submit

Member Login:

submit

Forgot Password? | Register for an Account

Advances in PH Journal

Pulmonary Venous Hypertension: A Pulmonologist's Perspective

Francisco Soto

//

Reviews

Sign in to add a review

0 comments
Leave a Comment

Vol 10, No 1 (Spring 2011)

Increased pulmonary hypertension (PH) awareness in the general public and among health providers has led to an increase in referral of patients who are found to have elevated estimates of pulmonary artery systolic pressure (PASP) on a transthoracic echocardiogram (TTE), without other strong features suggestive of precapillary PH (pulmonary arterial hypertension; PAH). Some of these patients undergo TTE as part of their workup for unexplained dyspnea that appears out of proportion to their other comorbidities. Many of these patients are older individuals with underlying conditions such as systemic hypertension (HTN), diabetes mellitus (DM), coronary artery disease (CAD), and obstructive sleep apnea (OSA). While some of them may have PAH, accumulated experience in the PH community suggests that many of these patients will be ultimately found to have elevated left ventricular (LV) filling pressures and impaired LV relaxation as the cause of their dyspnea and elevation of pulmonary pressures on TTE.[1,2] These findings are consistent with a form of Group 2 PH termed LV diastolic dysfunction, more recently termed heart failure with preserved left ventricular systolic function (HFpEF) (Table 1).[2] Other forms of Group 2 PH, defined as “pulmonary hypertension owing to left-sided heart disease,” include LV systolic dysfunction or left-sided valvular disease.[2] For the purpose of this article, the term pulmonary venous hypertension (PVH) will be used to refer to Group 2 PH, HFpEF. Being able to accurately

discriminate PAH (precapillary; Group 1 PH) from PVH (postcapillary; Group 2 PH) is critical to determine and apply the appropriate treatment course. This is a difficult, yet frequently encountered clinical dilemma, which can fall into a “gray zone” with respect to clinical classification. We will provide important clinical features that should heighten the clinician's awareness and suspicion of this rapidly growing phenomenon. These features are critical when trying to differentiate PAH (Group 1 PH) from PVH (Group 2 PH).

EPIDEMIOLOGY

HFpEF accounts for a significant proportion of unexplained chronic dyspnea.[3] While the precise incidence of PH in HFpEF is not known, it is recognized to be the most common form of PH seen clinically.[1] A study by Rifaie and colleagues reported a PH prevalence of approximately 20% in elderly patients with HFpEF with female gender, atrial fibrillation, and early mitral annular diastolic velocity (e') being independent predictors of PH.[4] Others have suggested a much higher PH prevalence in HFpEF. Using an estimated PASP >35 mm Hg on echocardiography to define PH, Lam et al reported a PH prevalence of 83% in a community-based study of 244 patients with HFpEF.[5] A direct correlation was seen between PASP and pulmonary capillary wedge pressure (PCWP), derived echocardiographically. Authors found PH to be a strong predictor of mortality with a hazard ratio of 1.3 per 10 mm Hg (P<0.001). Clinical characteristics distinguishing PAH patients from PH in HFpEF have also been recently described.[6] In a study by Thenappan and colleagues, PH-HFpEF patients were older, had higher prevalence of cardiovascular comorbidities, had worse exercise capacity and renal function, and more frequently had left atrial enlargement.

DIAGNOSTIC CRITERIA FOR PH OWING TO LEFT HEART DISEASE (GROUP 2): HFpEF

PAH typically occurs as a manifestation of progressive obliteration of the pulmonary arterial circulation.[7] PH due to left heart disease is thought to occur mostly when left-sided ventricular or valvular diseases produce a chronic increase in left atrial pressure, which results in passive backward transmission of the pressure leading to increased pulmonary venous pressure.[2] This has been referred to as reactive PH, as pulmonary artery pressure will decline in response to optimization of fluid status. However, chronic elevation of left atrial pressure can result in pulmonary arterial remodeling and lead to less reactive changes, which may manifest as “PH out of proportion to left heart disease” (discussed in Hemodynamics section) and/or “fixed” PH.[8,9]

