Pulmonary Artery Pulse Pressure and Wave Reflection in Chronic Pulmonary Thromboembolism and Primary Pulmonary Hypertension

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OBJECTIVES

The purpose of this time-domain study was to compare pulmonary artery (PA) pulse pressure and wave reflection in chronic pulmonary thromboembolism (CPTE) and primary pulmonary hypertension (PPH).

BACKGROUND

Pulmonary artery pressure waveform analysis provides a simple and accurate estimation of right ventricular afterload in the time-domain. Chronic pulmonary thromboembolism and PPH are both responsible for severe pulmonary hypertension. Chronic pulmonary thromboembolism and PPH predominantly involve proximal and distal arteries, respectively, and may lead to differences in PA pressure waveform.

METHODS

High-fidelity PA pressure was recorded in 14 patients (7 men/7 women, 46 ± 14 years) with CPTE (n = 7) and PPH (n = 7). We measured thermodilution cardiac output, mean PA pressure (MPAP), PA pulse pressure (PAPP = systolic – diastolic PA) and normalized PAPP (nPAPP = PAPP/MPAP). Wave reflection was quantified by measuring Ti, that is, the time between pressure upstroke and the systolic inflection point (Pi), ΔP, that is, the systolic PA minus Pi difference, and the augmentation index (ΔP/PAPP).

RESULTS

At baseline, CPTE and PPH had similar cardiac index (2.4 ± 0.4 vs. 2.5 ± 0.5 l/min/m²), mean PA (59 ± 9 vs. 59 ± 10 mm Hg), MPAP (57 ± 13 vs. 53 ± 13 mm Hg) and nPAPP (0.97 ± 0.16 vs. 0.89 ± 0.17). Chronic pulmonary thromboembolism had shorter Ti (90 ± 17 vs. 126 ± 16 ms, p < 0.01) and higher ΔP/PAPP (0.26 ± 0.01 vs. 0.09 ± 0.07, p < 0.01).

CONCLUSIONS

Our study indicated that: 1) CPTE and PPH with severe pulmonary hypertension had similar PA pulse pressure, and 2) wave reflection is elevated in both groups, and CPTE had increased and anticipated wave reflection as compared with PPH, thus suggesting differences in the pulsatile component of right ventricular afterload. (J Am Coll Cardiol 2001;37:1085–92)

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Primary pulmonary hypertension (PPH) and chronic pulmonary thromboembolism (CPTE) are both responsible for severe chronic pulmonary hypertension. In these two diseases, the mechanism of death is mainly right ventricular failure (1–3). Therefore, it is of major importance to improve our understanding of right ventricular afterload in PPH and CPTE. Chronic pulmonary hypertension results from an increase in pulmonary vascular resistance, which is a simple measure of the opposition to the mean component of flow. However, given the low resistance/high compliance nature of pulmonary circulation, the pulsatile component of hydraulic load is also important to consider (4,5). Several studies have documented the relationship between pulsatile pressure and flow (impedance) in the frequency domain.

They have shown that the pulsatile load is also increased in chronic pulmonary hypertension, as attested to by the increased characteristic impedance and enhanced wave reflection (6–10). This has generally been attributed to decreased pulmonary artery (PA) compliance and complex changes in reflection sites. In pulmonary hypertension, Milnor et al. (6) have pointed out that decreased PA compliance is of similar importance to increased resistance in the elevation of right ventricular afterload. This abnormal pulsatile load may have detrimental effects on ventricular-vascular coupling by increasing the pulsatile part of ventricular power and, thus, unfavorably loading the still-ejecting right ventricle (5–10).

Impedance is not easy to assess in clinical practice, and, therefore, the pulsatile component of right ventricular afterload is generally ignored in patients with chronic pulmonary hypertension. Taking into account both these technical limitations and certain limitations of Fourier analysis (11), it has recently been suggested that time-domain analysis of pulse pressure and pressure waveform could provide valuable information on pulsatile arterial load (11–13). Pulse pres-
Pulse pressure (i.e., systolic minus diastolic pressure) indicates the amplitude of pulsatile stress. Pulse pressure is mainly determined by both the characteristics of ventricular ejection and arterial compliance, such that the lower the compliance, the higher the pulse pressure in both aorta (14–17) and PA (5,18,19). Moreover, pressure waveform analysis performed in the time-domain makes it possible to calculate the timing and extent of wave reflection in systemic (20,21) and pulmonary (12,13) circulation.

