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Review

Central Blood Pressure as an Index of Antihypertensive Control: Determinants and Potential Value

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ABSTRACT

The measurement of central blood pressure has generated interest as a tool in predicting cardiovascular events. The purpose of this article is to review the meaning and measurement of the central blood pressure and consider its potential value as an index of the antihypertensive response. Indirect estimation of central aortic pressures is obtained by the study of the radial pulse wave compared with a central pulse wave contour measured at the carotid or femoral artery level. The sum of the forward pressure wave created by ventricular contraction and of the reflected pressure wave from the peripheral arterial system produce the peak systolic blood pressure in the aorta. Measurement of the peripheral reflected-wave contribution to aortic blood pressure can be quantified as the augmentation index. Also, the increase in the rapidity of this travelling wave can be measured as the pulse wave velocity. These 2 parameters are considered to be valid indices of the peripheral arterial stiffness. Along with the calculation of systolic and diastolic aortic pressures, these measurements can give a better understanding of the actual central blood pressure to which core organs like heart, brain, and kidneys are submitted. There is tantalizing evidence for the potential value of central blood pressure as a useful index of antihypertensive action, but until clear evidence is obtained, its use should continue to be considered exploratory.

RÉSUMÉ

La mesure de la pression artérielle centrale a suscité l'intérêt comme outil de prédiction des événements cardiovasculaires. Le but de cet article est de passer en revue la signification et la mesure de la pression artérielle centrale, et d'examiner sa valeur potentielle comme indice de la réponse antihypertensive. L'estimation indirecte des pressions aortiques centrales est obtenue par l'étude de l'onde de pouls radiale qui est comparée au contour de l'onde de pouls centrale mesurée au niveau de la carotide ou de l'artère fémorale. La somme de l'onde de pression antérograde créée par la contraction ventriculaire et de l'onde de pression réfléchie provenant du système artériel périphérique produit la pression artérielle systolique maximale dans l'aorte. La mesure de la contribution de l'onde réfléchie périphérique à la pression artérielle aortique peut être quantifiée en tant qu'indice d'augmentation. Aussi, l'augmentation de la rapidité de cette onde progressive peut être mesurée en tant que vitesse de l'onde de pouls. Ces 2 paramètres sont considérés comme étant des indices valables de la résistance artérielle périphérique. Outre le calcul des pressions systoliques et diastoliques aortiques, ces mesures peuvent offrir une meilleure compréhension de la pression artérielle centrale actuelle à laquelle les organes principaux comme le cœur, le cerveau et les reins sont soumis. Il existe des données scientifiques intéressantes sur la valeur potentielle de la pression artérielle centrale comme indice utile de l'action antihypertensive, mais jusqu'à ce que des données scientifiques claires soient obtenues, son utilisation devrait encore être considérée de manière exploratoire.

Since its introduction more than a century ago, brachial artery measurement of blood pressure (BP) has been performed for the diagnosis of hypertension and follow-up of its treatment. Compelling evidence has supported the value of higher levels of brachial artery BP as a strong risk factor for heart disease and strokes[1] and have shown that its reduction using antihypertensive medication is associated with an improvement in prognosis.[2] There is growing evidence that measurement of central (aortic) BP, which is the pressure directly exerted on the brain, heart, and kidneys, is different from the BP measured in the arm, because of an amplification effect that increases the central BP. Hypertension is characterized by a reduction in the calibre and number of small peripheral arteries with an increase in mean arterial pressure, which is a product of cardiac output and peripheral vascular resistance.[3] These peripheral arteries are muscular, with a high proportion of collagen fibres and therefore are less distensible. In comparison, aortic and carotid arteries are predominantly made of elastin fibres. The arterial wall of these large arteries will permit filling during systole by distension and will push blood forward in a steady flow during diastole as the artery recoils. Therefore, arterial stiffness is lowest in the elastic ascending and thoracic aorta and highest in distal arteries, such as the tibial artery. Progressive loss of elasticity is encountered with

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age and hypertension and is responsible for the increase in pulse pressure (PP) in peripheral arteries.[3] The pressure wave generated by the left ventricle travels down the arterial tree and then is reflected back centrally at the arterial-arteriolar junction. Consequently, the total pressure waveform in the aorta becomes the sum of the forward travelling waveform generated by the left ventricle and the backward reflected wave from the peripheral muscular and stiffer arteries. The backward (reflected) wave causes an increase in the central peak systolic BP, generating an increased PP. This increase in total aortic PP is called the augmentation pressure (Fig. 1) and is expressed as a percentage of the total pressure by the augmentation index (AIx). Arterial stiffness of limb vessels rapidly increases with distance from the heart, leading to a narrower wave with greater systolic BP. As a result, brachial systolic BP and PP are greater than central pressures in young individuals, whereas diastolic BP is constant.[4] Hypertension, high lipid levels, and smoking all lead to increase of the central BP.[5] With aging, there is a disappearance of the PP gradient along the arterial tree, leading to greater central BP because of a more pronounced stiffening of the central rather than peripheral arteries.[4]

