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ASH Position Paper

American Society of Hypertension position paper: central blood pressure waveforms in health and disease

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Abstract

A number of devices are available which noninvasively estimate central aortic blood pressure using a variety of approaches such as tonometry or oscillometry. In this position paper, we discuss how the central pressure waveform is generated and measured, how central pressure waveforms appear in health and disease, the predictive value of central blood pressure measurements, the effects of interventions on waveforms, and areas of future need in this field of clinical and research endeavor. J Am Soc Hypertens 2016;10(1):22–33. © 2016 American Society of Hypertension. All rights reserved.

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Introduction

There is a century-old tradition of indirect (noninvasive) measurement of the systolic blood pressure (SBP) and diastolic blood pressures (BPs) in the brachial artery, using a cuff, as an index of the hemodynamic state of the arterial circulation.[1] However, BP is determined by a complex interaction of the pressure generated by the left ventricle (LV) and the impedance to blood flow exerted by the vasculature, and measurement of brachial cuff BP alone does not provide information as which of those two factors are predominant.[2,3] In addition, it is now understood that the SBP generated by the LV is further affected by reflected pressure waves from points of discontinuity in the arterial tree, such as bifurcations and abrupt decreases in vascular caliber.[4,5] Reflected waves modulate the arterial waveform at various points in the circulation, so that the pressures in the aorta are different (less) from those in more distal vessels, such as the brachial artery.[5,6] This is clearly important, as it is the properties of the arterial tree and aortic hemodynamics

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that constitute the input impedance (or afterload) to LV ejection and will therefore be a determinant of LV hypertrophy and myocardial oxygen demand.[2,3]

Measurement of brachial SBP and diastolic BP alone provides only indirect information about the adequacy of tissue and organ perfusion. It does not tell us what the central aortic BP is and therefore the impedance to LV ejection. It does not tell us about the relative contributions to BP of LV ejection and of distal vascular stiffness and resistance. It also does not provide information about the quantitative role of reflected pressure waves in generating the central SBP. To further our understanding of the hemodynamics of the circulation, efforts over the last several decades have sought to noninvasively assess central aortic BP, and from an analysis of the arterial waveform to dissect out the relative contributions of LV ejection and wave reflection, and to discern the longitudinal effects of these interactions on organs supplied directly by the aorta, such as the myocardium, the kidney, and the brain.[7]

In this American Society of Hypertension sponsored position paper, we will review the physiology of the central aortic pressure waveform and how it is generated and measured, how it changes with aging and comorbidities, its predictive utility for target organ damage, the effects of medication on the waveform, and then conclude with some recommendations regarding areas of needed research in this aspect of clinical hemodynamic measurement.

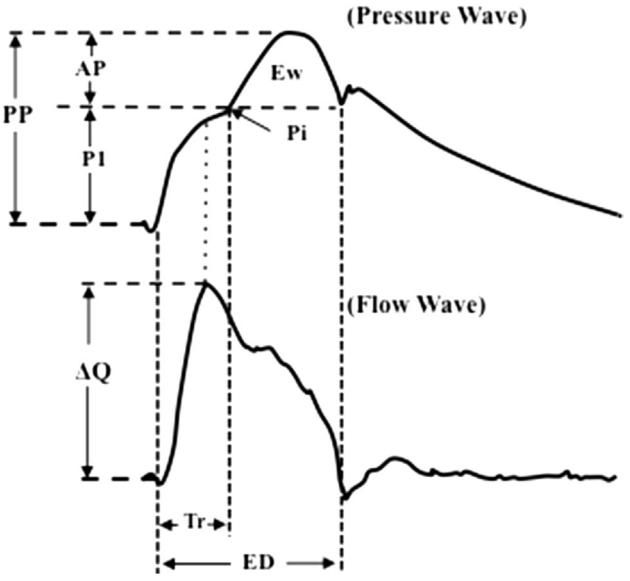

Figure 1. Measured ascending (central) aortic pressure (top) (Murgo type A wave) and flow velocity (bottom) waveforms from a middle-aged subject. AP is the estimated amplitude of the forward-traveling pressure wave, and P1 is the estimated amplitude of the backward-traveling (or reflected) pressure wave. ΔQ is peak aortic flow velocity, and T_r is the time of onset of the reflected pressure wave. P_i is the inflection point, ED is ejection duration, and Ew indicates wasted LV energy. AP, augmented pressure; LV, left ventricle; PP, pulse pressure.

Generation of the Central Aortic Pressure Waveform

The systemic arterial tree is made up of a system of distensible tubes along which pressure and flow waves, generated by LV ejection, are transmitted and reflected. Early recordings of ascending aortic pressure and flow waves, and pulse wave analysis (PWA), were obtained in the cardiac catheterization laboratory using high-fidelity multisensor (pressure and velocity) catheters.[5] During one cardiac cycle, the generated wave has sufficient time to travel to the periphery and back.[5] Pressure and flow waves measured in the ascending (or central) aorta are markedly different because reflected waves influence their contour. The initial flow pulse generated by LV ejection produces a forward (or incident) pressure wave (Figure 1). This forward-traveling wave, with estimated amplitude P1, propagates along the aorta at a finite velocity and is reflected backward toward the heart. This backward-traveling wave, with estimated amplitude augmented pressure (AP), has the same wavelength as the forward-traveling wave, and when the two waves encounter each other, they combine through superposition (addition) to form the measured pressure wave.[8–11] The contour of the forward wave depends on the forward flow wave and the elastic properties (characteristic impedance, Z_c) of the ascending aorta.[2,5] The characteristics of the reflected pressure wave depends on a more complex set of determinants than the forward wave, namely the physical properties (stiffness, taper, and branching) of the entire arterial tree (elastic plus muscular arteries), pulse wave velocity (PWV), the round-trip travel time (T_r) of the wave from the heart to the periphery and back, and the distance to the major "effective" reflecting site in the lower body[5,12]; the major part of reflections occurs at the arterioles.[5,13,14] Aortic augmentation index (AIx), defined as the ratio of the AP divided by the central pulse pressure (PP; see Figure 1), is an estimate of the percent contribution of wave reflection to central aortic PP. The reflection magnitude (RM) is the ratio of the backward (P_b) and forward (P_f) waves and is obtained by the technique of wave separation (see Figure 2). Increased arterial stiffness, for example with age and hypertension, increases PWV and causes early return (T_r decreases) of the reflected wave from the lower body reflecting sites to the heart during systole when the ventricle is still ejecting blood.[5] As T_r decreases, both AP and systolic duration of the reflected wave increase. This mechanism augments both ascending aortic SBP and PP, an effect that increases arterial wall stress, potentiates the development of coronary artery atherosclerosis, elevates LV afterload, and increases LV mass and myocardial oxygen demand while decreasing stroke volume.[15] Because the reflected wave and associated boost in pressure (LV