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Please note: Dr. O'Rourke is a founding director of AtCor Medical Pty. Ltd., manufacturer of systems for analyzing arterial pulse.

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Increased Aortic Stiffness in Hypertrophic Cardiomyopathy

Different Methods, Same Conclusions?

We read the report by Boonyasirinant et al. (1) with great interest. The major finding of this study was that increased aortic stiffness, as indicated by increased magnetic resonance imaging (MRI)-derived pulse wave velocity (PWV), is evident in hypertrophic cardiomyopathy (HCM) patients and is more pronounced in those with myocardial fibrosis. The results are impressive, but we feel that a few additional comments are necessary.

Boonyasirinant et al. (1) used MRI-PWV analysis to quantify aortic stiffness in their clinical study. Measurement of PWV by different tonometric, piezoelectronic, oscillometric, and MRI methods are widely used scientific tools. However, there is another way to evaluate aortic stiffness, for which 2 important variables should be noted: 1) the change in volume due to blood injection into the aorta; and 2) the pressure change caused by this volume change (2). Together with measurement of forearm systolic blood pressure (SBP) and diastolic blood pressure (DBP) changes, aortic systolic diameter (SD) and diastolic diameter (DD) or cross-sectional areas at different levels of aorta can be measured with echocardiography, computed tomography, or MRI. Using these parameters, indexes or moduli can be calculated characterizing aortic elasticity (3). The most important parameters are listed here:

- Aortic strain = (SD − DD)/DD
- Aortic stiffness index (beta) = ln (SBP/DBP)/[(SD − DD)/DD], where SBP and DBP are the systolic and diastolic blood pressures, and ln is the natural logarithm
- Aortic distensibility = 2 × (SD − DD)/[(SBP − DBP) × DD]
- Aortic elastic modulus E(p) = (SBP − DBP)/[(SD − DD)/DD]
- Young's circumferential static elastic modulus E(s) = E(p) × DD/2h, where h indicates diastolic intima-media thickness

Boonyasirinant et al. (1) were the first to demonstrate alterations in aortic distensibility in HCM. However, further investigations are warranted to examine the previously mentioned parameters in HCM, especially with versus without left ventricular outflow gradients. Moreover, correlations between PWV and echocardiography-derived parameters should be confirmed in HCM as well.

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Reply

We thank Dr. O'Rourke and colleagues and Dr. Nemes and colleagues for the interest in our study (1). A common thread is that each promotes alternative methods for computing aortic stiffness to the method we chose to employ, namely pulse wave velocity (PWV) computed by magnetic resonance imaging (MRI).

We are in agreement with Dr. Nemes and colleagues that more work is needed between and among these various approaches. We also recognize that many techniques exist to quantify aortic stiffness, and each is subject to its own characteristic strengths and weaknesses. Reliable quantification of PWV is dependent on accurate measurement of both the aortic flow (or pressure) wave at 2 measurement sites as well as the distance between these 2 sites. Previously, in numerous studies, MRI has been shown as a reliable technique for quantifying the aortic flow wave; its longitudinal dimensional accuracy is unparalleled, although it does suffer from a relatively low temporal resolution when compared with alternate techniques.

One of the concerns raised by Dr. O'Rourke and colleagues was that our PWV values for normal controls were lower than previously reported. Despite this assertion, our values are, indeed, consistent with several previous reports (2–5). Nonetheless, it is important to keep in mind that 2 different techniques measuring the same physiological parameter are unlikely to provide precisely the same results, and, in fact, it has been shown recently that PWV results may vary not only based on the technique used to acquire the data, but also based on the analysis method used to determine their value (6).

There are several possible explanations for the differences in PWV between measurement techniques, and each could contribute to the observed discrepancies. For instance, PWV increases along the length of the aorta as the artery becomes less elastic (7), and has been demonstrated in a study using MRI (3). The technique of arterial tonometry, which is restricted to measurement sites at the carotid and femoral arteries, provides an average PWV between these points, but it is incapable of determining regional values such as those we reported between the ascending and proximal descending aorta. At least in normal subjects, PWV values by MRI, restricted to the thoracic aorta, should be somewhat lower than values obtained by arterial tonometry. In addition, nonimaging techniques are subject to errors in determining the aortic length between measurement sites, particularly in the case of tortuous aortas, which occur, for example, as part of the normal aging process and in the setting of significant atherosclerotic disease. We are unaware of corrections available to compensate for this uncertainty.

Dr. O'Rourke and colleagues also raised concerns about the relatively large SDs seen in the hypertrophic cardiomyopathy population. This would be a legitimate concern if large SDs were present in the normal control subjects; however, they were not. Instead, this more reasonably reflects the heterogeneity within this large group of hypertrophic cardiomyopathy patients including, but not limited to: variations in myocardial mass, the presence or absence of interstitial fibrosis, the degree and amount of interstitial fibrosis, and the presence or absence of left ventricular outflow tract obstruction.

Of course, there exist other alternative measures of aortic stiffness, some of which are mentioned by Dr. Nemes and colleagues. These measures are attractive because they offer a local estimate of stiffness, though they rely on accurate measurement of aortic distension and, in some cases, aortic pressure. For ease of measurement, arterial distension is sometimes approximated by its 1-dimensional analog (i.e., change in diameter), and cuff (brachial) pressure is often used as an approximation of central pressure, despite the fact there is an assumption of absence of central stenoses, and that amplification along the arterial tree can result in large differences between these pressures (7).

In this and previous studies, PWV has been shown to be a robust and completely noninvasive index of arterial stiffness, regardless of the method used in its quantification. However, when MRI is the method of choice, PWV can be included as part of a comprehensive and clinically validated imaging evaluation of cardiovascular disease, and with no additional time, cost, or equipment needed. As with any quantification technique, care must be taken to consider the methodology being used when comparing results between patient groups and between and among different studies.

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