The first aim of this study was to compare PA pulse pressure (PAPP) in PPH and CPTE. The rationale was that PPH and CPTE lead to severe pulmonary hypertension via different mechanisms. In PPH, pulmonary obstructive vasculopathy involves distal, medium-to-small sized muscular (resistive) arteries. By contrast, in CPTE, the thrombi follow an aberrant path of organization and recanalization, leaving endothelialized residua that narrow and stiffen major (main, lobar or segmental) pulmonary arteries, that is, elastic arteries (1–3). These pathophysiological differences could lead to differences in the pulsatile component of hydraulic load (22–26). Cardiac ejection into a low compliance system generates a higher pulse pressure than in a normally compliant system (27–29). Furthermore, constriction of the main PA is associated with a higher PAPP than in the case of distal obstruction as determined in a study of distal artery microembolization in dogs with comparable PA hypertension severity (25). Thus, we tested the hypothesis that the PAPP would be higher in CPTE than it would be in PPH.

The second aim of our study was to compare wave reflection in PPH and CPTE. The timing of the return of the reflection wave to the entrance of the pulmonary bed is a fraction of the distance to the reflected site(s) and pulse wave velocity (30,31). The composite reflection wave has been shown to arrive during the diastolic period in healthy subjects and during the midportion of right ventricular ejection time in PPH (6,8,10,12,13). Thus, we hypothesized that abnormal mechanical properties and geometry of proximal arteries would lead to early, enhanced wave reflection in CPTE versus PPH. The distending mean PA pressure (MPAP) being a contributory factor in both PA compliance and pulse wave velocity (5,31), CPTE and PPH were, therefore, matched for MPAP.

**METHODS**

**Patients.** The study was approved by the ethics board of Paris-Sud University, and informed consent was obtained for all patients. From December 1996 to July 1997, 14 consecutive patients referred to our catheterization laboratory for severe pulmonary hypertension were studied. The study population comprised patients with PPH (n = 7) or CPTE (n = 7). There were seven men and seven women (mean age ± SD = 46 ± 14 years). Pulmonary hypertension was defined by an MPAP in excess of 25 mm Hg at rest in room air and with a mean pulmonary occlusion pressure ≤12 mm Hg at right-sided catheterization. Primary pulmonary hypertension was diagnosed according to the criteria of the National Institutes of Health Patient Registry for the Catheterization of Primary Pulmonary Hypertension (32). Secondary causes of pulmonary hypertension were excluded after thorough clinical and laboratory evaluations, chest radiography, pulmonary function testing, echocardiography and perfusion lung scan or pulmonary angiography (32). Chronic pulmonary thromboembolism diagnosis was based on the existence of multiple perfusion defects on the perfusion lung scan and a typical angiographic pattern, that is, pouchings, webs, stenosis, parietal irregularities, abrupt narrowing and vascular amputations (33). All patients were given anticoagulants. No patient was undergoing vasodilator treatment at the time of the study. Patients with right-to-left interatrial shunting or with significant tricuspid insufficiency on echocardiography were excluded from the study.

**Catheterization technique.** Hemodynamic evaluation was carried out on supine patients breathing room air, according to our routine protocol (34,35). Right heart catheterization was performed using the Seldinger technique with an 8F sheath via the jugular or basilic vein. The right heart catheter was a 7.5F, 2 lumen, thermodilution pressure-measuring tipped catheter with a high-fidelity transducer (Cordis/Sentron, Roden, the Netherlands) (36,37). The Sentron system has a frequency bandwidth of 0 to 180 Hz, and good agreement between the system and Millar microtip catheter has been previously demonstrated in both the time-domain and the frequency-domain (36). Pressure data were computed on a Toshiba 3200 SX, and we used a sampling frequency of 1,000 Hz, without filtering of the analog signal, as previously recommended (36). Filtering at half the digitization rate is known to avoid aliasing. However, it is unlikely that it would have any impact on our measurements (36). The catheter was placed into either the right or left PA. Cardiac output was measured in triplicate by the thermodilution technique (34). Pulmonary artery blood samples were taken for measurements of mixed venous oxygen saturation. Electrocardiogram was monitored continuously. Respiratory cycles were determined with a respiratory inductive photoplethysmograph (Respitrace, Ambulatory Monitoring, Arsley, New Jersey).

