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Systolic and Mean Pulmonary Artery Pressures
Are They Interchangeable in Patients With Pulmonary Hypertension?

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Pulmonary hypertension (PH) is a common complication of numerous diseases, including left-sided heart diseases and chronic lung diseases and/or hypoxia, where PH is associated with exercise limitation and a worse prognosis. Other forms of PH include pulmonary arterial hypertension (PAH), chronic thromboembolic PH (CTEPH), and PH with unclear multifactorial mechanisms. Over the past decade, it has been documented that systolic pulmonary artery pressure (sPAP) may help estimate mean pulmonary artery pressure (mPAP) in adults with high accuracy and reasonably good precision (mPAP = 0.61 sPAP + 2 mm Hg). This strong linear relationship between sPAP and mPAP was unexpected from a classic physiologic point of view. Consistent results have been obtained from independent teams using either high-fidelity micromanometer-tipped PA catheters or fluid-filled catheters. Overall, the strong link between sPAP and mPAP has been documented over a wide range of PAPs, heart rate, cardiac output, wedge pressure, and causes of PH, during changes in posture and activity, and irrespective of patient's sex, age, and BMI. A review of available invasive data confirms that patients with CTEPH and idiopathic PAH matched for their mPAP exhibit essentially similar sPAP. Pressure redundancy may be explained by the dependence of PA compliance upon mPAP. The 25 mm Hg threshold used to define PH accurately corresponds to an sPAP of 38 mm Hg. Although the limits of the echocardiographic estimation of sPAP are widely documented, results from invasive studies may furnish an evidence-based sPAP-derived mPAP value, potentially useful in the multiparameter echocardiographic approach currently used to diagnose and follow patients with PH.

CHEST 2015; 147(4):943-950

ABBREVIATIONS: CTEPH = chronic thromboembolic pulmonary hypertension; dPAP = diastolic pulmonary artery pressure; iPAH = idiopathic pulmonary arterial hypertension; mPAP = mean pulmonary artery pressure; PA = pulmonary artery; PAH = pulmonary arterial hypertension; PAP = pulmonary artery pressure; Pej = pulmonary artery mean ejection pressure; PH = pulmonary hypertension; Pnotch = pulmonary artery notch pressure; sPAP = systolic pulmonary artery pressure

Pulmonary hypertension (PH) is defined on right-sided heart catheterization as resting mean pulmonary artery pressure (mPAP) ≥ 25 mm Hg. In adults, PH is a common complication of numerous diseases, including left-sided heart diseases and chronic lung diseases and/or hypoxia, where PH is associated with exercise limitation and a worse prognosis.[1,2] Other forms of PH include pulmonary arterial hypertension (PAH), chronic thromboembolic PH (CTEPH), and PH with

Manuscript received July 17, 2014; revision accepted October 7, 2014.

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DOI: 10.1378/chest.14-1755

unclear multifactorial mechanisms.[3] Thus, there is an urgent need to noninvasively diagnose PH in the majority of patients. In this respect, the point/counterpoint editorials by Rudski[4] and Rich[5] published in *CHEST* on this topic is especially welcomed. Both Rudski[4] and Rich[5] must be congratulated for having elegantly supported the "yes" and "no" answers, respectively. Although a multiparameter echocardiographic approach has been rightly advocated by numerous authors to rule in or rule out PH diagnosis,[4-8] the accurate estimation of systolic pulmonary artery pressure (sPAP) remains at the center of the debate.[4,5]

sPAP is currently estimated from both the continuous Doppler maximum velocity of tricuspid regurgitation and the estimated right atrial pressure, assuming that sPAP and right ventricular peak systolic pressure are equal. Because PH is defined by using mPAP, Rudski[4] discussed the corresponding sPAP threshold. This appears to be a difficult task. It was first indicated that if mPAP is two-thirds diastolic pressure plus one-third systolic pressure, and if one assumes an mPAP of 25 mm Hg and a pulmonary artery diastolic pressure of 15 mm Hg, this would correlate with an sPAP of about 45 mm Hg.[4] It was acknowledged that many authors use the previously proposed upper limit of normal sPAP of >30 to 35 mm Hg.[9] Two consensus documents were also quoted, indicating that "In the absence of other potential etiologies of PH, such as left heart disease or advanced lung disease, an estimated RV systolic pressure of greater than 40 mm Hg generally warrants further evaluation in the patient with unexplained dyspnea"[6] and that "If the estimated SPAP is >35 to 40 mm Hg, stronger scrutiny may be warranted to determine if PH is present, factoring in other clinical information."[7]

The cutoff values applied to large populations must be evidence based. In this respect, this commentary has two aims: (1) to summarize the main recent findings favoring that mPAP and sPAP are strongly related and provide essentially redundant estimates of pulmonary circulation, and (2) to discuss the potential implications for PH pathophysiology and diagnosis.

mPAP and sPAP Are Strongly Related in Most Forms of PH

In 2004, our group demonstrated that mPAP and sPAP were related through a strong linear relationship in adult patients prospectively studied by using a high-fidelity micromanometer-tipped pulmonary artery (PA) catheter,[10] according to the following equation:

$$\text{mPAP} = 0.61\,\text{sPAP} + 2\ \text{mm Hg}$$

Results were obtained in 31 subjects, namely nine control subjects, nine subjects with PAH, seven subjects with CTEPH, and six patients with postcapillary PH. This observation has been further confirmed by using both micromanometer-tipped[11-13] and fluid-filled[14,15] PA catheters (Table 1).

A strong linear relationship between mPAP and sPAP has been documented by Syyed et al[11] in 65 subjects (of whom 47 had PH of various causes) who were retrospectively studied by micromanometer-tipped PA catheters. A high degree of accuracy was maintained following changes in posture and activity.[11] A retrospective analysis of all studies having documented pulmonary artery pressure (PAP) by using micromanometer-tipped PA catheter measurements involved a total of 166 individuals, of whom 58% had PH caused by many different conditions. It confirmed high accuracy (0 mm Hg mean bias) and reasonably good precision (3 mm Hg SD of the bias) of our empirical mPAP estimate, with sPAP explaining 98% of mPAP variance.[12] The strong linear relationship between mPAP and sPAP has also been documented in patients with precapillary PH performing moderate to vigorous supine cycling.[13]

Two large-scale studies using fluid-filled PA catheters have documented the mPAP vs sPAP empirical relationship in patients with left-sided heart disease.[14,15] The corresponding equation was identical[14] or remarkably similar[15] to ours[10] (Table 1), although signal distortions are known to be unavoidable when using conventional catheters for pulsatile pressure analysis. Overall, the strong link between mPAP and sPAP has been documented over a wide range of mPAPs, diastolic PAP (dPAP), heart rate, cardiac output, PA wedge pressure, and causes of PH and irrespective of patient's sex, age, and BMI.[10-15]

Thus, in an attempt to predict mPAP, one pressure is enough, namely sPAP (single-pressure model). Thus, one does not necessarily need to know the dPAP value and then apply either the classic rule of thumb (two-thirds dPAP + one-third sPAP) or the proposed geometric mean of sPAP and dPAP[12] (dual-pressure models). From a classic physiologic point of view, this link between the steady and the pulsatile components of PA pressure was unexpected, and such a link is less