Cardiopulmonary Hemodynamics in Pulmonary Hypertension: Pressure Tracings, Waveforms, and More


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The most recent pulmonary hypertension (PH) classification divides PH into 5 categories based on pathophysiology and clinical characteristics.[1] As part of the diagnostic workup of PH, the current guidelines recommend performing a right heart catheterization (RHC) with a pulmonary artery catheter (PAC).[2] A RHC provides valuable diagnostic information, especially to discriminate between the first 2 categories: pulmonary arterial hypertension (PAH) and pulmonary venous hypertension (PVH).

A diagnosis of PAH is established in the presence of a resting mean pulmonary arterial pressure (PAP) greater than 25 mmHg and a left atrial or left ventricular (LV) filling pressure of no more than 15 mmHg.[1] The same mean PAP value cutoff and a left-sided filling pressure of greater than 15 mmHg fulfills criteria for a diagnosis of PVH. On the other hand, clear distinction between PAH and PVH can be problematic, and may be improved by additional maneuvers.

Several hemodynamic numbers routinely obtained during a RHC provide prognostic value in idiopathic PAH, including right atrial pressure (RAP), mean PAP, and cardiac index. For example, a baseline RAP greater than 20 mmHg carries a 6-month mortality rate of almost 100% without treatment.[3] Other interventions performed during the initial RHC provide additional hemodynamic information that helps guide treatment decisions and clarifies the etiology of the patient's PH.

Some of these interventions include an acute vasodilator challenge to determine the presence of vasoreactivity or an exercise or fluid challenge.[4,5] This article reviews characteristics of hemodynamic and waveform measurements. In addition, evaluation of data that includes cardiac outputs or indices, stroke volume or arteriovenous oxygen differences, mixed venous oxygen saturations, and others to define a profile of cardiopulmonary physiology is emphasized.

Characteristics of Hemodynamic Measurements and Specific Case Scenarios

Pulmonary capillary wedge pressure and A wave

A national registry sponsored by the National Institutes of Health proposed the hemodynamic definition for primary pulmonary hypertension as mean PAP of at least 25 mmHg and a left heart filling pressure of no more than 12 mmHg.[6]

The left heart filling pressure cutoff that determines PH as being precapillary in nature (eg, PAH) has been subsequently increased from 12 mmHg to no more than 15 mmHg.[1] While these criteria are fairly well established and applied, the methodology to measure those values is less standardized. For example, a potential for significant discrepancy between left-heart filling pressure numbers determined by left ventricular (LV) end-diastolic pressure versus pulmonary capillary wedge pressure has been found.[7]

In this study, approximately 40% of patients with PH whose pulmonary capillary wedge pressure was no more than15 mmHg were found to have LV end-diastolic pressure greater than 15 mmHg, therefore affecting their classification status. Likewise, the methodology for the measurement of right heart pressures can vary between practitioners. Some groups measure it at the end of exhalation; others measure them during a breath hold maneuver; and still others rely on the electronic pressures obtained by catheterization laboratory software.

The influence of intrathoracic pressure on intracardiac pressure measurement is least at end expiration. Therefore, intracardiac measurements should be recorded at end expiration when intrathoracic pressure is near zero.[8]

In order to obtain retrograde transmission of left atrial events through the pulmonary capillary bed (pulmonary capillary wedge pressure measurement), the PAC tip must be located in a lung segment where pulmonary venous pressure exceed the alveolar pressure (physiologic zone 3).[8,9] Most of the lung is in zone 3 in the supine patient and the catheter tip will be naturally directed to this zone.

Evidence of distinct A and V waves indicates the catheter tip is in zone 3. Conditions such as hypovolemia, advanced parenchymal lung disease, or positive pressure ventilation will make the alveolar pressure exceed the pulmonary venous pressure, therefore creating zones 1 or 2. In this case, pulmonary capillary wedge pressure becomes a measure of alveolar pressure rather than left atrial pressure. Distinct A and V waves are less likely to appear in zones 1 or 2.[8]

The pulmonary artery catheter education project (www.pacep.org)—an outstanding internet-based program endorsed by most critical care societies—has outlined guidelines for a more accurate and consistent measurement of right heart pressures. The program recommends obtaining all measurements at the end of

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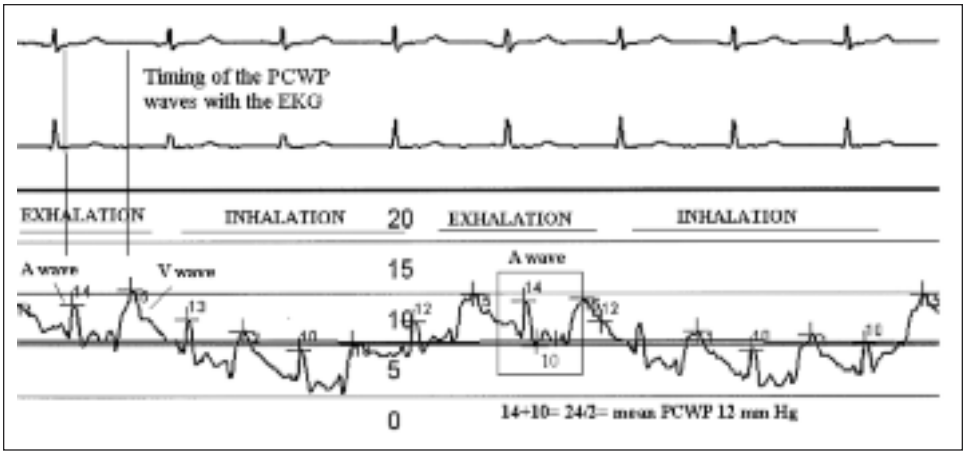

Figure 1. The proper timing to measure pulmonary capillary wedge pressure (PCWP) based on electrocardiogram (EKG) waveforms. This is followed by identification of the A wave during exhalation (right before the beginning of the pressure decline). Top and bottom values of the A wave are then determined and averaged to find the mean pulmonary capillary wedge pressure.

