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The right ventricle in patients with chronic heart failure and atrial fibrillation

Ewa Majos, Rafał Dąbrowski, Hanna Szwed

2nd Ischemic Heart Disease Department, Institute of Cardiology, Warsaw, Poland

Abstract

Under normal conditions function of the right ventricle (RV) is determined by the heart rhythm, RV filling time, RV systolic synchrony and interdependence between both ventricles. Failure of the left ventricle (LV) can lead to RV failure. Impaired function of the RV significantly worsens the prognosis in patients after myocardial infarction and with LV failure. Permanent atrial fibrillation (AF) is one of the most common arrhythmia in patients with depressed RV function. Frequent coexistence of chronic heart failure (CHF) and AF causes overlapping of the arrhythmia and RV dysfunction in the setting of CHF. They may lead to hemodynamic compromise and worsen prognosis in patients with chronic RV failure of various etiologies. RV structure and function can be assessed in 2D, 3D echocardiography, cardiac magnetic resonance imaging and computed tomography. (Cardiol J 2013; 20, 3: 220–226)

Key words: right ventricle, chronic heart failure, atrial fibrillation, CRT, echocardiography

Introduction

Dysfunctions of both ventricles often coexist in chronic heart failure (CHF) patients. Left ventricular (LV) failure can lead to right ventricular (RV) dysfunction. Occurrence of RV failure worsens prognosis in patients with dysfunctional LV. The aim of this paper is to reflect current reference on RV performance as a consequence of developing CHF and atrial fibrillation (AF). Existing dysfunction or exposure to RV overload can be a substrate for AF occurrence as well. Factors that determine RV function under normal conditions and as a consequence of developing CHF and/or AF are discussed below. Methods of right heart imaging are presented. The role of RV function at different stages of CHF seems to be underestimated and little attention has been paid so far to its function in AF.

Right ventricular function under normal conditions

Function of the RV depends on its structure and myofibers architecture. The ventricle is triangular or crescent-shaped and is built of the inlet, the apical myocardium and the outlet tract. Its relatively thin myocardium is composed of a superficial circumferential layer, which continues to the LV, and a deep longitudinal layer which facilitates shortening of the ventricle in its long axis during the systole. Other mechanisms that enable RV contraction are concentric inwards motion of the free wall and traction on the superficial fibers of the free wall secondary to the systole of the LV [1–3].

Due to elastance of the thin RV myocardium and low resistance of highly distensible pulmonary vessels, pressures in the right side of the heart are significantly lower and the RV is more dependent

Address for correspondence: Dr Ewa Majos, MD, 2nd Ischemic Heart Disease Department, Institute of Cardiology, ul. Spartańska 1, 02–637 Warszawa, Poland, tel. + 48 22 343 40 50, fax: +48 22 844 95 10, e-mail: ewamjs@gmail.com

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on the afterload than the LV. Preload determines function of both ventricles [3–5].

Beside the preload, afterload and muscle contractility, RV function is determined by the heart rhythm, RV filling time, RV systolic synchrony and interdependence between both ventricles. Maintenance of sinus rhythm and atrio-ventricular synchrony is crucial for RV function especially in chronic RV failure and acute RV infarction [1, 4]. It was shown, that preserved function of both atria affects the RV more than the LV [2]. Ventricular interdependence is mainly dependent on function of the intraventricular septum. Studies in animal models proved that from 20% to 40% of systolic volume and pressure of the RV are generated by the LV systole. Diastolic interdependence is significantly marked under pathological conditions when volume or pressure overloaded RV shifts the intraventricular septum into the LV chamber and reduces the LV preload and cardiac output. On the other hand, volume or pressure of overloaded LV causes pressure increase in the RV [1, 6].

