EDITORIAL COMMENT

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Size and function of the atria

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Assessment of atrial size, anatomy and function is important in various clinical settings and can be performed with different imaging techniques. Assessment of atrial size provides important prognostic information and is routinely performed with transthoracic echocardiography. Information on regional atrial function can also be provided by transthoracic echocardiography and is important in the setting of treatment of atrial fibrillation. Catheter ablation procedures for atrial fibrillation require accurate imaging of the left atrium and surrounding structures. Intracardiac echocardiography, multislice computed tomography (CT), and cardiovascular magnetic resonance imaging (CMR) may provide detailed information on right and left atrium and pulmonary vein anatomy [1].

The atria perform three different functions during the various phases of the cardiac cycle, i.e. serving as reservoir during systole, passive conduit during early

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Department of Cardiology, Leiden University Medical Centre, Albinusdreef 2, 2333 ZA Leiden, The Netherlands e-mail: E.E.van_der_Wall@lumc.nl diastole, and booster function during late diastole. In this issue of the journal, Willens et al. report a descriptive study on the effects of age and pulmonary arterial hypertension on all three phases of right atrial function by measuring changes in right atrial volume during the cardiac cycle using two-dimensional transthoracic echocardiography [2]. In their study, Willens et al demonstrated that passive right atrial emptying was lower and booster function was greater in older healthy subjects and in patients with pulmonary arterial hypertension. Reservoir function was higher in pulmonary hypertension. These changes are analogous to the changes reported in the left atrium in response to increased afterload and impaired relaxation [3].

The active contractile component of the atria has an important role in patients with ventricular dysfunction to augment ventricular volume. Augmented atrial booster function is one of the mechanisms compensating for decreased early filling in patients with reduced ventricular compliance. Loss of atrial contraction as a result of atrial fibrillation or ventricular pacing reduces cardiac output by approximately 15–20%. The ability to optimally redistribute ventricular filling among reservoir, conduit and booster pump functions is a potentially important adaptation that may occur in the atria in response to changing dynamics.

In patients with congenital or valvular heart disease, increased ventricular stiffness or decreased ventricular compliance may cause an increased atrial pressure. The increased atrial wall tension leads to stretch and dilation of the atrial myocardium. Thus, atrial volume increases with severity of diastolic dysfunction.

Atrial volume is a barometer of ventricular filling pressure and reflects the burden of diastolic dysfunction in subjects without atrial fibrillation or significant valve disease. Atrial enlargement carries important clinical and prognostic implications [1, 3]. In large population- based studies it has been demonstrated that left atrial size is an important predictor of cardiovascular outcome. Elevation of filling pressure is uniformly found in the presence of symptomatic congestive heart failure. Evidence for a prognostic role for left atrial volume to predict incident congestive heart failure is emerging [3]. In the general population left atrial size has been determined to be a predictor of stroke and death. The relationship between left atrial size and death has been demonstrated in high-risk groups, such as patients with dilated cardiomyopathy, left ventricular dysfunction, atrial arrhythmias, acute myocardial infarction, patients undergoing valve replacement for aortic valve stenosis and mitral regurgitation [3].

Left atrial size is potentially modifiable with medical therapy, but whether left atrial size reduction translates to improved outcomes remains to be established.

Atrial fibrillation is another important factor associated with atrial dilatation. Atrial enlargement may be the consequence of atrial fibrillation, but atrial enlargement may also lead to atrial fibrillation [1, 3]. To what extent the shifts among reservoir, conduit and booster pump functions of the atria contribute to the development of atrial arrhythmias warrants further study.

The atria play an important role in adult congenital heart disease [4]. Atrial function is often altered due to longstanding pressure or volume overload. Moreover, many adult patients with a congenital heart defect have had intracardiac surgery with the use of cardiopulmonary bypass in the past. These patients will all have a right atrial scar, the sequel of the atriotomy necessary for the cannulation for the cardiopulmonary bypass or necessary for visualisation of intracardiac structures. This scarring leads to loss of atrial compliance and compromises atrial reservoir function. Moreover, atrial function can be substantially modified by some forms of surgical correction or palliation, such as the Mustard or Senning procedure in patients with transposition of the great arteries or a Fontan operation in patients with univentricular hearts [5-10]. Cardioplegic arrest may also cause deterioration of right atrial function. Altered caval vein flow patterns indicate abnormal atrial filling and the reservoir function of the right atrium is diminished postoperatively. Both the longstanding atrial overload and the atrial scarring form substrates for atrial arrhythmias. Loss of atrial reservoir function (reduced compliance) and the loss of booster function (atrial fibrillation) is poorly tolerated in patients with a compromised ventricular function.

In patients with right ventricular hypertrophy, such as in pulmonary arterial hypertension or in corrected tetralogy of Fallot, the forceful right ventricular contraction leads to an increased downward displacement of the tricuspid valve annulus, thereby increasing atrial filling from the pulmonary veins during systole. The antegrade pulmonary flow during diastole is decreased due to a decreased compliance. The higher filling pressures will cause a more forceful atrial contraction. Right ventricular volume overload, due to pulmonary regurgitation and/or tricuspid regurgitation in Fallot patients will also elevate right atrial pressure and volume. Either due to elevated right ventricular pressure or as a result of increased right ventricular volume, the right atrial wall is often stretched in patients with corrected tetralogy of Fallot. This will not only lead to an altered atrial function, but also to a high prevalence of atrial arrhythmias. In case of atrial fibrillation, the contribution of the atrial booster will then disappear, which may substantially worsen the hemodynamic status in these patients [11-22].

The longterm prognosis of adult patients with congenital heart disease is insufficiently known and largely dependent on right-sided cardiac function [23–26]. Noninvasive follow-up modalities are of utmost importance for these patients, in order to allow early detection of pressure or volume overload of the right ventricle before the occurrence of failure or irreversible myocardial damage. Early detection of right ventricular dysfunction may allow timely medical management and preventive surgery. CMR provides the diagnostic imaging of choice for the evaluation of right sided heart anatomy and function in congenital heart disease by its potential to obtain

both anatomic detail and flow quantification [27–29]. CMR is useful for detecting intra- and extra-cardiac conduit obstruction following cardiac surgery in complex congenital heart disease. Technical advances of CMR are the excellent spatial resolution, the characterisation of myocardial tissue, multiplane versatility and the potential for three-dimensional imaging. By means of dobutamine stress cardiac reserve may be determined in various forms of congenital heart disease. In addition, the use of plasma neurohormones may play a role in early detection of cardiac dysfunction in asymptomatic patients with congenital heart defects [30-35].

The utility of atrial volume and function for monitoring cardiovascular risk and for guiding therapy becomes increasingly clear and may prove to have an important impact in clinical decision making.

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