an increase in pulmonary artery pressure has been reported in patients with valvular or coronary artery disease. [57] as well as in patients with chronic obstructive pulmonary disease.[58,59] rapid improvement or normalisation of right ventricular ejection fraction has been found after reduction of pulmonary artery pressure with nitroglycerin. [53] single lung transplantation [58] or left heart valve replacement.

The close correlation between right ventricular ejection fraction and mean pulmonary arterial pressure for patients with cardiac valve diseases indicates that right ventricular afterload rather than contractility is the major determinant of right ventricular ejection fraction.[14]

In most patients with aortic valve disease, right ventricular ejection fraction is maintained, whereas it is typically reduced in patients with mitral valve disease. [56, 60-64] Mitral valve disease generally influences the right ventricle more than aortic valve disease. [65] Left atrial volume overload in mitral insufficiency and pressure overload in mitral stenosis may cause an increase in pulmonary vascular resistance with an increase in the afterload of the right ventricle and, thus, a decrease in right ventricular ejection fraction. In contrast, left ventricular function is usually normal or even enhanced in mitral regurgitation due to low impedance leak. [66]

Conclusion:

For many years cardiologists were not much interested in studying right ventricular function and the role of the right ventricle in heart failure

and in other disease states have therefore been largely underestimated. In recent years, many studies have demonstrated the prognostic value of RV function in cardiovascular disease. Right ventricular (RV) function may be impaired in pulmonary hypertension (PH), congenital heart disease (CHD), and coronary artery disease and in patients with left-sided heart failure (HF) or valvular heart disease.

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