Measurement of Central BP and Prognostic Value

Central pressures are derived from noninvasive techniques of measurement of radial or carotid pulses, and a validated generalized transfer function is used to estimate central pressures from the peripheral signal.[6] These involve applanation tonometry, in which transcutaneous pressure transducers at the end of a probe obtain pressure waveforms that are almost identical to those obtained using intra-arterial measurement.[7] This technique is suitable for radial, carotid, or femoral arteries. The carotid waveform is then used as a surrogate for that of the aorta. Another method is a mathematical description of the charge from the input to output signals to derive an aortic waveform from measurements obtained at the radial artery. Computerized programs then adjust for heart rate, height, and age. Hence, central systolic BP, diastolic BP, and PP are obtained and indices of arterial stiffness such as AIx and pulse wave velocity (PWV) are estimated.[8] The general transfer functions of applanation tonometry have a range of error, but it is less than for standard brachial cuff pressure with a sphygmomanometer or an oscillometric device.[9] Intrinsic variations in the measurements provided by different instruments are possible and can be operator-dependent.

The measurement of the AIx, which is a quantification of the arterial wave reflection on total BP, can vary with changes in heart rate, cardiac contractility, and age. PWV is the measurement of aortic pulse velocity; it is assessed by measuring the distance between 2 arteries (usually the carotid and femoral arteries) and dividing by the transit time. Greater arterial stiffness, which means less compliant arteries, will result in quicker wave travel to and from the periphery. PWV has emerged as a better marker of arterial stiffness, because of its relative ease of measurement and reliability, although some variability has been encountered in different types of populations, with age and different BP levels. It is not influenced by smoking, dyslipidemia, or sex, but to some extent by heart rate and diabetes.[10] Mitchell and colleagues,[11] in a prospective study of 2232 participants in the Framingham Heart Study after a mean follow-up of 7.8 years, concluded that the best individual predictor of a first major cardiovascular (CV) event by a pulsatile hemodynamic measure is the PWV (Fig. 2). Vlachopoulos et al. conducted a systematic review of 15,877 subjects and concluded that aortic PWV is a strong predictor of future CV events and all-cause mortality and has a predictive value independent of classic CV risk factors and other potential confounders.[12] For total CV events and CV mortality, the relative risk of a high PWV was greater in high-risk populations compared with low-risk populations. These findings suggest that measurement of arterial stiffness can capture CV risk from a genetic background and cumulative damage from CV risk factors on the arterial wall. Recently, the Reference Values for Arterial Stiffness Collaboration group in Europe has published reference values for the PWV.[13] Although normal PWV values might overlap between younger and older individuals and many CV risk factors are not all quantifiable (stress, positive family history, and others), the mean normal value for individuals aged younger than 30 years is 6.2 m/s and for age 70 years and older is 10.9 m/s.[13]

Central BP might be a better predictor of target organ damage than standard brachial BP. For example, it correlates

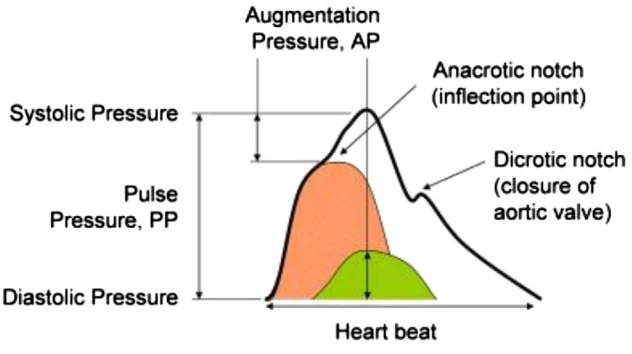

Figure 1. The augmentation index is a ratio calculated from the blood pressure waveform (augmentation index = Δ P/pulse pressure). It is a measure of the enhancement (augmentation) of central aortic pressure by a reflected pulse wave (shown in **green** in the graph). P, pressure. Reproduced with permission from USCOM.

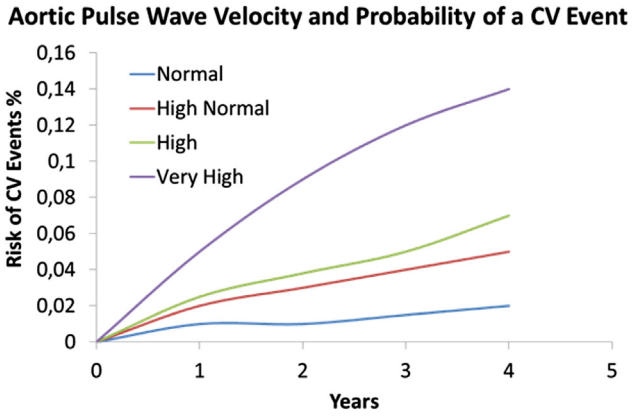

Figure 2. PWV and risk of a major CV event. The greater the PWV, the greater is the risk of a major CV event over time. CV, cardiovascular; PWV, pulse wave velocity. Data from Mitchell et al.[11]