Current guidelines[10] propose that a diagnosis of PAH by right heart catheterization (RHC) can be made when the mean pulmonary arterial pressure (mPAP) is ?25 mm Hg and the left atrial or LV filling pressure, measured as PCWP or LV end diastolic pressure (LVEDP), is ?15 mm Hg. A diagnosis of PVH is made when the mPAP is ?25 mm Hg with LV filling pressure >15 mm Hg. In this scenario, the pulmonary vascular resistance (PVR) is usually normal (<3 Wood units). These hemodynamic criteria can be helpful to discriminate between clear-cut cases of PAH vs PVH. Unfortunately these criteria may be less helpful when evaluating more complex cases such as elderly patients with PH suggested by TTE and who have concomitant systemic comorbidities, or those with suspected PH but who have borderline LV

filling pressure numbers that do not clearly place the patient in either the PAH or PVH category. Trying to resolve these situations with mixed clinical features can be quite daunting for the provider if the only parameter to classify them as either type of PH is a marginal LV filling pressure of < or >15 mm Hg. For example, if taken in isolation, a 1 mm Hg difference in LVEDP could potentially lead to the final decision regarding whether a patient will receive expensive and potentially cumbersome PAH-specific therapies (in the case of mPAP >25 mm Hg and LV filling pressure of 15 mm Hg), or will receive traditional preload and afterload reduction agents (as in the case of mPAP >25 mm Hg and LV filling pressure of 16 mm Hg) and will lead to much less emotional stress given the more “benign” nature of a PVH diagnosis. Further, there may be a mixed picture of PAH and PVH where this cutoff is less reliable and additional tools including detailed clinical history, other hemodynamic parameters, and provocative testing all become critical.

IMPLICATIONS OF AN ACCURATE PAH VS PVH DIAGNOSIS

The PAH vs PVH diagnostic dilemma carries more than just classification implications since the treatment approach to these entities is dramatically different.[11] For example, PAH-specific therapies have been shown to improve symptoms and survival in patients with Group 1 PH. However, they are not approved for Group 2 PH, where HFpEF belongs, and can lead to worsening heart failure symptoms including pulmonary edema (Table 1).[12,13]

Table 1:

Updated Clinical Classification of Pulmonary Hypertension (Dana Point, 2008)

1 Pulmonary arterial hypertension (PAH)
1.1 Idiopathic PAH
1.2 Heritable
1.2.1 BMPR2
1.2.2 ALK1, endoglin (with or without hereditary hemorrhagic telangiectasia)
1.2.3 Unknown
1.3 Drug- and toxin-induced
1.4 Associated with
1.4.1 Connective tissue diseases
1.4.2 HIV infection
1.4.3 Portal hypertension
1.4.4 Congenital heart diseases
1.4.5 Schistosomiasis
1.4.6 Chronic hemolytic anemia
1.5 Persistent pulmonary hypertension of the newborn
1? Pulmonary veno-occlusive disease (PVOD) and/or pulmonary capillary hemangiomatosis (PCH)
2 Pulmonary hypertension owing to left heart disease
2.1 Systolic dysfunction
2.2 Diastolic dysfunction
2.3 Valvular disease

3 Pulmonary hypertension owing to lung diseases and/or hypoxia
3.1 Chronic obstructive pulmonary disease
3.2 Interstitial lung disease
3.3 Other pulmonary diseases with mixed restrictive and obstructive pattern
3.4 Sleep-disordered breathing
3.5 Alveolar hypoventilation disorders
3.6 Chronic exposure to high altitude
3.7 Developmental abnormalities
4 Chronic thromboembolic pulmonary hypertension (CTEPH)
5 Pulmonary hypertension with unclear multifactorial mechanisms
5.1 Hematologic disorders: myeloproliferative disorders, splenectomy
5.2 Systemic disorders: sarcoidosis, pulmonary Langerhans cell histiocytosis: lymphangioleiomyomatosis, neurofibromatosis, vasculitis
5.3 Metabolic disorders: glycogen storage disease, Gaucher disease, thyroid disorders
5.4 Others: tumoral obstruction, fibrosing mediastinitis, chronic renal failure on dialysis