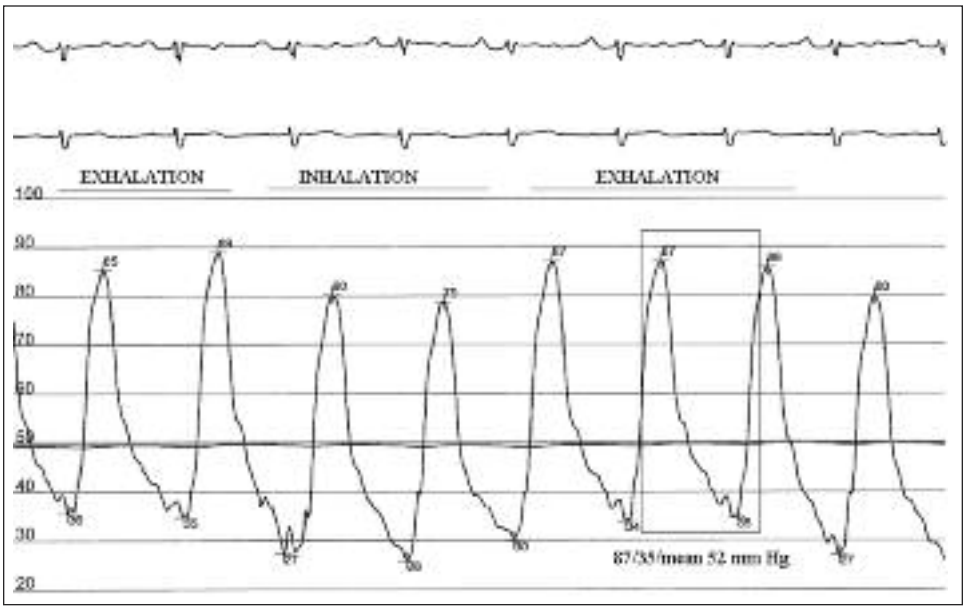

Figure 2. The timing to measure pulmonary arterial pressure (PAP) during exhalation (right before the beginning of the pressure decline). Once the PAP waveform is identified, systolic and diastolic values are obtained. Mean PAP is calculated using these 2 values.

exhalation. For adequate and accurate measurement of RAP and pulmonary capillary wedge pressure, identification of a distinct A wave is ideal.

The A wave occurs with atrial contraction. Since pulmonary capillary wedge pressure records the backward reflection of the left atrial contraction, the timing of the A wave is delayed. It is found near the end or after the QRS complex on the electrocardiogram (EKG) tracing. The V wave occurs when blood fills the atria as the tricuspid or mitral valve is closed. During pulmonary capillary wedge pressure measurement, the V wave is usually found well after the T wave of the EKG tracing.

Figure 1 shows the recommended methodology to estimate pulmonary capillary wedge pressure. Identification of A and V waves with respect to the EKG is crucial. The next step is to identify the inhalation and exhalation segments. Tracings on Figure 1 correspond to spontaneous breathing. Pulmonary capillary wedge pressure (and all other waveforms) should be measured during exhalation. The Pulmonary Artery Catheter Education Project (PACEP) program recommends finding the beginning of the pressure decline (right before going into inhalation), locating the A wave just prior to the decline, then measuring the top and bottom of the A wave values, and averaging these values.

Whenever needed, some maneuvers can be performed to confirm adequate position of the catheter tip and wedge pressure measurement, including a chest X ray, bedside fluoroscopic evaluation, or an oxygen (O2) saturation measurement from blood drawn from the distal port of the PAC while wedged (slow blood draw). In this case, if the catheter tip is properly wedged, the blood O2 saturation should be very close to a simultaneous blood sample of the patient's systemic arterial saturation. PAP should also be measured at the end of expiration (**Figure 2**).

V wave

A prominent V wave could potentially influence the measurement of pulmonary capillary wedge pressure and lead to a higher pressure estimate. Such a large V wave typically occurs in the presence of significant mitral valve regurgitation or impaired LV compliance (**Figure 3**). When the latter is present, administration of a systemic vasodilator trial (eg, nitroprusside) can lead to acute and dramatic resolution of the prominent V wave and significant reduction or normalization of the A wave.

On the other hand, in the presence of impaired relaxation of the LV, maneuvers that transiently increase venous return and/or pulmonary vasodilation (eg, exercise challenge, fluid bolus, pulmonary vasodilator trial) can lead to a rapid increase in LV filling pressure. This will manifest itself by an increase in the amplitude of the A wave and especially the V wave during pulmonary capillary wedge pressure measurement (**Figure 4**). Such a rapid V wave increase would suggest the presence of left ventricular diastolic dysfunction (LVDD) being unmasked by those maneuvers. The patient in **Figure 4** had risk factors for LVDD. However, his baseline pulmonary capillary wedge pressure was normal and a vasodilator trial with nitric oxide was performed, which led to a large pressure increase in the V wave. This trial unmasked impaired relaxation of the LV. His normal pulmonary capillary wedge pressure at rest is possibly explained by a lower intravascular volume after diuretic use.

Cardiac output and cardiac index

In clinical practice, determination of cardiac output and cardiac index is typically done by either the thermodilution method or Fick method (using the Fick principle). Normal values are: cardiac output, 4 to 8 L/minute; cardiac index, 2.6 to 4.2 L/minute/m^2. A low cardiac index has been shown to offer prognostic value for patients with PAH.[3] However, assessment of cardiac index alone would potentially neglect the impact of heart rate on cardiac output/cardiac index numbers. Therefore, the stroke volume index (cardiac index/heart rate) should also be calculated and assessed.