Right ventricular function in chronic heart failure

Ventricular interdependence implies the fact that dysfunctions of both ventricles often coexist. LV failure can lead to development of RV failure. On the other hand, impaired function of the RV significantly worsens the prognosis in patients with LV failure after myocardial infarction (MI) [5, 7]. Substudy from the VALIANT ECHO study in a group of 600 patients after MI with LV dysfunction demonstrated that decreased RV function prognoses worse outcome. Patients with reduced RV fractional area change (RVFAC) were at higher risk of all-cause mortality, death from cardiovascular events and sudden death. Additionally, they were more prone to develop CHF and were at higher risk of stroke than control individuals. Each 5% decrease in baseline RVFAC was associated with a 1.53 (95% CI 1.24–1.88) increased risk of fatal and nonfatal cardiovascular outcomes [8]. Substudy from the SAVE study in subjects after MI with impaired LV function showed that RV dysfunction was an independent risk factor of total mortality, cardiovascular mortality and occurrence of CHF. Each 5% reduction in the RVFAC resulted in 16% increased odds of cardiovascular mortality [9]. Other studies showed, that in patients with advanced CHF and LV ejection fraction (LVEF) < 40% prognosis strongly depends on RV function. RV function influenced the total outcome in these patients more significantly than LV function [10, 11].

Coexistence of CHF and AF is frequent. This is mainly due to common pathogenesis of both conditions and the fact that one enhances development and triggers symptoms of the other. Thus, in clinical practice both states should often be considered and treated together [12–14]. It is estimated that AF occurs in up to 50% of patients with CHF depending on NYHA class [14]. In patients with arrhythmia, LV diastolic function is often worse and may result in deterioration of RV function [15]. Therefore, frequent coexistence of RV dysfunction and AF in the setting of CHF is probable.

Cardiac resynchronization therapy (CRT) is a method of treatment in advanced CHF. It involves simultaneous stimulation of the RV and the LV. CRT is well established in patients with sinus rhythm. According to current guidelines it may be also used in patients with permanent AF (IIb/C) [16]. Patients responding to the CRT experience improvement of the LV function, subsequent clinical status and reduction in number of hospitalizations due to CHF [17, 18]. Improvement of parameters used for evaluation of the LV function is observed: LVEF, end-diastolic volume, end-diastolic dimension, end--systolic volume and, end-systolic dimension [19]. CRT upgrades RV function which can be confirmed in the traditional echocardiographic examination by Tei-index and tricuspid annual systolic motion (TAPSE) evaluation [20–23]. The use of tissue Doppler imaging led to similar conclusions [24]. Recent studies seem to give a contrary opinion on how CRT influences markedly depressed RV function. It was proven, that significantly weakened RV function predisposes to a lack of improvement after CRT in terms of NYHA class, 6-minute walking test distance and LVEF [25–27]. On the other hand, it was shown that CRT may lead to improvement of the RV function regardless of its volume and size [27].

It is still not clear whether patients with AF may benefit from CRT to the same extent as the ones without arrhythmia. A systemic meta-analysis of 23 observational studies involved 7 495 patients with CRT. It showed that AF was associated with lower rate of clinical response and increased risk of death in comparison to individuals without AF [28]. Contrary to that, some papers suggest that in patients with permanent AF treated with CRT a significant clinical improvement, comparable to the sinus rhythm group, can be achieved [29–31]. The effect of CRT has not been compared between patients with paroxysmal and persistent AF. One of few reports regarding this subject showed, that CRT leads to similar

prognostic, symptomatic and echocardiographic benefits in patients with paroxysmal, permanent AF and sinus rhythm [32]. On the other hand, lower incidence of AF episodes in patients with CRT and paroxysmal AF can be achieved as a result of improved LV and left atrial function [33, 34]. Specific evaluation of the CRT benefit in patients with paroxysmal and permanent AF in the context of the RV function is lacking.

The right heart in patients with atrial fibrillation

Relationship between the dimensions and function of the right atrium and occurrence of AF is properly documented [35, 36]. Enlargement and remodeling of both atria predispose to development of AF. Studies using stress echocardiography showed the prognostic values of the right atrium area to maintain sinus rhythm after AF ablation [35]. However, on the basis of existing evidence, area of the right atrium can be used to assess prognosis only in patients with non-valvular AF [36].