An additional consideration relates to the emotional burden of these diagnoses. This can be particularly true for the PVH patient mislabeled as having PAH, given the prospect of the psychological impact of such a misdiagnosis (eg, concerns about life expectancy), potential side effects of PAH therapies, need for lifestyle modifications in order to safely administer these treatments (eg, precautions for use of continuous subcutaneous or intravenous infusions), and unnecessary personal and societal financial burden. On the other hand, failing to identify a true PAH patient and mislabeling the case as PVH carries the risk of delaying treatments that could dramatically improve exercise tolerance, quality of life, and life expectancy. Eventually, many of the PAH cases that are initially misclassified as PVH will experience clinical deterioration and will most likely be correctly classified as PAH once they undergo reevaluation of their diagnosis. Unfortunately, a delay in PAH diagnosis can be detrimental since placebo vs treatment trials of PAH suggest that patients who face treatment delay (eg, placebo arm) might not be able to reach the same potential clinical response when compared to similar PAH patients who start treatment without delay.[11,14]

ESTABLISHING A CLINICAL SUSPICION: THE ROLE OF PRETEST PROBABILITY

With respect to PH, little has been documented regarding pretest probability (PRETEP), but the idea is one that potentially could address the challenges of differentiating PAH from PVH. While the concept of PRETEP has only been around for the last 30 years,[15] physicians have always used its basic principles when trying to determine the likelihood that a given patient

might have a specific disease based on signs, symptoms, and history. PRETEP is defined as the probability of the target disorder before a diagnostic test result is known (www.cebm.net). The PRETEP is especially useful for: interpreting the results of a diagnostic test; selecting one or more diagnostic tests; choosing whether to start therapy (a. without further testing [treatment threshold]; or b. while awaiting further testing and deciding whether it's worth testing at all [test threshold]).

Based on the initial assessment of a case, a number that quantifies the “likelihood of disease” is generated to help establish the clinical suspicion or PRETEP. Once a PRETEP is determined, a test will typically be ordered to see if the result will transform the PRETEP or clinical suspicion number into: a high enough number that is strongly suggestive of presence of disease or into a low enough number that makes disease presence much less likely and almost excludes it. The likelihood “number” that is generated after a test result is interpreted in the context of the initial symptoms and clinical suspicion is called post-test probability (POSTTEP). A specific POSTTEP number that will trigger a treatment intervention will vary depending on the provider experience threshold, the disease being suspected, and the immediate and long-term risks associated with initiating or delaying treatment. For example, a POSTTEP of 90% for suspicion of pulmonary embolism will likely prompt the clinician to initiate anticoagulation unless specific contraindications are present. On the other hand, a clinician might want a POSTTEP of 100% before deciding to initiate chemotherapy for a given cancer.

BUILDING A PRETEP FOR PVH

Based on the current guidelines, if LV filling pressure is greater than 15 mm Hg in the absence of significant valvular disease or LV systolic dysfunction, and the transpulmonary gradient (TPG) is <15 mm Hg, mPAP of 25 mm Hg or higher is likely the result of elevated LV filling pressures. These cutoffs are useful but alone are usually not enough to solve the PAH vs PVH dilemma. Therefore, establishing a PRETEP for PVH (or PAH) before diagnostic tests such as echocardiogram or RHC are obtained during the workup of an individual patient may be useful to help guide workup.

Table 2 provides a list of variables that, if present, should enhance the likelihood that elevated LV pressures are the likely explanation for patients' symptoms and PH findings. Their presence should prompt the provider to consider PVH as a likely diagnosis.

Table 2:

Findings That Increase the Clinical Suspicion for PVH

Medical History
History of systemic hypertension (particularly if not optimally controlled)
Diabetes mellitus
Coronary artery disease
Obstructive sleep apnea
Atrial fibrillation

Symptoms*
Orthopnea
Paroxysmal nocturnal dyspnea (PND)
*Orthopnea and PND can also occur in PAH, but are usually in late stages of PAH and the diagnosis is typically clear-cut at this stage.

Electrocardiogram
Lack of right axis deviation
Lack of right atrial enlargement
Evidence of left atrial enlargement
Evidence of left ventricular hypertrophy

Echocardiogram Features
Absence of right heart chamber enlargement
Evidence of left atrial enlargement
Presence of left ventricular hypertrophy
Impaired diastolic relaxation indices
Elevated left ventricular filling pressures as determined by E/E? ratio (ie, >15 mm Hg)
Modest elevation of pulmonary pressures (ie, 50s rather than 80s)

Computed Tomography of the Chest
Absence of right heart chamber enlargement
Evidence of left atrial enlargement

THE ROLE OF PRETEP IN COMMON PVH CASE SCENARIOS

Case Scenario 1

A 72-year-old woman underwent a TTE as part of a workup for dyspnea of unknown etiology. She has history of systemic hypertension for over 20 years and mild DM, which is managed with oral agents. She has experienced progressive decline in exercise tolerance and worsening dyspnea for over 3 years. She currently has WHO functional class III symptoms. The TTE suggests elevation in pulmonary pressures of around 55 mm Hg. The right ventricle size and function appear completely normal. The right atrium is minimally enlarged and the left atrium is moderately enlarged. There are no comments on diastolic function on the TTE report.

