Right ventricular function in critically ill patients

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ABSTRACT

The right ventricular function is crucial for maintaining hemodynamic stability in critically ill patients who suffer from sudden increases of right ventricular pressure overload and/or severely decreased right ventricular contractility. The morphological and functional assessment of the right ventricle is usually performed by bedside echocardiography and hemodynamic measurements with a pulmonary artery catheter. The therapeutic approach to patients with right ventricular failure includes measures to decrease right ventricular afterload and to improve its coronary perfusion and contractility.

Key words: right ventricle, failure, systolic function

BASIC ANATOMIC, PHYSIOLOGIC AND PATHOPHYSIOLOGIC DATA

Sixty years ago, the right ventricle (RV) was considered a passive conduit for blood flow and only a "weak sister" of the strong and powerful left ventricle (1). More than thirty years later, the RV function was revisited and it became clear, that RV failure is responsible for shock in patients with massive pulmonary embolism and RV myocardial infarction (2-4).

The thin free wall of the RV forms the anterior and inferior part of the RV and the posterolateral wall represents the intraventricular septum, which normally bulges into the RV. The RV blood supply occurs during systole and diastole; the oxygen extraction is very high, and ATP amount is lower than in the LV. The free wall is very compliant and is capable of maintaining low systemic pressure and preventing pulmonary edema in the case of LV failure. These characteristics make the RV very sensitive to hypoxia. The geometry of the RV is quite complex with clearly separated inflow and outflow tracts. Because of the complex geometry, the LV models are not valid for the RV. The contraction of the RV is less synchronous than that of the LV and occurs as a peristaltic wave starting in the inflow tract and propagating through the apex to the outflow tract.

In physiologic conditions, when the pulmonary vascular resistance is low, RV function is indeed of secondary importance. On the other hand, in pathologic conditions with increased pulmonary vascular resistance and/or severely impaired RV contractility, RV function becomes essential for maintaining adequate pulmonary blood flow. The basic consequence of the sudden increase of RV afterload and/or decreased contractility is RV dilation. Since a dilated RV reduces the LV volume in the rigid pericardial sac, the LV filling is reduced with consequently decreased LV stroke volume and mean arterial pressure. Arterial hypotension provokes RV ischemia that further reduces RV systolic function (5).

The RV function is therefore relevant in patients with chronic lung diseases, primary pulmonary vascular disease, pulmonary thrombembolic disorders, sepsis related acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) and after cardiac surgery.

Patients with chronic lung diseases frequently present with chronic cor pulmonale, which is characterized by RV free wall hypertrophy and various degrees of dilation with preserved contractility. Patients with chronic cor pulmonale and RV failure have a poor prognosis because of limited systemic oxygen transport. In patients with primary pulmonary hypertension, a progressive increase in afterload causes RV hypertrophy and dilation. The decrease of RV performance is related to increased mortality. The risk stratification in patients with pulmonary thrombembolism is usually based on RV function or dysfunction (6). Concomitant RV infarction is observed in 1 out 3 of patients with inferior myocardial infarction and has a negative impact on survival. RV dilation and failure is related with increased mortality in patients with ARDS (7). In patients who have undergone cardiac surgery, RV failure is responsible for some early deaths, since RV is very sensitive to ischemia in the early postoperative period. Also after cardiac transplantation, persistent RV failure is related with a poor prognosis.

ASSESSMENT OF RIGHT VENTRICULAR FUNCTION

Generally, we can evaluate RV function by various methods. Clinically, we consider predominant acute RV failure in patients with distended jugular veins, hypotension and clear lungs. Electrocardiography (ECG) can indicate RV hypertrophy, but its specificity and sensitivity is low. The same is true for the typical ECG signs (S1Q3T3) of acute RV overload in pulmonary embolism (8). Nevertheless, the right precordial ECG leads (V3R and V4R) show typical ST elevation in patients with RV infarction and should be recorded in every patient with inferior myocardial infarction. Chest X ray is usually not diagnostic for RV failure and contrast angiocardiography is used only for diagnostic assessment. Currently magnetic resonance imaging is the most accurate and sensitive method for assessment of RV morphology and function, but it cannot be used at the bedside in critically ill patients. The same is true for nuclear imaging methods.