RV function in patients with AF is not well defined, although the importance of heart rhythm for the RV function is well known [4, 37]. Elongation of the cardiac cycle, thus lengthening the RV filling time results in diastolic volume increase and consequently in increase in RV ejection fraction (RVEF) [4]. Study in a small group of patients showed that in permanent AF the RV function presented as RVFAC is dependent on the RR interval preceding it and on the mean heart rate [37]. In another study in patients with paroxysmal and persistent AF RV end-systolic diameter was markedly larger than in individuals without the arrhythmia (20 ± 2 mm in AF group vs. 17 ± 2 mm in control group). RVEF in patients with AF was deteriorated in comparison to healthy individuals: 69 ± 8%, 60 ± 11% and 74 ± 3% in paroxysmal, permanent and control group, respectively. End-diastolic diameter did not differ between the groups [38]. The results of this study showed, that level of NT-proBNP, which is a marker of cardiac dysfunction, correlated with RVEF and was significantly higher in patients with permanent AF [39].

Atrial fibrillation in the failing right heart

Atrial tachyarrhythmias are most common in patients with depressed RV function. Their occurrence may be one of clinical manifestations of RV failure [2].

The results of the ACAP-HF Program in a group of 900 patients showed that occurence of the RV failure in the course of decompensated chronic LV failure increases the risk of AF development. Weakened RV function was one of the strongest predictors of AF occurrence. Two thirds of AF cases occurred in patients with RV dysfunction. Moreover, patients with RV dysfunction had higher risk of cardiac readmission and mortality than control individuals: 4.7%/year vs. 2.9%/year. Finally, patients with deteriorated RV function who developed AF proved to be the ones with the worst prognosis in the whole group [40]. In patients with RV infarction hemodynamic stabilization depends also on maintenance of sinus rhythm [41].

Atrial flutter or AF may also lead to hemodynamic compromise and worsen prognosis in patients with chronic RV failure, not only of ischemic etiology [2, 42–44]. In pulmonary hypertension the RV is exposed to pressure overload. Hemodynamic and clinical stabilization depends mainly on its function. In patients with chronic pulmonary hypertension persistent AF is associated with significantly increased risk of death due to RV failure. Occurrence of supraventricular arrhythmias is often preceded by deterioration of RV function. In one of the studies mortality due to RV failure in patients with permanent AF was approximately 80%. By contrast, in patients in whom sinus rhythm was restored after first episode of AF, total mortality was 6% [42].

Observation of patients with congenital heart defects showed that abnormalities of RV structure predispose to developing atrial arrhythmias more than left-sided lesions. In one of the studies, risk of development of any atrial arrhythmia was circa 61% in the RV-defect group and 55.4% in the LV-defect group [43]. An isolated atrial septal defect, if not corrected, may cause a long lasting volume overload of the RV. The RV tolerates volume overload better than pressure overload. Dilatation of its cavity tends to normalize within 1–24 months after closure of the defect. However, in some patients it may last for over 5 years and AF may develop even decades after successful closing procedure [44]. A large analysis in a group of over 38 000 patients with various congenital heart defects affecting the right heart showed 15% prevalence of atrial arrhythmias in adult patients. Other studies reported from 25% to 30% arrhythmia prevalence. Risk factors for AF or other atrial arrhythmia development included age, CHF and right atrial size. The results of the studies showed that atrial arrhythmias are associated with 2.5 fold increased risk of morbidity and 50% increased risk of mortality [45, 46].

Right ventricular pacing and heart failure

RV pacing creates specific clinical conditions that affect RV function and may induce AF and CHF [47, 48]. As a non-physiological mode of pacing it causes a loss of atrio-ventricular systolic synchrony. It has been proven that RV pacing exacerbates HF in patients with LVEF < 40% [49]. In post-MI patients, RV apical pacing was associated with a worsening of LV function, suggesting at the same time that among MI survivors, the need for pacemaker may be a marker of worse outcome [50]. Cumulative RV pacing > 2% and EF < 40% are independent predictors of ventricular tachycardia/ /ventricular fibrillation occurrence, higher mortality rate and HF hospitalizations in patients after cardioverter-defibrillator implantation [51].