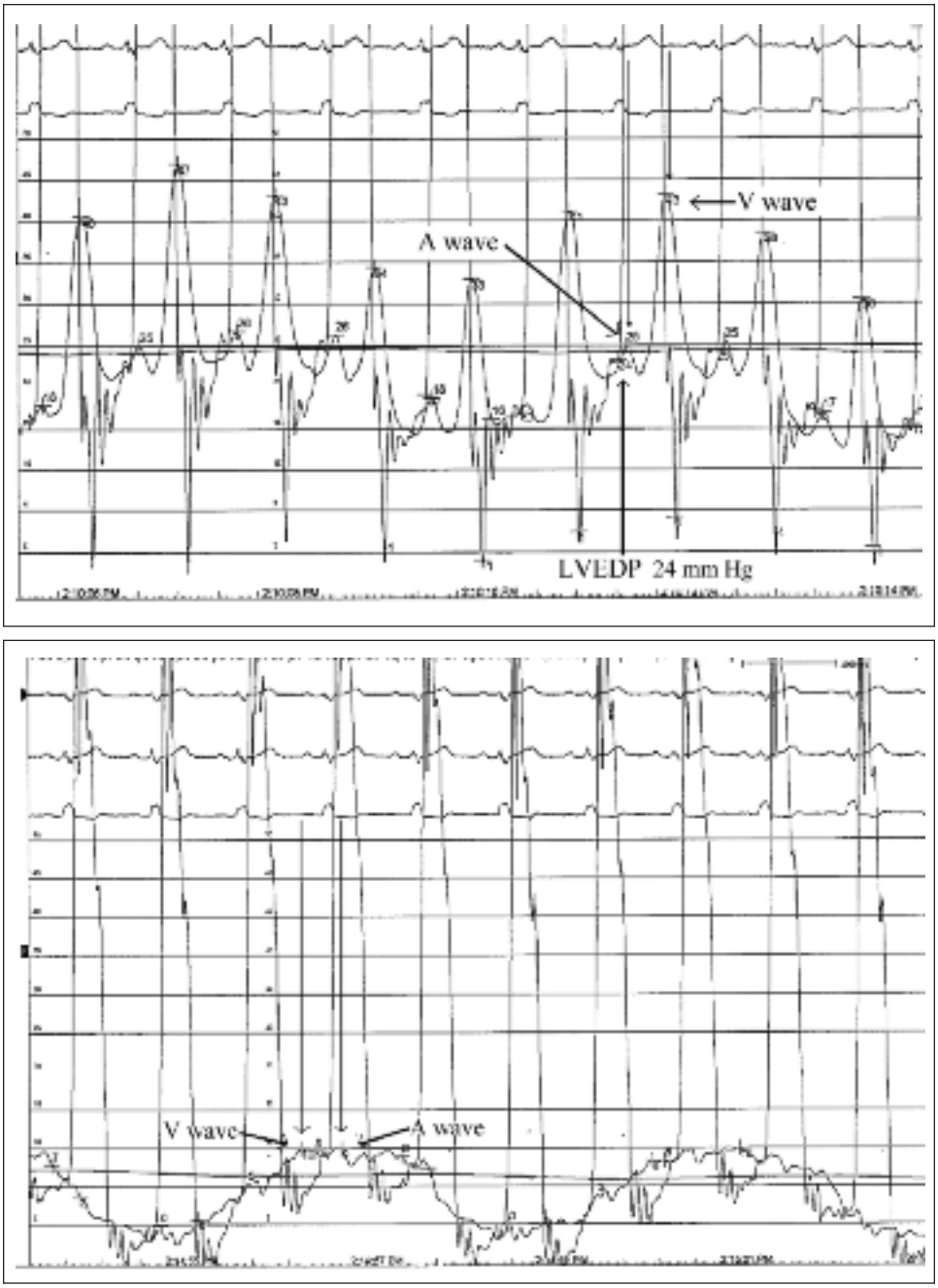

Figure 3. Normalization of pulmonary capillary wedge pressure (PCWP) with intravenous nitroprusside (NTP) in a patient with pulomnary venous hypertension secondary to left ventricular diastolic dysfunction. The top figure shows severe elevation of V wave pressure at baseline (43 mmHg). Left ventricular end-diastolic pressure (LVEPD) is high (24 mmHg), same as the PCWP (24 mmHg). HTP was administered (bottom figure) with acute reduction in systemic systolic blood pressure from 150 to 100 mmHg. Normalization of V wave pressure (10 mmHg), PCWP (8 mmHg) and pulmonary artery pressure (not shown) was seen. thin arrows indicate the timing of the A and V waves compared to electrocardiographic waveforms. No significant mitral valve regurgitation was demonstrated by echocardiogram of left ventriculogram.

Table. Higher Stroke Volume Index in Patient B Suggests Better Right Ventricular Myocardial Performance

Patient	Cardiac Index (L/m2)	Heart Rate	Stroke Volume Index (cc/m2)
A	2	100	20
B	2	50	40

In the **Table**, both patients have identical cardiac index numbers but stroke volume index for patient B is twice as high, which suggests better right ventricle (RV) myocardial performance. Assessment of stroke volume index values is also important when comparing hemodynamic numbers before and after PH therapy. Even in cases where the cardiac index does not change much, a significant increase in stroke volume index would still suggest improved RV function.

Thermodilution method

In this method, cold or room-temperature solution is injected in the right atrium through the proximal port of the PAC.[8] A thermistor near the catheter tip measures pulmonary arterial (PA) temperature continuously and detects the transient drop in PA temperature after the solution injection. A curve is generated by plotting the decline in PA temperature (°C) versus time (seconds). The area under the curve (AUC) is measured by the cardiac output computer and a cardiac output is provided (L/min). The AUC is inversely related to the cardiac output (eg, higher area, lower cardiac output) because the injected solution is diluted by body temperature blood flow. Some practical limitations to values obtained by this method in patients with PH include severe tricuspid regurgitation and presence of a congenital heart defect (eg, atrial septal defect). In general, the injections are performed in triplicate and averaged to yield the cardiac output measurements.

