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REVIEW

Imaging in pulmonary hypertension: Focus on the role of echocardiography

Imagerie dans l'hypertension pulmonaire : le rôle de l'échocardiographie

Pamela Moceri*, Delphine Baudouy, Olivier Chiche, Pierre Cerboni, Priscille Bouvier, Claire Chaussade, Emile Ferrari

Service de cardiologie, hôpital Pasteur, CHU de Nice, 30, avenue de la Voie-Romaine, BP69, CS 51069, 06001 Nice cedex 1, France

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Summary Patients with pulmonary hypertension must be evaluated using a multimodality approach to ensure a correct diagnosis and basal evaluation as well as a prognostic assessment. Beyond the assessment of pulmonary pressures, the echocardiographical examination allows the evaluation of right ventricular adaptation to elevated afterload. Numbers of variables are commonly used in the assessment of the pulmonary hypertension patient in order to detect changes in right heart geometry, right-to-left interaction and right ventricular dysfunction. Whereas an isolated change in one echocardiographical variable is not meaningful, multiple echocardiographical variable modifications together provide accurate information. In this review, we will link pulmonary hypertension pathophysiological changes with echocardiographical indices and describe the clinical implications of echocardiographical findings.

Abbreviations: 2D, Two-dimensional; 3D, Three-dimensional; dPAP, Diastolic pulmonary artery pressure; IVA, Isovolumic acceleration; IVCc, Isovolumic contraction velocity; LV, Left ventricle/ventricular; LVOT, Left ventricular outflow tract; mPAP, Mean pulmonary artery pressure; MPI, Myocardial performance index; PA, Pulmonary artery; PAH, Pulmonary arterial hypertension; PAP, Pulmonary artery pressure; PR, Pulmonary regurgitation; RA, Right atrium/atrial; RAP, Right arterial pressure; RV, Right ventricle/ventricular; sPAP, Systolic pulmonary artery pressure; TAPSE, Tricuspid annular plane systolic excursion; TR, Tricuspid regurgitation; VTI, Velocity-time integral.

* Corresponding author.
E-mail address: moceri.p@chu-nice.fr (P. Moceri).

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Résumé L'imagerie cardiaque multi-modalité est indispensable à l'évaluation diagnostique et pronostique des patients atteints d'hypertension pulmonaire. Bien plus que la mesure des pressions pulmonaires, l'échocardiographie permet l'évaluation de la fonction ventriculaire droite traduisant l'adaptation du cœur droit à l'élévation des résistances pulmonaires. Alors que la modification isolée d'une variable échographique n'est que peu informative, celle de plusieurs paramètres échographiques est plus souvent pertinente. Dans cette revue, nous décrivons les modifications physiopathologiques du cœur droit et du couple VD-AP dans l'hypertension pulmonaire par l'approche échographique et soulignons l'intérêt clinique de ces différents paramètres échocardiographiques.

Background

Pulmonary hypertension is a progressive and severe pulmonary, vascular and cardiac disorder. Pulmonary hypertension is defined by an increase in mean pulmonary artery pressure (mPAP) to ≥25 mmHg at rest as determined by right heart catheterization, the 'gold standard' [1]. Usually, patients are screened with echocardiography (Table 1), and right heart catheterization allows measurement of cardiac output and differentiation between pre- and postcapillary pulmonary hypertension. Pulmonary hypertension can be divided into subsets sharing similar underlying pathophysiology, detailed in Fig. 1 [2].

Although the degree of pulmonary hypertension characterizes this condition, it does not correlate with symptoms or survival. Right ventricular (RV) physiology (RV mass and function) is closely related to functional class, exercise capacity and survival [3,4]. Advanced therapies, which aim to decrease pulmonary vascular resistance, usually significantly improve RV function, but the observed decrease in mPAP is only moderate.

Table 1 Echocardiographical screening for patients at risk of pulmonary hypertension.

TR V_{max}	
≤2.8 m/s + echocardiographical risk factors	PH possible
2.9–3.4 m/s	PH possible
>3.4 m/s	PH likely
Echocardiographical risk factors suggesting pulmonary hypertension	
Dilated right ventricle/right atrium	
Increased PR velocity	
Pulmonary acceleration time <105 ms	
TAPSE <20 mm	
'D-shaped' interventricular septum	

PH: pulmonary hypertension; PR: pulmonary regurgitation; TAPSE: tricuspid annular plane systolic excursion; TE: tricuspid regurgitation.

In clinical practice, ultrasound is by far the most common RV imaging modality, especially in the setting of pulmonary arterial hypertension (PAH). Transthoracic echocardiography provides a number of variables for evaluating right heart haemodynamics (Table 2). It is critical to recognize early signs of PAH with echocardiography in order to reduce the delay between first symptoms and time of diagnosis. Echocardiography is particularly suitable in pulmonary hypertension studies as it is non-invasive, cost-effective and widely available.

Thus, the aim of this paper is to describe the cardiac alterations related to pulmonary hypertension, and to review and discuss the extent to which echocardiography plays a key role in early diagnosis and prognosis in pulmonary hypertension.

Right ventricular anatomy and pathophysiology

The right ventricle (RV) is the most anterior chamber of the heart and can be divided into three chambers: the inlet, the apex and the infundibulum. In comparison with the left ventricle (LV), the RV is characterized by prominent trabeculations that limit the accuracy of contour tracing to delineate the endocardial border. A complex shape, opposite to the ellipsoidal shape of the LV, also characterizes the RV [5]. When viewed in cross section, the RV is crescent shaped, whereas it appears triangular when viewed longitudinally or from the side. However, the RV shape is influenced by RV-LV interactions, depending on the position of the interventricular septum. In normal conditions, the RV volume is larger than the left ventricular (LV) volume, whereas the RV mass represents approximately only one fifth of the LV mass [6]. The myofibre arrangement is also particular, in that almost only two muscle layers compose the RV wall: the superficial layer (circumferential) and the deep layer (longitudinally arranged), thus explaining in normal RVs the predominance of longitudinal contraction, with no intermediate circumferential layer.

Regarding RV contraction physiology, three individual components lead to normal contraction: the inward motion of the free wall; longitudinal shortening; and the traction of the free wall, secondary to LV contraction [7]. In consequence, RV contraction relies mostly on LV-RV interaction and longitudinal deformation.