While it is certainly possible that this patient might have idiopathic PAH or another form of precapillary PAH, if this were the case, one would expect to see some evidence of significant right ventricular chamber strain or hypertrophy, especially after 3 years of progressive dyspnea. Therefore, the lack of right heart dysfunction should add extra “points” to the clinical suspicion for PVH as the leading diagnosis. The echocardiogram revealed left atrial enlargement, which is likely the passive response to chronically elevated LV filling pressures. Although there is no comment about LV diastolic dysfunction on the echocardiogram report, one should not always assume that this was addressed adequately. Finally, the advanced age, female gender, history of systemic hypertension, and diabetes lend to the suspicion of HFpEF.

Case Scenario 2

A 68-year-old woman with history of systemic HTN and OSA is evaluated for progressive dyspnea. TTE estimates a systolic pulmonary pressure of 51 mm Hg. PH is suspected and workup is initiated. Several tests are ordered, including a computed tomography (CT) of the chest with IV contrast (Figure 1).

Figure 1:

Computed tomography findings suggestive of pulmonary venous hypertension.

A CT chest was done looking for evidence of thromboembolic disease. This was not found but detailed assessment of the heart chamber images were obtained, which revealed lack of right heart involvement and showed significant left atrial dilatation. Such findings in a patient who already has risk factors for left heart dysfunction (ie, advanced age, HTN, OSA) should raise the suspicion for PVH as the cause of her PH and dyspnea.

CT chest findings in Figure 2 correspond to a different patient. In this figure, there is severe enlargement of right heart chambers with small left atrial size. These are the findings that one would expect to see in precapillary PH (PAH) as compared to postcapillary PH (PVH).

Figure 2:

Computed tomography findings suggestive of pulmonary arterial hypertension.

Case Scenario 3

A 58-year-old man with history of systemic HTN, CAD, and OSA underwent a RHC after a TTE showed estimated PASP of 62 mm Hg. He complained of progressive dyspnea for at least 4 years. His RHC revealed a PASP of 57 mm Hg, pulmonary artery diastolic pressure of 23 mm Hg, and mPAP of 34 mm Hg. His PCWP was 14 mm Hg. Based on these hemodynamic findings and a negative workup for other etiologies of PH, the patient's case was labeled as idiopathic PAH. He was started on an oral pulmonary vasodilator agent. After 3 months of such therapy, his dyspnea actually worsened, prompting the clinician to recommend adding another oral agent to his treatment. The family then requested a second opinion. Based on the lack of clinical response and the multiple risk factors for left heart disease, his new provider sought to evaluate his case in more detail before adding another agent for PAH treatment. Four-chamber apical view images of his initial TTE (before pulmonary vasodilator was started) are shown below (Figure 3).

Figure 3:

Apical four-chamber echocardiographic view suggestive of pulmonary venous hypertension.

The lack of right heart enlargement plus the presence of mild left atrial enlargement shown in the TTE image all increase the clinical suspicion for postcapillary PH (PVH) as the likely etiology for the PH findings seen on the RHC. Based on these findings and clinical suspicion, the pulmonary vasodilator was held and a repeat RHC was done a couple of weeks later. Pulmonary artery pressures were similar to the initial RHC numbers. PCWP was again around 14-15 mm Hg. However, a pigtail catheter was placed in the LV this time and a LVEDP of 22 mm Hg was found. No gradient was found between the pulmonary artery diastolic pressure (23 mm Hg) and the LVEDP (22 mm Hg). The significant elevation in resting LVEDP (normal: 6-10 mm Hg) and the lack of pressure gradient between diastolic pressure and LVEDP[16,17] was consistent with PVH. Another possible maneuver in the borderline case where the response to advanced PH therapy does not align well with the clinical picture would be to give a fluid challenge in the catheterization lab while monitoring the filling pressures. Based on these findings, pulmonary vasodilator agents were permanently discontinued, and the management focused on dealing with his underlying risk factors for PVH and addressing preload and afterload reduction.