Fick method

The Fick method measures pulmonary blood flow using principles described by Adolph Fick in 1870. In the absence of a significant intracardiac shunt, the pulmonary and systemic blood flows are equal. The Fick equation is:

Cardiac output (L/min) = oxygen consumption (mL of O_2/min)/arteriovenous oxygen difference (mL of O_2/L of blood).

This formula shows that arteriovenous oxygen difference is inversely proportional to cardiac output. For more accurate results, the Fick method requires documentation of both O_2 consumption and arteriovenous oxygen difference.[8] Ideal determination of O_2 consumption can be done by collecting the patient's exhaled air over several minutes, something that is not practical in the catheterization laboratory or intensive care unit. Metabolic carts at bedside can be used to estimate O_2 consumption using indirect calorimetry. This approach requires strict attention to detail. Finally, O_2 consumption can be assumed as basal (125 mL of O_2/min/m^2). While this

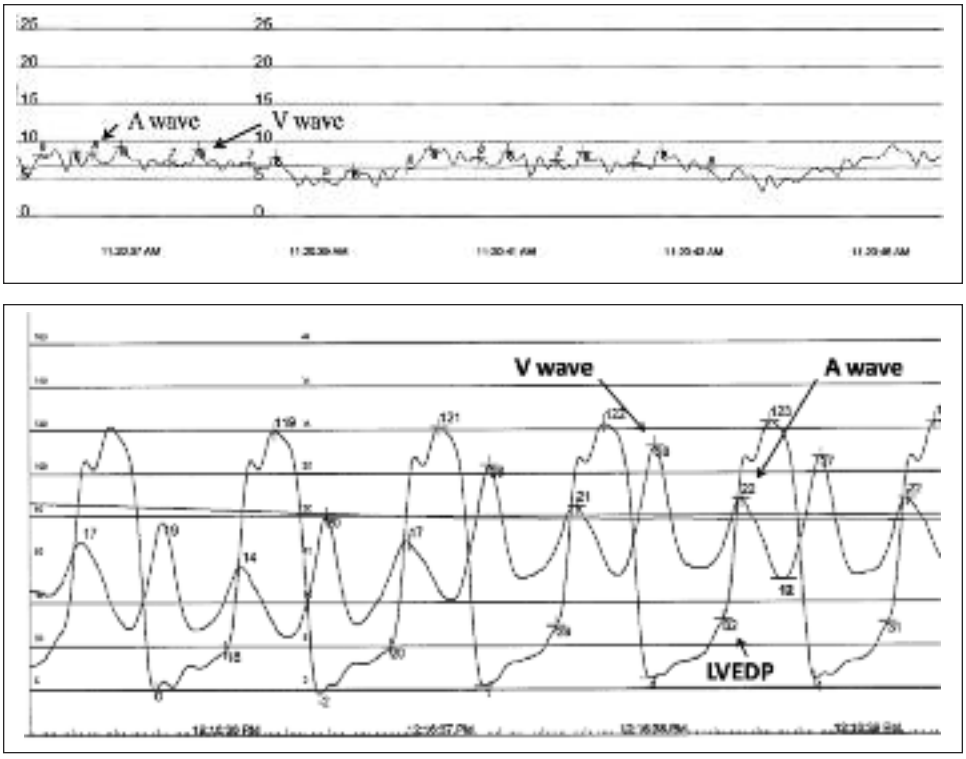

Figure 4. Increase in pulmonary capillary wedge presure (PCWP) and V wave pressure after a vasodilator trial with nitric oxide (NO). The top figure shows normal resting PCWP (7 mmHg) and normal V wave pressure (9 mmHg) in a patient with mild pulmonary hypertension (PH). Resting mean pulmonary artery pressure was 30 mmHg (not shown). After administration of NO (bottom figure) there was a significant increase in the V wave pressure (28 mmHg) and PCWP (18 mmHg) The left ventricular end-diastolic pressure (LVEDP) increased from 15 mmHg at baseline (not shown) to 32 mmHg. these findings point to left ventricular diastolic dysfunction as the likely cause for PH in this case. Of note, a significant difference is seen between LVEDP and pulmonary capillary wedge pressure measurements.

is the more practical approach to calculate cardiac output by the Fick method, one must keep in mind that when assuming OO_2 consumption as basal, one may underestimate this value in critically ill patients. Calculation of arteriovenous oxygen difference requires simultaneous determination of arterial and mixed venous O_2. Using an estimated arterial oxygen saturation obtained by pulse oximetry in this formula could lead to potential inaccurate calculations and direct measurement from arterial blood should be considered. See additional discussion on arteriovenous oxygen difference below.

Thermodilution method versus Fick method

The literature indicates significant discrepancy between these 2 methods, especially on both low and high output states. Recent studies have specifically compared these 2 methods in patients with PH and did not find major discrepancies.[10,11] Some caveats to keep in mind when using either method, include:

- Thermodilution: At least 3 measurements should be obtained and they should be within 10% of each other to improve accuracy. Ideally, each injection should be done at the end of expiration for improved consistency.[12] However, this may be difficult to coordinate in patients who are in respiratory distress.
- Fick: Since heart rate is part of the cardiac output calculation by the Fick method, same heart rate should be entered for both the thermodilution method and the Fick method for improved correlation. When comparing changes in cardiac output/cardiac index by the Fick method (eg, before and after PH therapy), one must remember that significant changes in hemoglobin levels could account for differences in results.

