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creased arterial stiffness and excessive pressure pulsatility have emerged as important risk factors for a number of common disorders of aging, including cardiovascular disease, stroke, cognitive impairment, and renal disease. The triple-threat combination of robust associations between arterial stiffness and the foregoing disorders, a marked increase in arterial stiffness with advancing age and the graying of our society, has led to intensive efforts to identify mechanisms that contribute to arterial stiffening and widening pulse pressure to define interventions to prevent or reverse stiffness and potentially reduce the substantial burden of related disease.

Wave reflections complicate the task of evaluating arterial hemodynamics and play an unclear role in the foregoing diseases. When the heart ejects, ventricular outflow interacts with characteristic impedance of the proximal aorta to produce the forward pressure wave, which travels down the normally compliant aorta at a finite pulse wave velocity (PWV). When the forward wave encounters impedance mismatch, such as a branch point or a change in diameter or wall properties, a partial reflection occurs. Innumerable reflections arising from locations distributed throughout the arterial tree summate into a remarkably discrete reflected pressure wave with amplitude ~40% of the incident wave. The summated reflected wave returns to the central aorta in mid-systole, creating an inflection point and secondary late systolic pressure rise that often augments central aortic systolic and pulse pressure.1 Variable timing of this retrograde-traveling reflected wave creates regional inequalities in systolic and pulse pressure and, therefore, complicates interpretation of single point pressure measurements, such as standard brachial blood pressure, which may differ from central arterial pressure.

Augmentation index (AI), which expresses late systolic pressure augmentation as a percentage of pulse pressure, is frequently used to assess wave reflection (Figure). AI, which depends on both timing and amplitude of the reflected wave, has also been widely cited as a measure of arterial stiffness based on the assumption that increasing PWV leads to progressively earlier wave reflections from a relatively fixed reflecting site. The resulting progressive systolic overlap of the forward and reflected wave has been proposed as the principal determinant of increasing pulse pressure with advancing age.2 However, recent community-based studies have challenged these seemingly straightforward assump-

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generally the case, the forward and reflected waves summate, resulting in augmentation of pressure and deceleration of ventricular outflow. Based on examination of a series of studies performed using invasive catheter-mounted pressure and flow sensors, Westerhof et al.\textsuperscript{10} hypothesized that the shape of the truncated aortic flow waveform could be estimated by using a triangular pseudoflow waveform. The pseudoflow waveform is constructed by assuming 0 flow during diastole and connecting lines from 0 at the timing of the pressure waveform foot to some empirical maximum value at the estimated timing of peak flow and then back to 0 simultaneous with the dicrotic notch. Timing of peak flow was set equal to inflection point timing, signaling the foot of the reflected pressure wave. Alternatively, peak flow was assumed to occur at 30% of the ejection period. Measured pressure and pseudoflow waveforms are then used to separate forward and reflected wave components, of which the peak-trough amplitudes are easily assessed (Figure).

A major difference between AI and the Westerhof approach lies in the definition of the baseline used to compute the amplitude of the reflected wave. In the common situation where AI is $>0$, only the component of the reflected wave that projects above the forward wave horizon is considered, which effectively sets a horizontal baseline at the level of the forward wave peak (Figure). This approach suffers from a “tip-of-the-iceberg” phenomenon, because the bulk of the reflected wave is submerged along the falling edge of the forward pressure wave. In contrast, the Westerhof “triangulation” approach turns the pressure baseline on its side and thereby explores the full vertical amplitude of a reflected wave clinging to the falling edge of the forward wave peak (Figure). The Westerhof approach also considers diastole, where a substantial component of the reflected wave may be located in cases with a relatively late or broad reflected wave. In cases where the early forward wave peak is dominant (AI $<0$), AI provides essentially no information on reflected wave amplitude, per se, whereas the Westerhof approach is relatively insensitive to this transition from an augmented to a nonaugmented state. Thus, one would expect, and data presented in the article suggest, that the Westerhof approach offers a more robust estimation of reflected wave amplitude, free of the confounding effects of reflected wave timing (compare $R^2$ values in Westerhof Figure 4).

However, there are potential limitations to the new approach. The number of cases evaluated in this proof-of-principal article is small, and case selection bias (i.e., an indication for cardiac catheterization) may have affected the results. Furthermore, the choice of a triangular flow wave-
form shape may have limitations. Effects of wave reflection on the central pressure and flow waveforms represent a compromise between pressure augmentation and flow deceleration. The settling point for this compromise depends on ventricular structure, function, and contractility. In cases where the ventricle is able to augment pressure, for example, a concentrically hypertrophied, pressure-loaded left ventricle, pressure augmentation will predominate over flow deceleration, and the falling edge of the measured flow waveform will be convex. Linear extrapolation to 0 of the falling edge of the pseudoflow waveform will, thus, underestimate true flow, and reflected wave amplitude will be overestimated. In other cases, for example, those with impaired ventricular function or extreme reflected wave intensity, deceleration of ventricular outflow will be accentuated, the flow waveform will be concave, and reflected wave amplitude will be underestimated. It seems that the latter scenario predominated in this small series of cases, because the slope of the relation between estimated and actual reflected wave magnitude is 0.79 to 0.82 in Westerhof Figure 4, whereas values typically range between 30% and 50%. The new technique proposed by Westerhof et al., and the traditional AI approach described by Murgo et al are complementary. The Westerhof approach offers a potential advantage in terms of estimating amplitude of forward and reflected waves free of the vagaries of variable overlap and an uncertain baseline for the reflected pressure wave. On the other hand, AI provides an integrated summary of the relations among reflected wave timing, amplitude, and ventricular function. For example, higher AI in the presence of typical midsystolic timing and nominal reflected wave amplitude may be indicative of (rather than a cause of) ventricular hypertrophy. Conversely, low AI in the presence of increased reflected wave amplitude and early timing may be a sign of impaired ventricular function. When substantial central pressure augmentation is present, interventions that reduce wave reflection produce reductions in central systolic and pulse pressure that are generally underestimated by the change in brachial systolic and pulse pressure, because the reflected wave determines peak pressure in the aorta but not the arm. This important phenomenon might be overlooked if attention is focused on reflected wave amplitude without considering the associated change in central pressure augmentation. Regardless of which technique is used, it is important to emphasize that these derived measures of wave reflection are likely to have complex and potentially inverse relations with aortic stiffness. With this caveat in mind, triangulation will likely prove useful in our attempts to navigate the peaks of pulsatile hemodynamics.

Disclosures
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Triangulating the Peaks of Arterial Pressure
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