The deleterious effects of RV apical pacing have been attributed to the abnormal electrical and mechanical activation, secondary to this form of pacing. During RV apical pacing, the electrical wave front propagates mainly through the myocardium rather than via the His-Purkinje conduction system. This is characterized by wave front breakthrough at the interventricular septum and latest activation of the infero-posterior base of the LV. The paced region contracts early at a time of low load, but then it is stretched in systole as the lateral wall finally contracts. Asynchronous myocardial contraction significantly decreases the stroke volume and right-shifts the LV end-systolic pressure – volume relationship. Thus, RV apical pacing leads to ventricular dyssynchronization, systolic and diastolic ventricular dysfunction, increase of wall stress, and energetic inefficiency [52]. It also causes interventricular dyssynchronization, as the RV contracts earlier than in a physiological model.

It has been proven that right ventricular outlet tract (RVOT) pacing can reduce unfavorable effects and slow down cardiac remodeling caused by permanent RV pacing. In one of the studies clinical and echocardiographic benefits observed in the RVOT group after 7 years of pacing were reflected by lower NT-proBNP concentration in this group of patients [53].

CHF, as a result of impaired LV function, has been given close attention, but RV function during RV pacing still requires further research.

Imaging and assessment of right ventricular structure and function

Cardiac magnetic resonance imaging (CMR) is a precise method of RV structure and function assessment. It enables accurate quantification of heart chambers volumes and calculation of myocardial mass from tracing epicardial and endocardial borders and multiplying them by myocardial density [54]. Valvular regurgitant volumes and shunt severity can also be precisely calculated using velocity-encoded cine images [55]. Intra-observer and inter-observer variability in CMR RV measurements ranges from 3% to 6% and 4% to 9%, respectively [56, 57]. Because of its good spatial and temporal resolution, CMR-derived volumes and EF are considered the gold standard regarding other imaging modalities. However, CMR is not routinely used because of its still limited availability and high costs.

Computed tomography (CT) has also been considered a reliable method to assess the RV. One of the studies showed, that there was little variability between the measurements of RV by the two observers (kappa = 0.895–0.980, $p < 0.05$). There was also good correlation between all parameters obtained by CT and CMR ($p < 0.001$): RV end-diastolic volume (RVEDV 108.5 ± 21.9 mL, 113.5 ± 24.8 mL, r = 0.944), RV end-systolic volume (RVESV 69.8 ± 33.4 mL, 73.2 ± 35.4 mL, r = =0.972), RV systolic volume (RVSV 39.0 ± 13.2 mL, 40.2 ± 13.3 mL, r = 0.977), RV cardiac output (RVCO 2.6 ± 0.7l, 2.6 ± 0.7l, r = 0.958), and there was no significant difference between CT and CMR measurements in RVEF (38.8 ± 19.1%, 39.1 ± ± 19.3%, r = 0.990, n = 50, t = –0.677, $p > 0.05$). The results of the study suggest, that 320-slice volume cardiac CT is an accurate non-invasive technique to evaluate RV function [58]. Other paper shows that cardiac CT fully quantifies LV size and function. However, RV quantification with cardiac CT requires optimized contrast opacification of the RV [59]. When compared to radionuclide ventriculography, CT seems to be a reliable method for assessment of the RV as well. It has been shown that RVEF can be accurately assessed with ECG-gated multidetector computed tomography (MDCT) using commercially available software [60].