Even though cardiac index offers more relevant information than cardiac output, one must keep in mind that large changes in body weight (eg, secondary to fluid retention) could affect the body surface area calculation and therefore affect cardiac index values over time (cardiac index = cardiac output / body surface area). This applies to cardiac index estimates by both the thermodilution method and the Fick method.

Additional Hemodynamic Information: Vasodilator and Exercise Challenges

Based on the current referral pattern seen at most PH centers—large number of patients with underlying left heart disease—it can be quite difficult to discriminate between idiopathic PAH and PH secondary to left heart disease. Close observation of additional hemodynamic numbers and tracings can provide the necessary clues to make this distinction more accurate.

Vasodilator challenge

This intervention is typically performed to assess the presence of a reversibility component, which suggests a potential benefit from calcium-channel blockers.[4] The indication for this challenge mainly applies to patients in whom idiopathic PAH is suspected. Pulmonary vasodilators such as nitric oxide or intravenous prostacyclins are the agents more commonly used. When performing this trial, the clinician must keep in mind that these agents could lead to an acute increase in right-to-left blood flow and potentially overload a poorly compliant LV. Their use in patients with elevation of the LV filling pressures can lead to dangerous situations, including acute pulmonary edema.[13,14]

Close inspection should be given to LV end-diastolic pressure and pulmonary capillary wedge pressure values and waveforms during a vasodilator trial. The patient in **Figure 4** had risk factors for left ventricle diastolic dysfunction (LVDD). However, the resting pulmonary capillary wedge pressure was normal, which was suggestive of precapillary PH or PAH and a vasodilator trial with nitric oxide was performed. In this case, nitric oxide led to a significant increase in both LV end-diastolic pressure and pulmonary capillary wedge pressure which unmasked the presence of impaired relaxation of the LV. Even though the baseline pulmonary capillary wedge pressure was within normal limits, the wedge pressure changes seen with nitric oxide suggested PVH to be the most likely diagnosis. A dramatic V wave pressure increase was also consistent with this diagnosis.

Exercise challenge

This intervention is used by many PH centers during the hemodynamic evaluation of patients with suspected PH.[15,16] Unfortunately, the protocol for exercise challenge is not standardized. Nevertheless, detailed assessment of LV end-diastolic pressure and pulmonary capillary wedge pressure values and waveforms during an exercise challenge can provide very useful information when trying to determine the presence of PAH versus PVH.

In addition to a greater than 25 mmHg cutoff to establish the presence of PH at rest, the guidelines also propose a PH diagno-

sis if mean PAP is greater than 30 mmHg during an exercise challenge.[17] A limitation of the exercise-induced diagnostic criterion is that it does not address what are the expected simultaneous changes in LV filling pressure (eg, pulmonary capillary wedge pressure) to differentiate between PAH or PVH.

Figure 5 reveals a significant increase in mean PAP consistent with the exercise-related PH definition. However, LV end-diastolic pressure increased in concordance with the increase in pulmonary artery diastolic pressure (no pulmonary artery diastolic pressure–LV end-diastolic pressure gradient appreciated here), which suggests that the increase in mean PAP to greater than 30 mmHg is secondary to a retrograde increase of LV filling pressure. This is consistent with impaired LV relaxation probably unmasked by increased venous return during an exercise challenge. In this case scenario, initiation of pulmonary vasodilators would likely be contraindicated since they could lead to an increase in the patient's LV filling pressure, putting the patient at risk for pulmonary congestion and worsening dyspnea. This potential risk is reinforced by the fact that the use of PAH therapies in patients with LV systolic dysfunction has led to a higher frequency of congestive heart failure.[18-21]

Figure 6 reveals a mean PAP greater than 30 mm Hg after an exercise challenge but the LV filling pressure remains relatively normal. An increased gradient is appreciated between her pulmonary artery diastolic pressure and left ventricular end-diastolic pressure at peak exercise suggesting the presence of true PAH. This scenario is perhaps more suggestive of early PAH changes unmasked with an exercise challenge, especially in this patient with underlying systemic sclerosis. Unfortunately, the literature is not clear on how to approach or treat exercise-induced PAH. Many PH experts consider initiation of treatment with PAH-approved therapies in this case.