DISCUSSION

Current hemodynamic guidelines used to differentiate between idiopathic PAH and PVH are helpful, but the importance of including other known risk factors in making the determination needs to be emphasized. As the current population ages, and as the prevalence of other comorbid diseases such as DM, HTN, obesity, and OSA increases, PH providers will be faced with this diagnostic classification dilemma with greater frequency.[5,18] Most PH providers will eventually develop their own protocols and algorithms to address possible PVH cases. These approaches might include performing a RHC in every patient who has evidence of elevation in pulmonary pressures by TTE (ie, >40 mm Hg) in order to be safe. More experienced providers might follow a more conservative approach. For example, when an elderly patient is found to have elevated PASP by TTE, the presence of risk factors for left heart disease and the absence of signs of right heart involvement by TTE might prompt the clinician to aggressively address those PVH risk factors for a few months before the case is reevaluated with a repeat TTE or before a RHC is performed. Potential treatment interventions for PVH risk factors might include optimal blood pressure control, volume optimization, aggressive OSA management, weight reduction, and an exercise program. If at the time of the follow-up evaluation the symptoms have clearly improved or the TTE shows reduction in PASP without evidence of right heart involvement, the same conservative approach could be maintained while keeping a close follow-up of such patient. On the other hand, if follow-up data reveal signs of clinical or echocardiographic deterioration, full PH workup including RHC might be warranted. Unfortunately, since clear-cut guidelines that guarantee 100% safety and successful results with the approach described above are lacking, determining a strong

pretest probability based on comprehensive evaluation of the available data might be the best initial investment of time and effort.

If the suspicion for PVH is present but the provider elects to proceed with a RHC for a more thorough evaluation of suspected PH, additional interventions during the catheterization could be considered to help unmask or confirm impaired LV relaxation.[16,17] Such interventions include: a volume challenge, which is especially useful in patients who have been diuresed prior to the RHC; an exercise challenge (upper or lower extremity); a nitroprusside vasodilator trial; detailed assessment of filling pressure waveforms after a pulmonary vasodilator challenge (ie, nitric oxide—see Ask the Expert section for potential risks). The protocols for these interventions are not fully standardized and vary between institutions. A detailed summary of expected hemodynamic and waveform changes after such interventions is described elsewhere.[17] Finally, the clinician should have a low threshold to obtain a LVEDP measurement in addition to PCWP measurement when a PVH diagnosis cannot be confidently made after an exhaustive workup. This is very important since recent data point to the risk of solely relying on the use of PCWP to determine LV filling pressures.[19,20] Based on those studies, a large number of PVH cases could be misclassified as PAH because of PCWP measurement limitations.

CONCLUSION

In the near future, PH providers will face more and more challenging cases of PVH that will present as possible idiopathic PAH. Unfortunately, until new specific hemodynamic parameters that can accurately discriminate between PAH vs PVH are proposed and validated, many of these patients will be inappropriately misclassified and will endure unnecessary stress and treatment, and potentially detrimental effects.[12,13,19,20] Thus, the ability to establish a very clear pretest probability for PVH at the beginning of the workup might be one of the best tools available to face such challenging cases.

References

1. Oudiz, RJ. Pulmonary hypertension associated with left-sided heart disease. Clin Chest Med. 2007;28(1):233–241, x.
2. Simonneau, G, Robbins, IM, Beghetti, M . Updated clinical classification of pulmonary hypertension. J Am Coll Cardiol. 2009;54(1 Suppl):S43–S54.
3. Hoeper, MM, Barberà, JA, Channick, RN . Diagnosis, assessment, and treatment of non-pulmonary arterial hypertension pulmonary hypertension. J Am Coll Cardiol. 2009;54(1 Suppl):S85–S96.
4. Rifaie, O, El-Damanhory, H, Amr, M, Nammas, W. Prevalence and predictors of pulmonary hypertension in elderly patients with isolated diastolic heart failure. Kardiol Pol. 2010;68(6):655–661.
5. Lam, CS, Roger, VL, Rodeheffer, RJ, Borlaug, BA, Enders, FT, Redfield, MM. Pulmonary hypertension in heart failure with preserved ejection fraction: a community-based study. J Am Coll Cardiol. 2009;53(13):1119–1126.
6. Thenappan, T, Shah, SJ, Gomberg-Maitland, M . Clinical characteristics of pulmonary