**High-fidelity PA pressures (Fig. 1).** Mean pulmonary artery pressure was defined as the area under the pressure
curve divided by the pulse interval. Systolic PA pressure (SPAP) was determined automatically by the computer analysis. The onset of pressure pulse and the corresponding diastolic PA pressure (DPAP) was identified in all the patients as the time when pressure derivative (dP/dt) increased steeply. The intra- and interobserver variability for DPAP measurements was 6 ± 4% and 1 ± 1%, respectively. We calculated PAPP (PAPP = SAPP – DAPP). Mean pressure and pulse pressure reflect, respectively, the steady and pulsed components of the PA pressure (4,38,39). Given that the higher the mean pressure, the lower the arterial compliance and, thus, the higher the fluctuations around the mean, we calculated the PAPP/MPAP ratio. Pulmonary artery occlusion pressure was determined according to standard procedures. However, given the severity of pulmonary hypertension, it was possible to record PA occlusion pressure in 4/7 CPTE (12 ± 4 mm Hg) and 5/7 PPH (11 ± 2 mm Hg, p = 0.6) only. Total pulmonary resistance (TPR) was calculated as TPR = MPAP/cardiac output. The TPRi was calculated as the TPR/body surface area (b.s.a.) ratio.

Wave reflection: background. Pulmonary artery pulse wave velocity is around 2 m/s at normal distending pressure, such a value being about half that usually reported in the aorta (31). In both the aorta and the PA of healthy humans, the composite reflected pressure wave arrives during the early diastolic period because the shorter path length in the pulmonary circulation compensates for the relatively lower pulse wave velocity (20,27,31). An increase in pulse wave velocity has been noted when the distending, MPAP increases (6,40). In patients with pulmonary hypertension, this was considered partially due to a direct effect on the large arteries (6) or to a passive phenomenon resulting from greater stretch of the vessel with transfer of tension from the elastic to collagenous component of the wall (31).

Timing and extent of wave reflection (Fig. 1). The timing and extent of wave reflection were quantified by using pressure waveform analysis in the time-domain, as previously described (12,13,20). Indeed, Murgio et al. (20) have shown: 1) that the magnitude of the secondary rise in the aortic pressure waveform appears to be directly related to the magnitude of the oscillations in the impedance spectra and 2) that the transit time derived from the pressure data is inversely related to both the frequency at which the first minimum of the impedance modulus occurs and to the transit time derived from impedance data. This approach has recently been extended to pulmonary circulation (12,13). The human pulmonary pressure systolic waveform may exhibit an inflection point (Pi) that results from peak flow input into the vasculature before the effects of wave reflection. Pi indicates the end of the forward (or incident) pressure wave. The relative increase in pressure amplitude above the inflection point (ΔP = SPAP – Pi) is an estimate of the magnitude of the reflected pressure wave. The ratio of ΔP to PAPP defines a so-called augmentation index (ΔP/PAPP) (12,13). In normal subjects, there is very little wave reflection. Otherwise, the ΔP/PAPP ratio can be used to precisely quantify the extent of wave reflection in PA (12,13).

In this study, we calculated Pi, ΔP, ΔP/PAPP and the so-called transit time (Ti), that is, the time between pressure upstroke and Pi. In the majority of patients (11/14), the inflection in pressure was smooth, such that the first derivative (dP/dt) did not cross zero. As a result, the inflection point was defined as the time at which the dP/dt reached its first minimum. In the remaining 3/14 patients (two with CPTE, one with PPH), the pressure inflection was sharp, such that the dP/dt value at the time of inflection crossed zero. In these patients, the inflection point was defined as the time when the dP/dt crosses zero for the second time. Given that Ti is influenced by the systolic time, we calculated the pressure-derived systolic time between pressure upstroke and the PA incisura. The PA incisura corresponds to pulmonary valve closure and the
pulmonary component of the second sound. We, thus, calculated the Ti/systolic time ratio. As Ti is also influenced by the dimension of the vascular tree, we calculated the Ti/b.s.a. ratio. For Pi (mm Hg) measurements, the intra- and interobserver variability was 1\% and 3\%, respectively. For Ti (ms) measurements, the intra- and interobserver variability was 7\% and 4\%, respectively.