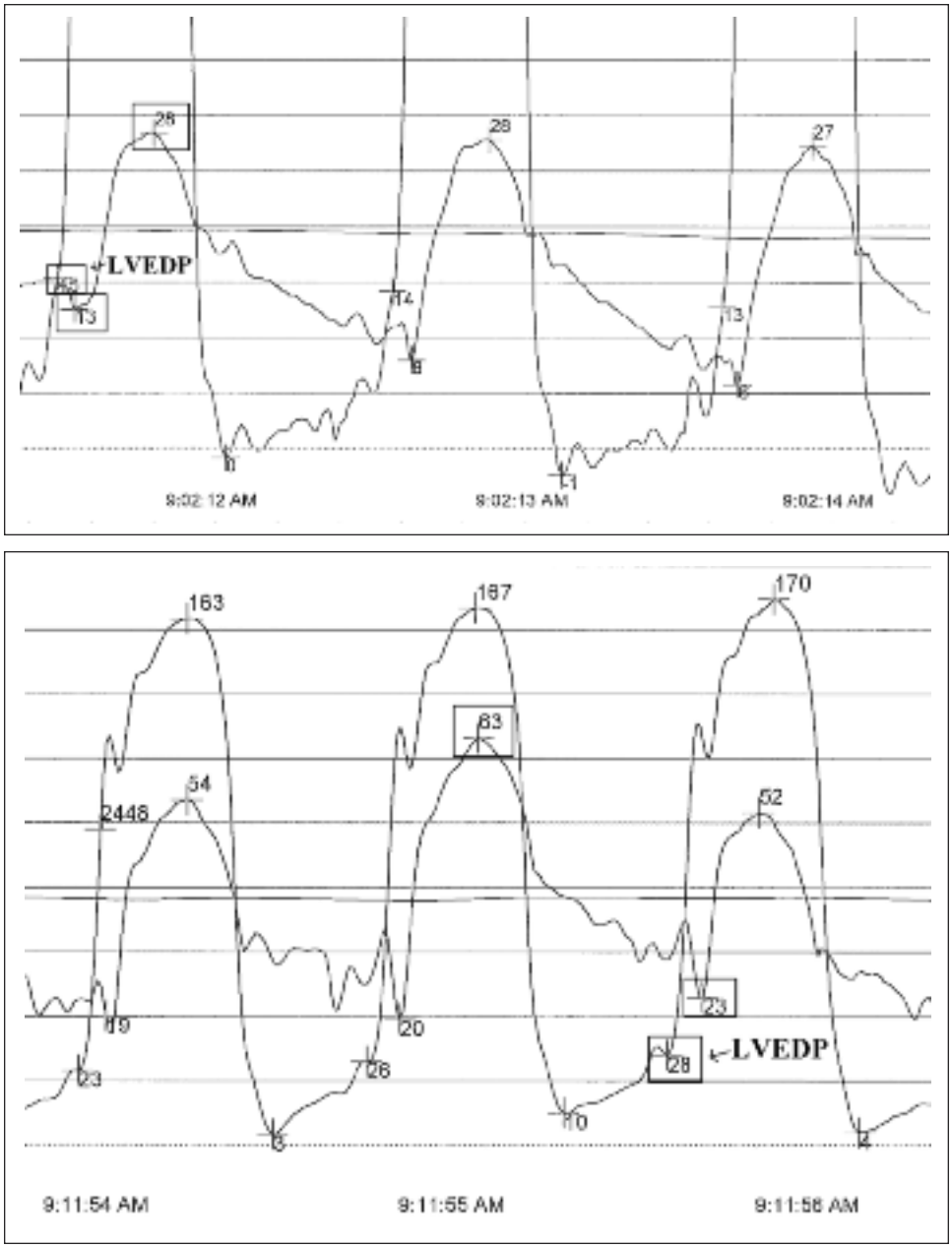

Figure 5. Increase in pulmonary artery pressures (PAP) during an exercise challenge in a patient with suspected pulmonary hypertension (PH) and progressive dyspnea. PAP was 28/13/mean 18 mmHg at baseline (top figure) and increased to 63/23/mean 36 mmHg after an exercise challenge (bottom figure), consistent with the definition of exercise-related PH. However, a very significant increase in left ventricular end-diastolic pressure (LVEDP) from 15 mmHg at baseline to 28 mmHg at peak exercise was also seen. These findings point to left ventricular diastolic dysfunction as the likely cause for the increase in PAP.

Fluid challenge

Similar findings to the ones described above (vasodilator and exercise challenge) can be seen when a fluid challenge is administered. A typical amount of fluid infused varies between 500 to 1000 cc of normal saline. Documenting a significant increase in LV filling pressure (LV end-diastolic pressure or pulmonary capillary wedge pressure) or significant changes in pulmonary capillary wedge pressure waveforms after such challenge would increase the likelihood that a diagnosis of PVH is present (**Figure 7**).

Systemic vasodilator challenge

Before newer and safer vasodilator agents became available (eg, nitric oxide), systemic vasodilator agents such as nitroprusside were used to assess for pulmonary vasoreactivity of patients with PAH.[22] This agent has also been used in patients with significant LV systolic dysfunction before heart transplant consideration.[23] However, its role in assessing vasoreactivity of patients with PVH secondary to LVDD is less clear.

At our institution, this agent is frequently used to assess hemodynamic response in cases where PVH is suspected but where the distinction between PAH and PVH is not completely clear. Its use has not been validated in prospective studies and the available experience is based on case series.[24] Having said that, in select cases it can provide a dramatic reduction or normalization of pressures that confirms the presence of LVDD as the cause of the patient's PH. This can be quite important, especially in cases where the patient has been labeled as "PH out of proportion to left

