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Review

Echocardiographic assessment of the right ventricle: Impact of the distinctly load dependency of its size, geometry and performance

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ABSTRACT

Right ventricular (RV) size, shape and function are distinctly load-dependent and pulmonary load is an important determinant of RV function in patients with congestive heart failure (CHF) due to primary impaired left ventricular function and in those with pre-capillary pulmonary hypertension (PH). In a pressure overloaded RV, not only dilation and aggravation of tricuspid regurgitation, but also systolic dysfunction leading to RV failure (RVF) can occur already before the development of irreversible alterations in RV myocardial contractility. This explains RV ability for reverse remodeling and functional improvement in patients with post-capillary and pre-capillary PH of a different etiology, after normalization of loading conditions.

There is increasing evidence that RV evaluation by echocardiography in relation with its loading conditions can improve the decision-making process and prognosis assessments in clinical praxis. Recent approaches to evaluate the RV in relation with its actual loading conditions by echo-derived composite variables which either incorporate a certain functional parameter (i.e. tricuspid annulus peak systolic excursion, stroke volume, RV end-systolic volume index, velocity of myocardial shortening) and load, or incorporate measures which reflect the relationship between RV load and RV dilation, also taking the right atrial pressure into account (i.e. "load adaptation index"), appeared particularly suited and therefore also potentially useful for evaluation of RV contractile function. Special attention is focused on the usefulness of RV echo-evaluation in relation to load for proper decision making before ventricular assist-device implantation in patients with CHF and for optimal timing of listing procedures to transplantation in patients with end-stage pre-capillary PH.

1. Introduction

Right ventricular (RV) performance is a reflection of contractility, preload and afterload, also being influenced by valvular function, heart rhythm, ventricular interdependence and synchrony of ventricular contraction. Acting more as a volume pump, the RV tolerates less pressure overload than volume overload and has higher sensitivity to afterload changes than the left ventricle (LV). Thus, RV performance is distinctly afterload-dependent and both reduction in systolic function and ventricular enlargement occur much earlier in the pressure overloaded RV than in the pressure overloaded LV [1,2].

Pulmonary load is an important determinant of RV function in patients with congestive heart failure (CHF) due to primary impaired LV function and in those with pre-capillary pulmonary hypertension (PH). Indeed, the most common causes of RV failure (RVF) are left-sided HF and pre-capillary PH [3,4,5]. Because of the high variability in RV adaptation to pressure overload and its ability to improve during afterload reduction by a LV assist-device (LVAD) in patients with end-stage CHF or after lung transplantation (LTx) in patients with pre-capillary PH, the reliability of RV assessment is paramount for prognostic evaluations and therapeutic decisions [1,6,7].

Echocardiography (echo) is the mainstay for RV evaluation but the markedly load-dependency of RV size, geometry and function induce particular challenges in RV echo-assessment. The review summarizes knowledge about RV echo-assessment in patients with afterload-induced RVF. Special attention is focused on interpretation of RV geometry and function measurements in relation to loading conditions, aiming to evaluate RV adaptability to load and to predict the reversibility of RVF by reduction of RV afterload.

2. Pathophysiology of RV response to pressure overload

Pressure and volume overload are main causes of RV dysfunction [8,9]. However, because the compliant thin-walled RV tolerates less pressure than volume overload, the most frequent initial cause of RVF is the

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pressure overload due to high filling pressures of the LV and/or high pulmonary vascular resistance (PVR) [3,4,8].

The onset of pressure overload-induced RVF can be abrupt like in acute massive pulmonary embolism because the RV is poorly constructed to compensate for acute afterload increase [8]. Its ability to increase systolic pressure acutely is highly limited and mainly dependent on increasing RV end-diastolic volume (preload). This mechanism fails already when the acute rise in PVR requires a mean pulmonary arterial pressure (PAP) of about 40 mm Hg to perfuse the pulmonary bed leading to rapid development of RVF [8,10]. More often the onset of pressure overload-induced RVF is gradual or insidious as in CHF due to primary impaired LV function, pulmonary arterial hypertension (PAH) or PH associated with hypoxic lung diseases [10]. Chronic pressure overload is better tolerated by the RV and myocardial hypertrophy often allows the rise of mean PAP beyond 60 mm Hg without development of RVF [3,9]. RVF due to RV pressure over-loading (increased PVR) is most often diagnosed in patients with left-sided HF and in those with pre-capillary PH of different etiologies [8,10].

In CHF, due to primary impaired LV function, cardiac output (CO) reduction induces elevation of LV diastolic pressure followed by pulmonary venous pressure increase with pulmonary congestion. Since both ventricles are in a circuit their stroke volume (SV) must be equal and any LV-SV reduction necessitates a corresponding RV-SV reduction to prevent pulmonary edema. Thus, already before any alteration in RV myocardial contractility, the high pulmonary venous pressure and contraction of small pulmonary arteries induced by LV-SV reduction will increase PVR, which reduces the highly afterload-dependent RV pump-function and consequently also the RV-SV [3,4,8]. In addition, imbalanced neurohormonal reactions to the impaired LV function increase also the RV preload by excessive renal fluid-retention with subsequent increase of venous return. RV overloading induces RV enlargement with tricuspid valve-ring dilation followed by tricuspid regurgitation (TR) [3,4]. Impaired LV function also affects RV systolic and diastolic function via the shared septum and other features of ventricular inter-dependence [3,4]. Thus, although CHF syndrome usually emerges from a failing LV, secondary RV dysfunction also contributes to CHF and association of severe LV and RV failure is often found in end-stage CHF [11]. However, increased renal retention of sodium and water with subsequent edema formation and hepatomegaly as a response to CO reduction can occur with pure left-sided HF without hemodynamic evidence of right-sided HF (RHF) [3].