RV can also be accurately evaluated by echocardiography. Simple and established method for determining global RV function in the traditional two-dimensional imaging is an evaluation of TAPSE measured in M-mode presentation of the apical 4-chamber view [61]. The method is reliable also in patients with AF [62]. Percentage change in the surface of the RV (RVFAC) can also be assessed [8]. It is possible to calculate the ratio of RV global function (MPI, myocardial performance index). Additionally, tissue Doppler imaging enables measurement of peak systolic velocity of the tricuspid

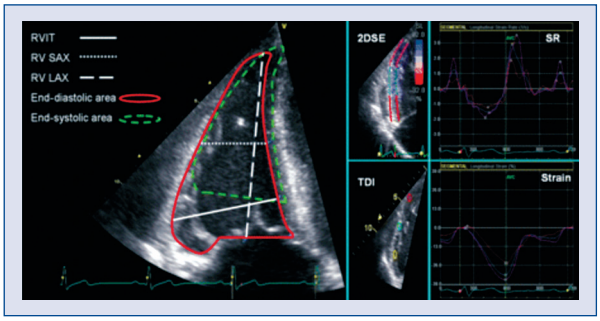

Figure 1. Right ventricular measurements [68].

annulus (S'), which is another complementary method for evaluating RV function [63]. RV free wall segmental motion and thickening are the points of evaluation as well. RV function can be assessed by eye-balling (Fig. 1).

A long-lasting overload of the RV can be indirectly estimated from the main pulmonary artery extension, measured in the parasternal short--vascular projection. The current flow through the pulmonary valve measured with use of pulse Doppler wave enables analysis of its spectrum and assessment of its acceleration time. Acceleration time is shortened when pulmonary arterial pressure is increased, RV systolic function is weakened and/or in paradoxical septal contraction occurring in acute conditions: acute pulmonary embolism and RV MI.

For the quantification of interventricular dyssynchrony, which is a predictor of outcome in CRT, conventional Doppler techniques are used. The electromechanical delay is calculated as the time from the onset of QRS complex to the onset of pulmonary systolic flow (RV electromechanical delay) or aortic systolic flow (LV electromechanical delay). The time difference between RV and LV electromechanical delay represents interventricular dyssynchrony [50].

Three-dimensional imaging echocardiography is a novel method which allows to obtain more reliable data on the structure and function of the RV, particularly on its RVESV and RVEDV. Additionally, the RVEF determined by this method shows good correlation with measurements in MRI [64–66].

According to current guidelines of the European Society of Echocardiography, assessment of the right heart structure should include RV, right atrium dimensions and areas in short, long parasternal axis and in apical 4-chamber view. RV function should be evaluated with use of the above mentioned markers of its systolic and diastolic function and estimation of EF in the traditional 2D imaging. In addition, EF in 3D imaging can be assessed [67]. Table 1 presents RV dimensions and parameters of its function under normal conditions according to the guidelines.

Table 1. Reference limits for recommended measures of right heart structure and function (from Guidelines for Echocardiographic Assessment of the right Heart in Adults: A Report from the American Society of Echocardiography) [67].

Abnormal	Chamber dimensions
RV basal diameter	> 4.2 cm
RV subcostal wall thickness	> 0.5 cm
RVOT PSAX distal diameter	> 2.7 cm
RVOT PLAX proximal diameter	> 3.3 cm
RA major dimension	> 5.3 cm
RA minor dimension	> 4.4 cm
RA end-systolic area	> 18 cm^2
Systolic function	
TAPSE	< 1.6 cm
Pulsed Doppler velocity peak at the annulus	< 10 cm/s
Pulsed Doppler MPI	> 0.40
Tissue Doppler MPI	> 0.55
Fractional area change	< 35%
Diastolic function	
E/A ratio	< 0.8 or > 2.1
E/E' ratio	> 6
Deceleration time	< 120 ms
3-dimensional imaging	
RVEF	< 44%

RV — right ventricle; RVOT — right ventricular outlet tract; PLAX — parasternal long axis; PSAX — parasternal short axis; RA — right atrium; TAPSE — tricuspid annulus plane systolic excursion; MPI — myocardial performance index; RVEF — right ventricular ejection fraction

Conclusions

RV function is crucial for outcome in patients with CHF, especially in patients with significant LV dysfunction. Its dysfunction often coexists with AF, not only in HF, but also in a number of conditions that depress RV acutely or chronically. RV structure can be precisely assessed with novel modalities of echocardiography, CT and magnetic resonance imaging.

Conflict of interest: none declared

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