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Counterpoint: Can Doppler Echocardiography Estimates of Pulmonary Artery Systolic Pressures Be Relied Upon to Accurately Make the Diagnosis of Pulmonary Hypertension? No

Widely available, noninvasive diagnostic modalities are understandably attractive to both physicians and patients but can also be as equally dangerous as they are easy to order. Indeed, such dangers have been realized in the field of pulmonary hypertension (PH), seemingly correlating with the increased popularity and accessibility of Doppler echocardiography (DE). However, prior to delving into the complexities of this topic, careful consideration and review of important PH-related nomenclature is needed to put into the proper perspective this clinically relevant question

of whether DE estimates of systolic pulmonary artery pressures (sPAPs) can be relied on in the accurate diagnosis of PH.

Pulmonary arterial hypertension (PAH) is a devastating, progressive disease that ultimately results in right ventricular failure and death despite modern-day therapeutics. Although PH itself is common to many medical conditions, PAH remains a relatively rare condition.¹ The World Health Organization classification of PH comprises five groups, with Group I reserved for those conditions associated with PAH.² To diagnose PAH requires that at least three conditions be met³: (1) the presence of a mean pulmonary artery pressure of ≥ 25 mm Hg, (2) the presence of an elevated pulmonary vascular resistance of at least 3 Wood units, and (3) the exclusion of the presence of the other conditions found in Groups II through V PH. Thus, although many health-care professionals loosely use the term “PH” when referring to PAH, this often results in confusion for both the patient and the treating physicians and is a critical distinction. For example, present-day PH-specific medical therapies are approved for only the treatment of Group I PH (ie, PAH). Although I would readily discuss the issues as they relate specifically to diagnosing PAH with DE, the question that is being posed to me in this forum and, thus, the one that I will attempt to answer is not whether DE can be used to reliably diagnose PAH (the answer to this question, in case you were wondering, is no), but whether it can be used to reliably diagnose PH (unfortunately, also no). Thus, in the spirit of the recent US political debates, I will do what our political nominees typically do not: I will actually answer the question posed, namely, can DE estimates of sPAPs be relied on to accurately make the diagnosis of PH?

ARE DE ESTIMATES OF SPAPs ACCURATE?

In 1984, Yock and Popp⁴ reported that the quantitation of the regurgitant tricuspid jet from DE measurements to estimate sPAP correlated significantly with sPAP measured by right-sided heart catheterization (RHC) in 20 patients. Several similar observations were published subsequently from which it became widely accepted that DE provides an accurate, noninvasive measurement of sPAP.⁵⁻⁸ However, what is underappreciated by many of us nonstatisticians and which may lead to erroneous conclusions is that measurements that correlate with each other do not necessarily agree with each other (ie, one of the measurements may be accurate and the other inaccurate, yet both may strongly correlate). This important concept was elegantly reinforced by Bland and Altman,⁹ who reminded us that two modalities

(ie, DE and RHC) designed to measure the same variable (ie, sPAP) will in fact almost always correlate with each other but may not actually be in agreement. Indeed, because this exact sort of discrepancy has been increasingly recognized by many cardiologists and pulmonologists in the clinical setting (ie, the poor agreement between DE estimates of sPAP and those measured at RHC), there was a strong impetus for two separate groups of investigators from large PH referral centers to carefully and methodically address this important question of the accuracy of DE estimates of sPAP in PH.

Fisher et al¹⁰ analyzed the accuracy of DE estimates of sPAP in 65 patients with PH who underwent invasive RHC within 1 h of the DE examination and found that DE estimates of sPAP were not accurate compared with the invasive gold standard. More recently, Rich and colleagues¹¹ studied 160 patients with PH undergoing DE and invasive sPAP measurements within a short time frame of each other and also studied an additional 23 patients in whom the DE and RHC were performed simultaneously. They too found that DE is a frequently inaccurate method of sPAP estimation in PH and concluded that DE estimates of sPAP should not be considered a reliable method in diagnosing and managing PH. Particularly remarkable between both of these studies was the striking similarity of the findings, namely that (1) DE and invasively measured sPAP correlated to almost the exact same degree ($r = 0.66$ and 0.68 , respectively, $P < .001$), yet (2) DE estimates of sPAP were highly inaccurate (in 48% and 51% of cases, respectively) compared with invasive measurements.^{10,11}

WHY ARE DE ESTIMATES OF SPAP INACCURATE AND CLINICALLY UNRELIABLE?

Numerous reasons easily explain the relative inaccuracy of DE estimates of sPAP. To best understand the many pitfalls associated with DE, let us review the steps involved and the data required to estimate sPAP during the DE examination.

To even contemplate using DE to estimate sPAP, tricuspid regurgitation (TR) must be present. Thus, in the absence of TR, sPAP simply cannot be determined by DE. When TR is present, however, the peak TR velocity must be precisely determined, and this is where many of the problems with DE accuracy typically begin. First, all intracardiac pressure measurements (including sPAP) must be measured at end expiration, and failing to do so often will result in significant pressure underestimation (particularly in the presence of obesity and lung disease).¹² Second, it is important that the Doppler beam and the TR jet be in nearly perfect, parallel alignment. If they are not

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