In pre-capillary PH of different etiologies the high afterload induces RV geometry alterations, massive hypertrophy and TR. However, in early stages, the RV tends to remain adapted to the increased afterload with increased contractility, myocardial hypertrophy and no relevant dilation [3,4,12]. In more advanced stages, RV systolic function cannot remain matched to afterload and RV dilation with TR plus systolic and diastolic dysfunction progressively develops [9]. Nevertheless, RV dilation with progressive increase of TR and reduction of RV performance induced by high afterload often occur already before alterations in myocardial contractility [12]. Even so, due to the additional volume-overloading, the dilated RV looses more rapidly its ability to increase contractility in response to increased afterload [9]. Overloading-induced right atrial (RA) dilation and dysfunction may also contribute to worsening of RV function [13].

By increasing RV wall tension, both RV systolic pressure increase and RV dilation will progressively impair RV coronary blood flow and induce myocardial ischemia which finally reduces RV contractility regardless whether the pressure-overloading was initially induced by post-capillary PH (left-sided HF) or pre-capillary PH [3]. Because the RV is more tolerant of ischemia in comparison to the LV, ischemia-induced reduction in myocardial contractility can remain reversible for a long time. However, chronic excessive afterload and preload with massive RV myocyte stretch, hypertrophy and sarcomeric stiffening can induce myocyte death (especially by apoptosis) and irreversible pathologic RV remodeling with increased myocardial fibrosis and stiffness, which will further alter RV function and also reduce the reversibility of RV dysfunction [3,5,8,14,15]. In patients with CHF, severe irreversible remodeling can be sufficient for persistence and progression of RVF independent of the neurohormonal status of the patient [15]. In such cases there is a high probability for persistence of RVF even after LVAD implantation.

3. Reversibility of right ventricular failure by RV afterload reduction

The high load-sensitivity of RV performance, related to the impact of preload and afterload on RV geometry and TR explains RV ability for reverse remodeling and functional improvement after normalization of loading conditions [16,17]. Sustained afterload increase is associated with homeometric adaptation (without chamber dilation) of RV contractility in order to maintain an adequate SV [8]. After exhaustion of homeometric adaptation and adaptive remodeling (hypertrophy) the RV responds to afterload increase by heterometric adaptation (Starling's law) which allows maintenance of SV at the cost of increased RV end-diastolic volume (EDV) with increase in right heart filling pressures [8,14]. Afterload-induced RVF arises when the RV becomes unable to maintain the CO in response to metabolic demand without heterometric adaptation (without elevated filling pressures), but RVF usually remains reversible for long time. In CHF patients, mechanical LV-support often induces acute PVR reduction accompanied by RV geometry and function improvement. Long-term LVAD-support usually further reduces the PVR facilitating RV reverse remodeling and functional recovery [18]. However, post-LVAD RVF is more frequent than RVF persistence after LTx in patients with pre-capillary PH (except congenital heart diseases) or after pulmonary end-arterectomy in patients with chronic thromboembolic PH (CTEPH); in both situations PVR normalization is usually followed by nearly complete RV reverse remodeling with normalization of contractile function [2,16]. LVAD implantation is rarely followed by complete RV recovery and severe post-operative RVF associated with high mortality occurs in about 25% of LVAD recipients [2,16]. The less predictable RV improvement in CHF patients receiving an LVAD is mainly related to differences in the etiology of myocardial injury which induced the CHF and differences in the direct impact of the initial myocardial injury on RV myocardium as well as the impact of ventricular inter-dependence changes induced by LV unloading on RV geometry and function.

4. Echo-assessment of the RV: limitations and impact of RV load

Because of difficulties in RV volume measurements (complex three-dimensional RV shape, limited echo-window due to RV position, limited definition of RV endocardial surface), 2D-echo-derived RV ejection fraction (EF) is less reliable and therefore, 2D-echo-derived RV volume and EF calculations are not recommended for clinical use or as a standard reference in research [19,20]. Three-dimensional (3D) echocardiography obviates geometric assumptions and is superior to standard 2D-echo for estimation of RV volumes and EF. Thus, although 3D-approaches are technically challenging and yet not widely available, they are currently recommended as method of choice RV volume and EF calculations [19–21]. As a complex summary-parameter that integrates several underlying physiological components, including ventricular size, contractile function and afterload, EF can be a valuable index for assessment of RV performance, but due to its afterload dependency, EF cannot be used as an index of myocardial contractility [20–23].

Currently, 2D-echo is still preferentially used for assessment of RV function because it allows easy measurements of RV fractional area-change (FAC_{RV}) and tricuspid annulus peak systolic excursion (TAPSE), which provide similar information to RVEF [24]. TAPSE and FAC_{RV} require no geometric assumptions and TAPSE has also the advantage to be obtained even with poor image quality [6,21,25]. However, FAC_{RV} neglects the contribution of RV outflow-tract to overall systolic function. Its measurements also show high inter- and intraobserver variability and are hampered by suboptimal endocardial definition (especially in systole) [21,