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Commentary

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MRI Catheterization in Cardiopulmonary Disease

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Diagnosis and prognostication in patients with complex cardiopulmonary disease can be a clinical challenge. A new procedure, MRI catheterization, involves invasive right-sided heart catheterization performed inside the MRI scanner using MRI instead of traditional radiographic fluoroscopic guidance. MRI catheterization combines simultaneous invasive hemodynamic and MRI functional assessment in a single radiation-free procedure. By combining both modalities, the many individual limitations of invasive catheterization and noninvasive imaging can be overcome, and additional clinical questions can be addressed. Today, MRI catheterization is a clinical reality in specialist centers in the United States and Europe. Advances in medical device design for the MRI environment will enable not only diagnostic but also interventional MRI procedures to be performed within the next few years. ***CHEST 2014; 145(1):30–36***

Catheterization plays a central role in the diagnostic evaluation of patients with intracardiac shunts, complex congenital heart disease, pulmonary vascular disease, cardiomyopathy, cor pulmonale, and heart failure. Elevated pulmonary arterial pressure is a hemodynamic finding common to all these disease processes. Establishing the cause of pulmonary hypertension requires complex diagnostic algorithms involving numerous noninvasive and invasive tests. Today, catheterization remains the best available investigative tool for confirming diagnosis, quantifying severity of disease, and determining treatment. Guidelines[1-3] recommend catheterization be performed in all patients with symptoms and echocardiographic suspicion of pulmonary hypertension or prior to initiation of therapy.

Hemodynamic parameters shown to be associated with an increased risk of death include increased mean pulmonary artery pressure, increased mean right atrial pressure, and decreased cardiac index.[4] Most guidelines define pulmonary hypertension based on elevated mean pulmonary artery pressure alone. However, with disease progression, mean pulmonary artery pressure may actually fall as the right ventricle fails. For this reason, pulmonary vascular resistance is a more compelling standard for the diagnosis of pulmonary hypertension because it takes into account both pressure and flow. Resistance measurement has not entered guideline care because accurate measurement of pulmonary flow is not possible in the presence of tricuspid regurgitation, typical in these patients, using conventional thermodilution techniques. It is important to consider that the cause of elevated pulmonary pressure is not always pulmonary vascular pathology. For example, in patients with high transpulmonary flow, such as in pregnant women or in patients with anemia, sepsis, thyrotoxicosis, or intracardiac shunt, pulmonary pressure can be elevated in the presence of normal pulmonary vascular resistance. Provocative testing with vasodilators, such as inhaled nitric oxide plus 100% oxygen, is recommended because vasoreactivity predicts responsiveness to prostacyclin analogs, endothelin-receptor antagonists, or phosphodiesterase

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type 5 inhibitors and also identifies those patients with a better prognosis.

Limitations of Catheterization

Catheterization techniques for measurement of cardiac output (necessary for the quantification of pulmonary vascular resistance) are subject to error. The thermodilution technique is inaccurate in patients with low flow states, intracardiac shunts, or significant valvular regurgitation (eg, tricuspid regurgitation).[5] Thermodilution should, therefore, be avoided in patients with pulmonary hypertension who often have significant tricuspid regurgitation. The Fick technique is inaccurate in conditions in which venous and arterial hemoglobin saturation values approach each other (eg, with large intracardiac shunts or during vasoreactivity testing with nitric oxide and 100% oxygen). The Fick principle incorporates total body oxygen consumption, but measuring oxygen consumption is labor intensive. Instead, most laboratories estimate oxygen consumption using assumptions such as LaFarge and Miettinen,[6] based on body surface area, age, and heart rate. If the Fick principle is used, this estimate can introduce significant error into cardiac output calculations.

Limitations of Noninvasive Evaluation

Echocardiography is typically the first test performed in patients with suspected pulmonary hypertension. The established method for estimating pulmonary artery pressure with echocardiography involves measuring the maximal velocity of tricuspid regurgitation.[7] Alternative markers of pulmonary hypertension, including pulmonary artery acceleration time,[8] flattening of the interventricular septum, and pulmonary regurgitant velocity, have been proposed in the absence of tricuspid regurgitation.[9] Calculating pulmonary vascular resistance is not possible because echocardiography cannot accurately measure left atrial pressure and arguably cannot accurately measure transpulmonic flow and because errors are common in measuring the Doppler envelope of the tricuspid regurgitation jet. Evaluating the right ventricle with echocardiography is difficult because of its complex geometry[10] and its anatomic position beneath the sternum, exaggerated in those most affected.[11] Echocardiography is further limited by poor acoustic windows in patients with large body habitus or with advanced lung disease (eg, COPD).

Cardiac MRI is the best available imaging modality for structural and functional assessment of the right ventricle.[12] Right ventricular dysfunction is a determinant of functional capacity and prognosis in pulmonary artery hypertension,[13] chronic heart failure,[14] myocardial infarction, and mitral regurgitation.[15] Whereas pulmonary artery pressure does not strongly correlate with symptoms or survival, right ventricular stroke volume and end diastolic dimensions by MRI are independent predictors of mortality in patients with primary pulmonary hypertension.[16] The 6-min walk test of functional capacity, used as the primary end point in most pulmonary hypertension pharmaceutical trials,[17] correlates better with right ventricular function than with pulmonary artery pressure. Pulmonary arterial stiffness, measured with MRI by relative cross-sectional area in systole and diastole, also predicts mortality in patients with pulmonary hypertension.[18] In patients with chronic heart failure, right ventricular function correlates better with exercise capacity than does left ventricular function,[19] and elevated pulmonary artery pressure and right ventricular dysfunction have been shown to be independent predictors of mortality.[14] Therefore, it is critical to combine hemodynamic variables with a functional evaluation of the right ventricle and the pulmonary vasculature.

MRI Catheterization Offers Additive Diagnostic Value Compared With Stand-alone MRI or Conventional Catheterization

MRI catheterization addresses all the previously mentioned limitations by simultaneously measuring the pressures, flows, and volumes of the desired cardiac chambers. Volumetric analysis of cardiac function (such as end-diastolic and end-systolic volumes) or MRI (velocity-encoded, also known as phase-contrast) flow techniques can measure stroke volume and pulmonic or systemic cardiac output. In addition, intracardiac shunts (Qp:Qs) can be identified from mismatched pulmonary artery and aortic flows. Hybrid parameters, such as pulmonary vascular resistance or pulmonary artery compliance, can be derived from assimilation of MRI measurements and catheterization pressures.

Although pulmonary vascular resistance can be measured accurately at rest using conventional Fick oximetric techniques, provoked pulmonary vascular resistance measurements during administration of inhaled oxygen and nitric oxide are inaccurate. Muthurangu and colleagues[20] elegantly demonstrated this discordance in 2004 using invasive pressure and cardiac output derived from phase-contrast MRI at baseline and during nitric oxide plus 100% oxygen vasoreactivity testing. Although unproven, similar inaccuracy can be expected during other stress-provoked measures of pulmonary vascular resistance.

Other parameters such as pulmonary artery compliance can be derived only from a combined approach.[21]