**Statistical analysis.** The data are expressed as means ± SD. Pressures and time parameters were averaged out over 15 cardiac cycles. Comparisons at baseline were performed using the Mann-Whitney U test.

**RESULTS**

The characteristics of the study population are listed in Table 1. Pulmonary function was normal in the two groups. Age, sex ratio, heart rate, cardiac index, stroke volume and mixed venous oxygen saturation were similar in each group. Pulmonary hypertension was of similar severity in the two groups, as attested to by similar MPAP and TPRi (Table 1). Therefore, patients were matched for the steady component of PA load, thus allowing optimal comparisons of the pulsatile component of PA pressure.

**PAPP.** Individual pressure waveform tracings from all 14 patients are presented in Figure 2. The PAPP was similar in CPTE (57 ± 13 mm Hg) and PPH (53 ± 13 mm Hg) (p = 0.56). The normalized PAPP (PAPP/MPAP ratio) was also similar in CPTE (0.97 ± 0.16) and PPH (0.89 ± 0.13) (p = 0.30). Individual data points are presented in Figure 3, attesting to major overlap of PAPP indexes, with no cutoff value between the two groups. The pressure-derived systolic time was similar in CPTE (292 ± 37 ms) and PPH (288 ± 36 ms).

**Wave reflection.** In one of the 14 subjects (belonging to the PPH group), the inflection point Pi could not be clearly identified at baseline. Pi was similar in the two groups (p = 0.31) (Table 2). Pi was strongly related to MPAP in both CPTE (slope = 1.15; ordinate = +11 mm Hg; r = 0.99) and PPH (slope = 1.34; ordinate = +2 mm Hg; r = 0.98) (each p < 0.01). The relative increase in pressure amplitude above the inflection point (ΔP) and the augmentation index ΔP/PAPP were higher in CPTE than they were in PPH (each p < 0.01) (Table 2). Wave reflection of varying extent could be observed within each group (Fig. 2 and 4), in such a way that there was no cutoff value for ΔP/PAPP, allowing discrimination between CPTE and PPH.

The transit time Ti, Ti/systolic time and Ti/b.s.a. were shorter in CPTE than they were in PPH (each p < 0.01) (Table 2, Fig. 4). Given that one CPTE and one PPH share a common value of Ti (98 ms), there was no cutoff value allowing discrimination between CPTE and PPH. When the Ti/systolic time ratio was considered, a cutoff value (0.36) allowed discrimination between the two groups, without overlap. When the Ti/b.s.a. ratio was considered, a 57 ms/m² cutoff value allowed discrimination between CPTE and PPH without overlap (Fig. 4).

**DISCUSSION**

**PAPP in PPH and CPTE.** The main result of this study was that PPH and CPTE patients with pulmonary hypertension of comparable severity had similar pulse pressure and normalized pulse pressure (i.e., pulse pressure divided...
by mean pressure). In contrast with our results, a recent study has reported higher PAPP and normalized pulse pressure in CPTE than in PPH (26). However, in the previous study (26), there were major differences in the severity of pulmonary hypertension (as assessed by MPAP) between CPTE and PPH, and this confounds pulsatility comparisons. Other limitations of previous study (26) include the use of fluid-filled catheters and a retrospective study design, with almost twice the number of patients with CPTE than with PPH. Our results are consistent with other studies suggesting that changes in elasticity and pulse pressure are primarily due to an increase in MPAP in pulmonary hypertension (38,39). Given that our patients had severe pulmonary hypertension of comparable severity, one hypothesis could be that the PAs approach a similar limit of compliance in both groups (41). The chronically hypertensive lung may be an “end-stage” organ, and advanced pulmonary hypertension of whatever etiology may appear the same from the viewpoint of PAPP as both diseases result in major increases in MPAP caused by obliteration of the pulmonary vascular bed. On the other hand, we cannot exclude the possibility that pulse pressure is an index that is not sufficiently sensitive to discriminate between the potential differences in PA elastic properties in the two groups.