hypertension in patients with heart failure and preserved ejection fraction. Circ Heart Fail. 2011;4(3):257–265.
7. Tuder, RM, Abman, SH, Braun, T . Development and pathology of pulmonary hypertension. J Am Coll Cardiol. 2009;54(1 Suppl):S3–S9.
8. Hogg, K, Swedberg, K, McMurray, J. Heart failure with preserved left ventricular systolic function; epidemiology, clinical characteristics, and prognosis. J Am Coll Cardiol. 2004;43(3):317–327.
9. Owan, TE, Hodge, DO, Herges, RM, Jacobsen, SJ, Roger, VL, Redfield, MM. Trends in prevalence and outcome of heart failure with preserved ejection fraction. N Engl J Med. 2006;355(3):251–29.
10. Badesch, DB, Champion, HC, Sanchez, MA . Diagnosis and assessment of pulmonary arterial hypertension. J Am Coll Cardiol. 2009;54(1 Suppl):S55–S66.
11. McLaughlin, VV, Archer, SL, Badesch, DB . ACCF/AHA 2009 expert consensus document on pulmonary hypertension a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians; American Thoracic Society, Inc.; and the Pulmonary Hypertension Association. J Am Coll Cardiol. 2009;53(17):1573–1619.
12. Califf, RM, Adams, KF, McKenna, WJ . A randomized controlled trial of epoprostenol therapy for severe congestive heart failure: The Flolan International Randomized Survival Trial (FIRST). Am Heart J. 1997;134(1):44–54.
13. Teerlink, JR. Recent heart failure trials of neurohormonal modulation (OVERTURE and ENABLE): approaching the asymptote of efficacy? J Card Fail. 2002;8(3):124–127.
14. Barst, RJ, Gibbs, JS, Ghofrani, HA . Updated evidence-based treatment algorithm in pulmonary arterial hypertension. J Am Coll Cardiol. 2009;54(1 Suppl):S78–S84.
15. Pauker, SG, Kassirer, JP. The threshold approach to clinical decision making. N Engl J Med. 1980;302(20):1109–1117.
16. Soto, FJ. Pulmonary Venous Hypertension: A Diagnostic and Therapeutic Dilemma. Advances in Pulmonary Hypertension. 2007;6(4):168–174.
17. Soto, FJ, Kleczka, JF. Cardiopulmonary Hemodynamics in Pulmonary Hypertension: Pressure Tracings, Waveforms, and More. Advances in Pulmonary Hypertension. 2008;7(4):386–393.
18. Lam, CS, Borlaug, BA, Kane, GC, Enders, FT, Rodeheffer, RJ, Redfield, MM. Age-associated increases in pulmonary artery systolic pressure in the general population. Circulation. 2009;119(20):2663–2670.
19. Halpern, SD, Taichman, DB. Misclassification of pulmonary hypertension due to reliance on pulmonary capillary wedge pressure rather than left ventricular end-diastolic pressure. Chest. 2009;136(1):37–43.
20. Soto, FJ, Siegel, R, Marks, D . Performance of pulmonary capillary wedge pressure (PCWP) vs. left ventricular end diastolic pressure (LVEDP) in the diagnosis/classification of patients with suspect pulmonary arterial hypertension (PAH). Chest. 2005;128(4 Suppl):137S.

Comments

Related Resources

- Misclassification of Pulmonary Arterial Hypertension Due to Use of Pulmonary Capillary Wedge Pressure (PCWP) Rather Than Left-Ventricular End-Diastolic Pressure (LVEDP)
- Case of Catastrophic Antiphospholipid Syndrome: Roundtable Discusssion
- 2014 International Conference and Scientific Sessions - Abstracts
- Editor's Memo
- Consensus or Controversy: Do Recent Advances Shift the Debate for the Use of Echocardiography Versus Cardiac Magnetic Resonance Imaging of the Right Ventricle in Pulmonary Arterial Hypertension?

Denise Lewis, RN

PH Professional Network Member
Pulmonary Vascular Disease Program Coordinator
Inova Fairfax Hospital
Falls Church, Virginia

801 Roeder Road, Ste. 1000, Silver Spring, MD 20910
301-565-3004 | PHAOnlineUniv@PHAssociation.org