Noninvasive Assessment of Pulmonary Artery Pressures
Moving Beyond Tricuspid Regurgitation Velocities

Sheldon E. Litwin, MD

Pulmonary hypertension (PHTN) has been classified according to the world health organization as (1) pulmonary arterial hypertension (including idiopathic, familial, and associated with collagen vascular disease or HIV), (2) PHTN with left heart disease, (3) PHTN associated with lung diseases and/or hypoxemia, (4) PHTN caused by chronic thrombotic and/or embolic disease, and (5) miscellaneous.1

Diseases and/or hypoxemia, (4) PHTN caused by chronic arterial hypertension (including idiopathic, familial, and associated with collagen vascular disease or HIV), (2) PHTN with left heart disease, (3) PHTN associated with lung diseases and/or hypoxemia, (4) PHTN caused by chronic thrombotic and/or embolic disease, and (5) miscellaneous.1

In addition, to accurately measure the mean right atrial pressure, it is necessary to know or estimate the mean right atrial pressure. However, to be useful there must be a large enough regurgitant volume to produce a Doppler signal that allows accurate measurement of the peak TR velocity. This is frequently not the case, even in settings where PHTN is likely.6 In addition, to accurately measure pulmonary artery systolic pressure from TR velocities, it is necessary to know or estimate the mean right atrial pressure. Noninvasive assessment of right atrial pressure is challenging and often inaccurate.7 Because of these and other problems, traditional echocardiographic Doppler assessment of pulmonary artery pressures may be unfeasible or may lead to overestimation or underestimation of pulmonary pressures in up to 40% of individual patients.4,5,7 In short, the traditional echocardiographic approach is not as consistent or as accurate as it needs to be for research studies or clinical decision making.4,8,9

Chronic PHTN is associated with loss of elasticity in the pulmonary vascular bed. Stiffening of the vasculature results in greater reflection of pressure waves from bifurcations in the pulmonary vessels during the pulsatile ejection of blood from the right ventricle. Summation of forward and reflected pressure waves will result in truncation of the forward flow.10

In 1983, Kitabatake et al11 reported that acceleration time (AT) or the ratio of AT to ejection time (ET) measured from the pulsed-wave Doppler signal in the right ventricular outflow tract decreased with increases in mean pulmonary artery pressure. They found a very strong inverse correlation between AT/ET and log mean pulmonary artery pressure ($r = -0.90$). Subsequently, several groups have successfully used similar concepts and parameters to assess pulmonary artery pressures or pulmonary vascular resistance.12 Although the studies are not directly comparable, correlations between the spectral Doppler measures of right ventricular outflow systolic time intervals and invasively measured pulmonary artery pressures appear to be at least as strong as those between TR velocities and invasively measured pulmonary pressures. Good-quality pulsed-wave Doppler signals of the right ventricular outflow tract can be obtained in the vast majority of patients—probably more frequently than we can obtain measurable TR velocities. Given the relative ease of recording and quantifying right ventricular outflow tract Doppler signals, it is surprising that these measures have not caught on as part of routine clinical practice.

In this issue of Circulation Cardiovascular Imaging, Thibault et al13 describe the utility of pulsed-wave Doppler measurements at the tips of the pulmonary leaflets in murine models of acute and chronic PHTN. In the field of PHTN research, as in many others, transgenic mouse models will have a significant role in helping to unravel the pathophysiology of the disorder. In addition to many different transgenic models of elevated pulmonary artery pressures, PHTN may be induced acutely or chronically in small animals by using pharmacological interventions.14 Animal models that are useful in predicting which new treatments may have beneficial effects in humans would be of great value. Although mice have many advantages when used as models to study the pathophysiology of PHTN, it is difficult to obtain apical

The opinions expressed in this article are not necessarily those of the editor or of the American Heart Association.

From the University of Utah Hospitals and Clinics and Salt Lake City VA Medical Center, Salt Lake City, Utah.

Correspondence to Sheldon E. Litwin, MD, Cardiology 4A100 SOM, University of Utah, 30 North 1900 East, Salt Lake City, UT 84121. E-mail Sheldon.Litwin@hsc.utah.edu

(Circ Cardiovasc Imaging. 2010;3:132-133.)

© 2010 American Heart Association, Inc.

Circ Cardiovasc Imaging is available at http://circimaging.ahajournals.org

DOI: 10.1161/CIRCIMAGING.110.945121

132
imaging windows in this species. The lack of apical windows limits the ability to accurately measure TR velocities. Even more problematic, mice tend to have very small amounts of TR, even when they have PHTN. Given the limitations of measuring TR velocities, an alternative noninvasive technique that is capable of serially assessing pulmonary pressures in mice would be welcome. The results from the current study look very promising. Using a high-frequency ultrasound sound, clearly defined spectral Doppler signals from the proximal pulmonary artery were obtained in the mice. The Doppler waveforms showed marked shortening of the pulmonary artery AT and AT/ET as right ventricular systolic pressures rose. This was true in both acute (due to infusion of the thromboxane agonist U-46619) and chronic (interleukin-6 overexpression) models of PHTN. The sensitivity and specificity of pulmonary artery AT and AT/ET for detecting right ventricular systolic pressure >2 SD above normal were high (100% and 86%). Interobserver variability in the measurements was quite good (<6%). Using the ratio of AT to ET mitigates the effects of different heart rates that are likely to be present in long-term therapeutic studies. Interestingly, AT/ET is similar in mice and humans despite heart rates that are almost 10-fold higher in mice.

The findings of the current study should stimulate new research using murine models of PHTN. It will be important to prove whether the pulmonary artery ejection time intervals can accurately detect small or moderate reductions in right ventricular or pulmonary artery systolic pressure during chronic treatment. Similarly, it appears that measurement of right ventricular outflow tract or pulmonary artery systolic pressure during chronic treatment. Similarly, it appears that measurement of right ventricular outflow tract or pulmonary artery systolic pressure during chronic treatment.

Sources of Funding
This work was supported in part by a grant from the Department of Veterans Affairs.

Disclosures
None.

References

Key Words: Editorials ◼ echocardiography ◼ hypertension ◼ pulmonary imaging ◼ pulmonary heart disease ◼ systolic time intervals
Noninvasive Assessment of Pulmonary Artery Pressures: Moving Beyond Tricuspid Regurgitation Velocities
Sheldon E. Litwin

doi: 10.1161/CIRCIMAGING.110.945121

Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2010 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circimaging.ahajournals.org/content/3/2/132

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Cardiovascular Imaging can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Cardiovascular Imaging is online at:
http://circimaging.ahajournals.org//subscriptions/