Cardiac index, heart rate and pressure pulse duration were similar in each group. This is consistent with the results showing that right ventricular ejection fraction and contractility decrease inversely with the level of elevated TPR (42). It is unlikely that enhanced right ventricular ejection contributed to the increased PAPP, given that stroke volume was low in the two groups. However, flow pulse was not recorded in our study, and similar stroke volume in CPTE and PPH may well be associated with differences in flow dynamics (e.g., peak velocity and acceleration).

**Wave reflection.** Abnormal pressure-wave reflection occurring in systole (10) was confirmed in PPH and was documented for the first time, to the best of our knowledge, in CPTE. In our study, CPTE exhibited greater wave reflection (i.e., higher augmentation index) and anticipated wave reflection (i.e., shorter transit time Ti) as compared with PPH. After taking into account the influence of b.s.a., which is a significant determinant of the dimension of the pulmonary vascular tree and, thus, wave reflection, a cutoff value was used to separate the two groups without overlap. Abnormal mechanical properties and geometry of proximal (main, lobar or segmental) pulmonary arteries in CPTE may lead to discontinuity in the caliber and elastic properties of these arteries, resulting in enhanced and earlier wave reflections (27). Changing the reflecting sites to a “more proximal” location may also be involved (8–13,19).

The normal pulmonary circulation exhibits very little wave reflection (5,8,27). In normally elastic PA, the low amplitude reflection peaks at or just after the dicrotic notch and seems to be one of the mechanisms optimizing the right ventricular-PA coupling (6,8,12,13). Clinical studies performed in the frequency domain have shown that wave reflection is increased and anticipated under conditions of

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**Table 2. Indexes of Wave Reflection**

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<thead>
<tr>
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<th>CPTE</th>
<th>PPH</th>
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<tbody>
<tr>
<td>Pt (mm Hg)</td>
<td>79 ± 11</td>
<td>87 ± 16</td>
</tr>
<tr>
<td>ΔP = SPAP – Pt (mm Hg)</td>
<td>15 ± 8</td>
<td>5 ± 4*</td>
</tr>
<tr>
<td>Augmentation index (ΔP/PAPP)</td>
<td>0.26 ± 0.09</td>
<td>0.09 ± 0.07†</td>
</tr>
<tr>
<td>Ti (ms)</td>
<td>90 ± 17</td>
<td>126 ± 16†</td>
</tr>
<tr>
<td>Ti/systolic time</td>
<td>0.30 ± 0.05</td>
<td>0.43 ± 0.08‡</td>
</tr>
<tr>
<td>Ti/b.s.a. (ms/m²)</td>
<td>48 ± 8</td>
<td>71 ± 10‡</td>
</tr>
</tbody>
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Values are means ± SD. *p < 0.05; †p < 0.01 vs. CPTE.

b.s.a. = body surface area; CPTE = chronic pulmonary thromboembolism (n = 7); PAPP = pulmonary artery pulse pressure; Pt = pulmonary artery pressure systolic inflection point; PPH = primary pulmonary hypertension (n = 6); SPAP = systolic pulmonary artery pressure; Ti = transit time.

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![Figure 3. Mean pulmonary artery pressure (MPAP), pulmonary artery pulse pressure (PAPP) and normalized pulmonary artery pulse pressure (PAPP/MPAP) in chronic pulmonary thromboembolism (CPTE) (open circles) and in primary pulmonary hypertension (PPH) (closed circles). Individual data points and means ± SD are indicated.](image-url)
pulmonary hypertension, whether in the setting of PPH (10), severe left heart failure and mitral stenosis (8,12) or coronary angioplasty (9). Reflected pressure waves add to the incident pressure, while flow reflections subtract from forward flow waves (27). Thus, in experimental and clinical settings where PA compliance is decreased and pulse wave velocity is increased, an earlier, increased reflected wave returns to the PA during systole rather than diastole, and this could contribute to the increase in PAPP (8–13,25), the prolonged time-to-peak pressure (43) and the midsystolic deceleration of pulmonary blood flow (44) in PPH.