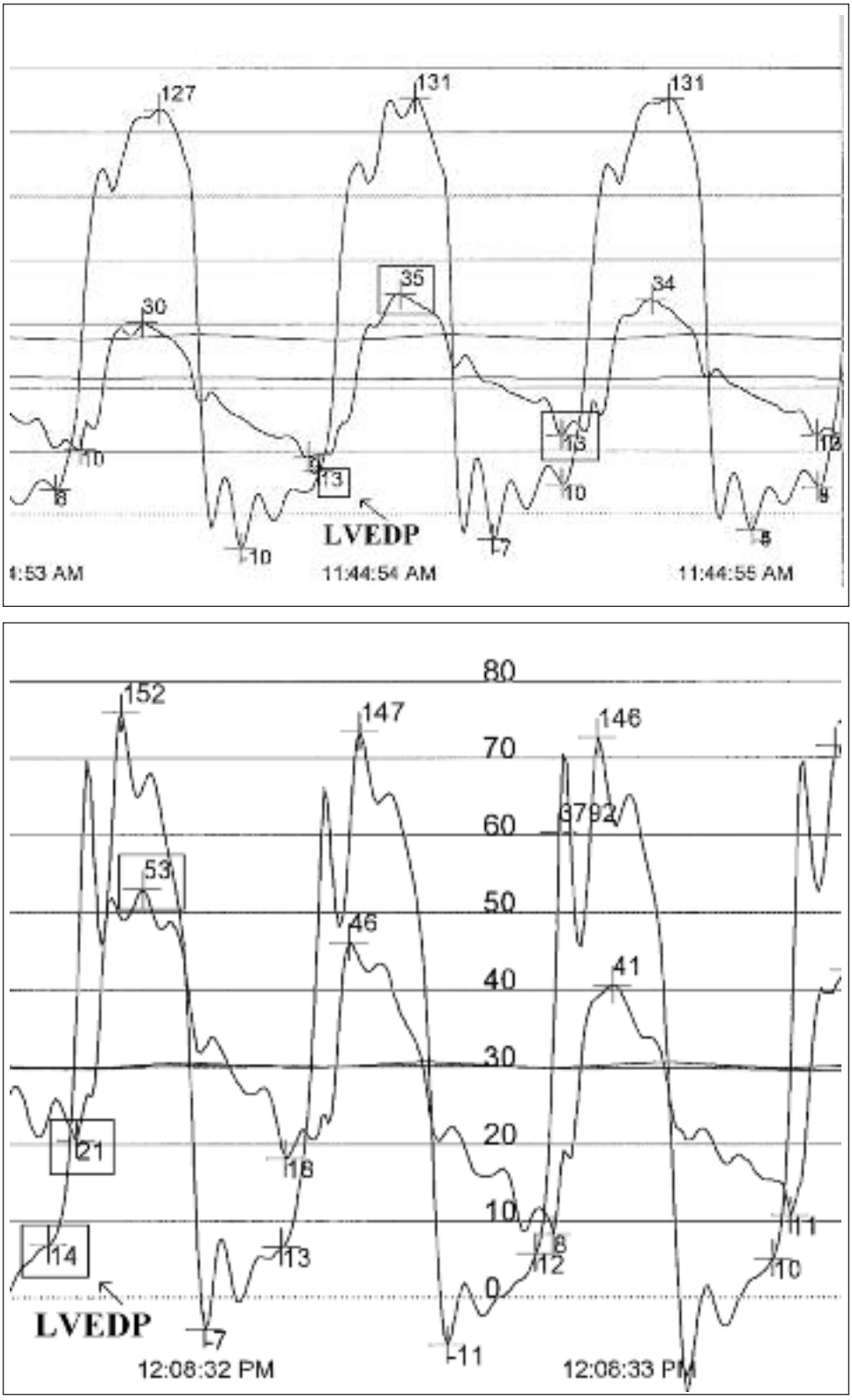

Figure 6. Increase in pulmonary artery pressures (PAP) in a patient with systemic sclerosis and suspected pulmonary hypertension (PH). PAP was 35/13/mean 20 mmHg at baseline (top figure) and increased to 53/21/mean 32 mmHg after an exercise challenge (bottom figure), consistent with the definition of exercise-related PH. Left ventricular end-diastolic pressure (LVEDP) was 13 mmHg at baseline and 14 mmHg at peak exercise. These findings point to early pulmonary arterial hypertension as the likely cause for the increase in PAP.

heart disease." Given the potential for abrupt systemic vasodilation, it is our practice to perform nitroprusside vasodilator trials while simultaneously monitoring LV end-diastolic pres- sure and systemic arterial pressures. We do not perform nitroprusside trials in clear-cut PAH cases or in cases where there is evidence of RV dysfunction. Those patients may have an impaired hemodynamic response and may not be able to adequately increase their cardiac output in the event of systemic hypotension. For such cases, other safer alternatives such as an exercise challenge or a fluid bolus could be considered in order to evaluate the LV pressure response to extra circulating volume or an increase in blood flow. Unfortunately, none of those interventions has been validated.

Documenting normalization or near normalization of pulmonary pressures while maintaining or increasing cardiac output provides additional evidence to suspect PVH as the main cause for the patient's PH. Such distinction is obviously crucial given the significant difference in the treatment approach for both groups. **Figure 3** depicts how the administration of nitroprusside led to normalization of pulmonary pressures. Mean pulmonary capillary wedge pressure also normalized and the V wave had a dramatic decrease in amplitude, which suggests improvement in LV relaxation.

Additional Catheterization Data

Mixed venous oxygen saturation

Mixed venous oxygen saturation determines the oxygen saturation of blood returning to the lungs before reaching the alveolo-capillary units. It is best measured from the distal lumen of a catheter in the pulmonary artery. A normal value is around 75%. A value lower than this (eg, 60%) probably represents a low cardiac output that has led to increased extraction of oxygen by the peripheral tissues. Improvement in mixed venous oxygen saturation numbers (before and after treatment) likely reflects improvement in cardiac output and cardiac index. Given the value of cardiac index as a predictor of survival in PAH, an increase in mixed venous oxygen saturation reinforces the notion that the therapeutic response is adequate.

However, before one relies heavily on this measurement's information, one needs to keep in mind that mixed venous oxygen saturation measures dissolved oxygen levels and it can be influenced by several factors. One of them is oxygen administration, which will typically improve oxygen saturation of arterial blood and this in turn will lead to some additional increase of mixed venous oxygen saturation. Therefore, a rapid increase in mixed venous oxygen saturation could simply be related to a higher fraction of inspired oxygen (FiO_2) without necessarily reflecting an improvement in cardiac output/ cardiac index.

Changes in mixed venous oxygen saturation values can have a more significant impact on treatment decisions when both measurements (before and after an intervention) are obtained on the same FI O_2. Taking into account both arterial and mixed venous O2 saturation and content (arteriovenous oxygen difference) can provide a more accurate picture of the cardiac output status (see below).

Arteriovenous oxygen difference

This is the amount of oxygen (mL) extracted by tissues from each liter of blood circulated. The value is obtained from subtracting venous oxygen content (CV O_2) from the arterial oxygen content (CA O_2) and is mainly influenced by cardiac output/cardiac index. For example, a low cardiac output should lead to lower mixed venous oxygen saturation values (thereby decreasing the total venous oxygen content) and a higher difference between CA O_2 and CV O_2 will be expected. Therefore, a high arteriovenous oxygen (AV O_2) difference represents a lower cardiac output. The AV O_2 difference calculation assumes that red cells with 100% oxygen carry 1.36 mL/O_2/gram/Hgb.[25]

Arteriovenous oxygen difference (simplified equation)= hemoglobin x 1.36 x (arterial oxygen saturation – mixed venous oxygen saturation). Normal value: 3-5 mL/O_2 x 100 mL of blood.