The assessment of RV function in critically ill patients therefore relies on hemodynamic measurements and especially on echocardiography. In patients monitored by pulmonary artery catheter, RV ejection fraction (REF) and end diastolic volume (REDV) can be measured in addition to global hemodynamic variables (right atrial pressure, pulmonary artery pressures, pulmonary artery occlusion pressure, cardiac output and mixed venous oxygen saturation). Both variables are important, since

RVEF should be interpreted according to RVEDV in normal individuals and in critically ill patients. RVEDV is also a clinically useful predictor of preload and can predict preload recruitable increase of cardiac output (9).

Echocardiography (transthoracic and transesophageal) is the mainstay of RV morphological and functional assessment. Since the calculations for LV are not valid for the RV because of its irregular and complex shape, visual estimation of RV function is usually used as a first orientation. The problems of RV visualization are usually due to retrosternal position of the RV, and difficult endocardial surface definition because of the trabeculated structure of the RV myocardium. RV dilation is the universal RV response to increased afterload and impaired contractility; therefore, we should measure RV dimensions. RV dimensions greater than 4 cm in the short axis and more than 9 cm in the long axis and dilation of the outflow tract more than 3.5 cm are usually considered as severely abnormal. However, more often we compare RV dimension with the dimensions of the LV. If the RV is enlarged, but smaller than the LV, the RV dilation is mild. If both ventricles are equal, the RV dilation is moderate, and when the RV is larger than the LV, the dilation is severe. After assessment of dimensions, we should measure the free wall thickness. The RV hypertrophy, which is a marker of chronic pressure overload, is present if the thickness is more than 5 mm and calculated RV mass is more than 35g/m2. In the parasternal, short axis

the eccentricity index of the LV should be estimated. A diastolic D-shape of the LV is characteristic in patients with volume RV overload, on the other hand a systolic LV D-shape is characteristic for RV pressure overload.

RV systolic function can be expressed by RVEF. Unfortunately, echocardiography is less accurate for RVEF and its correlation with MRI is weak. Tricuspid annular plane systolic displacement (TAPSE) as a measure of longitudinal RV function is frequently used. The normal values are 20 – 25 mm and the correlation with RVEF is good (10). Alternatively, we can measure tricuspid annular systolic velocity by tissue Doppler for estimation of right ventricular systolic function (11).

Central venous pressure (CVP) as a measure of RV preload and systolic pulmonary artery pressure (PAPs) as a measure of RV pressure afterload should be determined as an integral part of RV function assessment. Both pressures can be measured invasively or estimated by echocardiography. The inferior vena cava diameter and its change during inspiration are measured in the subcostal view by echocardiography. We can estimate PAPs by measuring the velocity of tricuspid regurgitation or pulmonary artery systolic velocity acceleration time.

TREATMENT OF RIGHT VENTRICULAR FAILURE

Treatment strategies for RV failure rely on the patophysiological grounds of RV func-

tion and dysfunction and include volume and preload optimization, afterload reduction and improvement of contractility and perfusion (12). These goals are achieved by careful volume management, vasopressor and inotrope support and use of selective pulmonary vasodilators. Fluid boluses should be used for preload optimization, but require careful monitoring of cardiac output. Vigorous fluid administration should be avoided, since it can decrease LV output because of further RV dilation and a leftward shift of the intraventricular septum (13). Norepinephrine is beneficial in hypotensive and tachycardic patients with compromised RV coronary perfusion, but in patients with RV failure and preserved arterial pressure dobutamine or levosimendan is preferred (14-16). RV afterload can be decreased by inhalation of nitric oxide or use of prostacyclins, endothelin receptor antagonists and inhibitors of phosphodiesterase.

Beside specific measures, principles of general supportive ICU treatment (thrombembolism and peptic ulcer prophylaxis, infection prevention, early nutrition, glucose control and proper sedation and analgesia, correction of low hemoglobin level) should be implemented. Mechanical ventilation can have potential adverse hemodynamic effects and therefore we should use the lowest tidal volume, plateau pressure, positive end expiratory pressure needed to provide adequate oxygenation (17).

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