Increased wave reflection is believed to be the main explanation of the midsystolic deceleration (notch) found on Doppler-derived systolic velocity pulmonary flow curves in pulmonary hypertension (25,45,46). Although it should be pointed out that the pressure wave cannot provide precise information on flow pulse, it is interesting to note that the mean Ti value we reported in PPH (126 ms) was close to that of the time-to-notch mean value (125 ms) reported by Torbicki et al. (46) in a study population of PPH similar to ours. Turkevich et al. (45) found an inverse correlation between time-to-notch and the severity of pulmonary hypertension. Others have suggested that some factors other than elevated PA pressure may be required for production of midsystolic notching (44) and that a shorter time-to-notch was mainly due to a more proximal reflection site (25,46). Consistent with the latter hypothesis, our study indicates that when patients are matched for MPAP, the more proximal location of the functional reflection site is associated with a shorter time-to-peak reflected pressure wave.

Although the main pulmonary branches are thought to be one major reflection site, it is accepted that reflections from the pulmonary microcirculation can be at least as important as those from the branching of large vessels (5). Assuming a similar pulse wave velocity of 4 m/s in PPH and CPTE (i.e., a twofold increase in pulse wave velocity in patients with elevated MPAP versus normotensive ones [40,47,48]), the Ti measured in our study would imply a functional reflection point at 25 cm in PPH (Ti = 126 ms) and 18 cm in CPTE (Ti = 90 ms). Turkevich et al. (45) have proposed that, given a time-to-notch of 120 ms in PPH and assuming a pulse wave velocity of 5 m/s in PPH, the reflection point was located at 30 cm, which they considered to be “compatible with a locus within the lung.” Obviously, however, the meaning of such extrapolations must be carefully discussed, especially in the case of the short pulmonary vessels, given that the extrapolated locations reflect functional aspects of the composite wave reflection phenomenon rather than true anatomical location of the reflection site(s) (31).

**Study limitations.** The PA input impedance defined in the frequency domain represents the most complete description of vascular hydraulic load (4–6). Although it is well-recognized that no single parameter can fully describe the reflection properties of a system of branching viscoelastic tubes as complex as the pulmonary vasculature (4–6,31), previous studies have demonstrated the usefulness of time-domain indexes of wave reflections (4–13,27,30). The fact that we could differentiate patients with PPH from patients with CPTE using pressure wave shape analysis at baseline must be viewed as one of the strengths of the study. The inflection point could be identified in 86% (6/7) of patients with PPH, and 100% (7/7) of patients with CPTE. Interestingly, midsystolic notching of PA flow velocity was found in 83% of patients with PPH and 100% of patients with CPTE in the study by Torbicki et al. (46). Further studies are needed to determine how time-domain indexes of wave reflection relate to frequency-domain indexes. The absence of an inflection point in 1/7 patients with PPH in no way indicates a lesser degree of wave reflection (43). In one patient with PPH, the augmentation index approached 0,
but PA pressure waveform was abnormal; the inflection point PI was clearly identified and occurred in mid systole, thus in keeping with elevated wave reflection. Finally, PPH and CPTE were matched for MPAP, but relative differences in the presentation of these patients for medical attention may also play a role in our results, a point that deserves further study.

Implications. First, it is suggested that PA pressure waveform analysis may be useful for distinguishing between PPH and CPTE, depending on the extent and timing of wave reflection. Second, in experimental and clinical settings where wave reflection is increased, the ventricular-vascular coupling is less efficient (8–15,30). In conditions of comparable severity of pulmonary hypertension, it may be postulated that more of the total expended power is "wasted" in oscillations in patients with CPTE than it is in patients with PPH. Wave reflection unfavorably loaded the still-ejecting right ventricle in the entire study population, and this could be more detrimental in CPTE than it is in PPH since this effect was achieved earlier in the ejection phase in patients with CPTE. This may lead to differences in the tolerance of the right ventricle to pulmonary hypertension between CPTE and PPH, a hypothesis that needs to be confirmed.

Conclusions. Our study indicated that patients with PPH and CPTE who had severe pulmonary hypertension of comparable severity had similar PAPP. Wave reflection was elevated in both groups, and CPTE had increased and anticipated wave reflection as compared with PPH. It was also suggested that pulsatile pressure is highly dependent on the prevailing MPAP in patients with pulmonary hypertension. The differences in wave reflection could contribute to differences in the pulsatile component of right ventricular afterload between PPH and CPTE.

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REFERENCES

34. Raffy O, Azarian R, Brenot F, et al. Clinical significance of the pulmonary vasodilator response during short-term infusion of prosta-