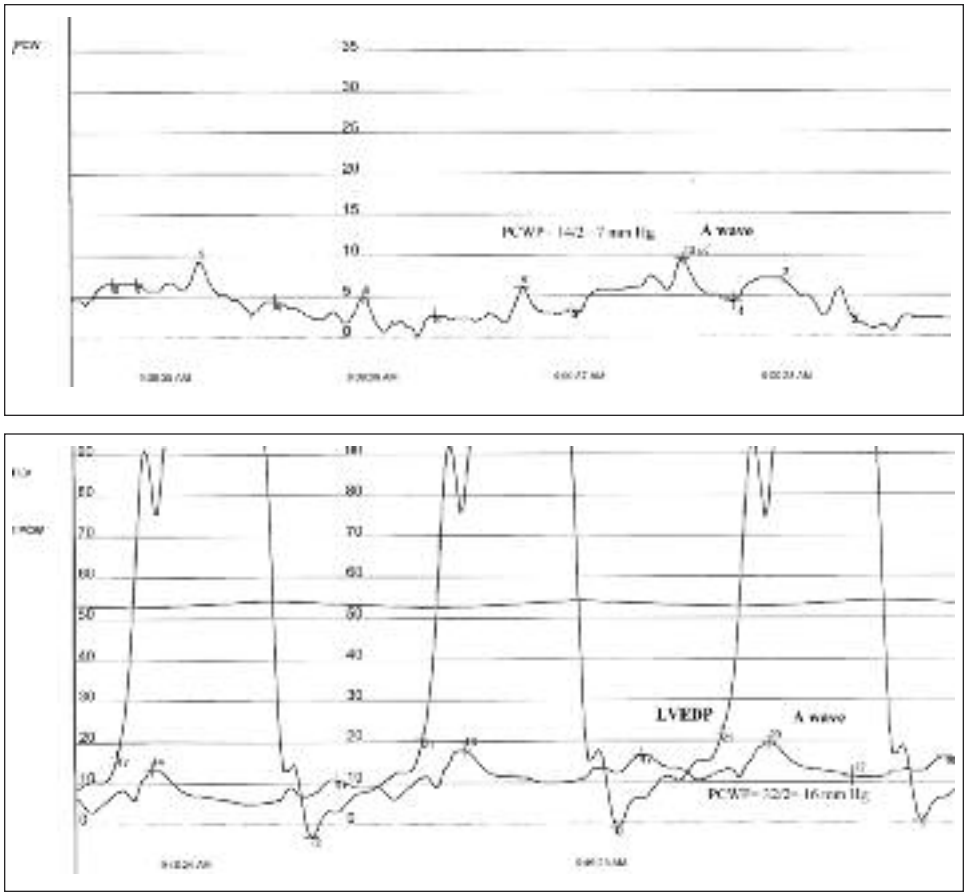

Figure 7. Increase in pulmonary capillary wedge pressure (PCWP) after a fluid bolus in a patient with pulmonary hypertension (PH) and normal PCWP. The PCWP was 7 mmHg at baseline (top figure) and increased to 16 mmHg after a fluid bolus with 500 cc of normal saline (bottom figure). Left ventricular end-diastolic pressure (LVEDP) increased from 7 mmHg at baseline (not shown) to 21 mmHg. An exercise challenge led to a similar increase in both PCWP and pulmonary artery pressures. These findings point to left ventricular diastolic dysfunction as the likely cause for PH in this case.

In the presence of limitations to determine accurately cardiac output/cardiac index in patients with PH—significant tricuspid regurgitation affects the thermodilution method or inaccurate estimate of O_2 consumption affects the Fick method—assessment of AV O_2 difference values provides an additional and reliable estimate of cardiac output/cardiac index. If AV O_2 difference is compared before and after PAH treatment, this comparison will be more meaningful when pre and post treatment hemoglobin levels, arterial oxygen saturation and FI O_2 used in the calculations are similar.

Because normal arterial oxygen saturation is around 93% to 98% and normal venous oxygen saturation is around 75%, a normal resting AV O_2 difference is around 3 to 5 mL of O_2/100 mL of blood. A number higher than this suggests a low cardiac output/cardiac index status and vice versa. Unfortunately, these values mainly apply to hemoglobin values in the normal range. Significant anemia or erythrocytosis will affect the normal range.

Summary

Right heart catheterization is routinely performed as part of the diagnostic workup of patients with suspected PH. Traditionally, clinicians have mainly focused on the more common hemodynamic values that assess right-sided pressures and pulmonary capillary wedge pressure. With the steady increase in referrals of patients with underlying left heart disease to PH centers for evaluation, PH experts are faced with significant difficulties in trying to discriminate between precapillary and postcapillary PH cases.

Careful observation of the hemodynamic response and change in waveforms at rest and after challenges such as exercise, pulmonary vasodilators, fluid administration, and others, may provide invaluable information to generate an integrated picture of cardiopulmonary function. Physicians who treat PH are therefore encouraged to look at the actual catheterization tracings to confirm the reported numbers, analyze the waveforms, and supplement these data with additional information such as mixed venous oxygen saturations or arteriovenous oxygen differences when appropriate.

Given the complexity of the cases evaluated at most PH programs, making an accurate diagnosis of PH and correctly classifying it is clearly becoming an art that requires detective skills. Validation of the diagnostic and therapeutic value of interventions or challenges frequently performed during initial PH catheterizations is crucial in order to achieve a more consistent and standardized approach to these cases.

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