Assessment of Arterial Stiffness with Pulse Wave Velocity or Augmentation Index: Which Method is the Best?: Scientific Letter

Arteriyel Sertliğin Nabız Dalga Hızı veya Artırma Göstergesi ile Değerlendirilmesi: En İyi Yöntem Hangisi?

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ABSTRACT Assesment of large arterial stiffness is increasingly used in clinal settings. Although there are several invasive and non-invasive methods such as carotid-femoral pulse wave velocity (PWV) and augmentation index (AI), researchers and clinicians still face problems in selecting the best methodology for their specific use. PWV, which is defined as the velocity of the arterial pulse for moving along the vessel wall, plays an important clinical role in defining patients under high cardiovascular risk and it is inversely correlated with arterial elasticity and relative arterial compliance. PWV along the aorta can be measured by using two ultrasound or pressure sensitive transducers fixed transcutaneously over the course of a pair of arteries separated by a known distance: the femoral and right common carotid arteries. PWV is calculated from measurements of pulse transit time and the distance, according to the following formula: PWV (m/s)= distance (m)/transit time (s). AI is defined as Dp/pp (where Dp is the difference between the late systolic peak and the mid-systolic peak and pp is the amplitude of the pulse pressure wave). Central blood pressure and pulse pressure, the AI and PWV, which are often used incorrectly as interchangeable indexes of arterial stiffness, increase with age, hypertension, diabetes mellitus and hyperlipidemia, and are associated with target organ damage such as left ventricular hypertrophy, microalbuminuria, carotid intima-media thickness and endothelial dysfunction. Althouhg the carotid-femoral (aortic) PWV is the gold standard test for assessing central arterial stiffness, the AI reflects stiffness of the systemic arterial tree. This paper summaries the advantages and disadvantages of PWV and AI in the assessment of arterial stiffness.

Key Words: Pulse; arteries; atherosclerosis; inflammation

ÖZET Büyük arterlerin sertliğinin değerlendirilmesi klinikte giderek artan sıklıkta kullanılmaktadır. Arteriyel sertliğin değerlendirilmesinde karotid-femoral nabız dalga hızı ve artırma göstergesi gibi invaziv ve non-invaziv yöntemler olmakla birlikte, araştırmacılar ve klinisyenler, bunların özgül kullanımları için en uygun yöntemi seçmede halen sorun yaşamaktadırlar. Nabız dalga hızı, arteriyel nabzın damar duvarı boyunca hareketinin hızı olarak tanımlanır ve arteriyel sertliğin bir göstergesi olarak yüksek kardiyovasküler risk altındaki hastaları tanımlamada önemli rol oynar. Arteryel elastisite ve rölatif arteriyel kompliyans ile ters ilişkilidir. Nabız dalga hızı aorta boyunca belli bir aralıkla ayrılmış bir çift arter boyunca (femoral ve sağ ana karotid arterler) transkütanöz yerleştirilmiş iki ultrason veya basınca duyarlı iletme sistemleri kullanılarak nabız transit zamanı ve uzaklık ölçümlerinden şu formüle göre hesaplanır: Nabız dalga hızı (m/s)=uzaklık (m)/ transit zamanı (s). Artırma göstergesi Dp/pp olarak tanımlanır (Dp: Geç sistolik zirve ve orta sistolik zirve arasındaki fark; pp: Nabız basınç dalgasının amplitüdü). Sıklıkla yanlış şekilde birbirinin yerine kullanılan arteriyel sertlik göstergeleri olan santral kan basıncı ve nabız basıncı, artırma göstergesi ve nabız dalga hızı yaş, hipertansiyon, diabet ve hiperlipidemi ile artar ve sol ventrikül hipertrofisi, mikroalbüminüri, karotid intima-media kalınlığı ve endotel disfonksiyonu ile ilişkilidir. Karotis-femoral (aortik) nabız dalga hızı merkezi arterial sertliğin değerlendirilmesinde altın standartken, artırma göstergesi sistemik arterial ağacın sertliğini yansıtır. Bu yazıda arteriyel sertliğin değerlendirilmesinde nabız dalga hızı ve artırma indeksinin avantajları ve dezavantajları özetlenmiştir.

Anahtar Kelimeler: Nabız; arterler; ateroskleroz; enflamasyon

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The arterial tree can be divided into central and peripheral compartments. Brachial pressure represents blood pressure in the peripheral compartment, while aortic (or carotid) pressure represents blood pressure in the central compartment. Systolic blood pressure and pulse pressure (the difference between the systolic and diastolic pressure) are notably higher in peripheral than in central arteries. This difference results from a difference in the summation of the forward and backward pressure waves along the arterial tree. If the usual resting pulse pressure is consistently greater than the normal value, the most likely basis is stiffness of the major arteries.[1] Assesment of arterial stiffness is increasingly used in clinical settings as the role of arterial stiffness in the pathogenesis of cardiovascular diseases ensues in the recent years.[1-15] Although there are several methods currently available to assess arterial stiffness, researchers and clinicians still face problems in selecting the best methodology for their specific use. This paper summeries the advantages and disadvantages of pulse wave velocity (PWV) and augmentation index (AI) in the assessment of arterial stiffness.

PULSE WAVE VELOCITY

PWV is a technique in which large artery elasticity is assessed by the analysis of the peripheral arterial waveform.[1] PWV was determined over both the trunk and the lower limb. Continuous pulse-pressure wave signals were recorded with two sensors positioned at both the base of the right common carotid artery and over the femoral artery. This technique measured especially the stiffness of the descending aorta.[1] Blood pressure and heart rate are known determinants of measured arterial PWV.[1]

Left ventricle contraction generates a pulse wave which propagates throughout the arterial tree, reflecting segmental arterial elasticity. The distance traveled by the pulse wave divided by the time taken to travel the distance gives the PWV [PWV (m/s)= distance (m)/transit time (s)].[1] As the arterial wall stiffness, waves in the lumen travel at a higher velocity. PWV can easily be measured along two accessible arterial segments.[1] Its unit is meters/second and it is closely associated with the arterial segment measured. The viscoelastic properties of the vessel wall and the blood density as shown by the Moens-Korteweg equation and the Bramwell-Hill equation determine PWV.[16-18] Theoretically, the wave velocity (C0), in a thin-walled, uniform, elastic vessel containing an incompressible, inviscous fluid, with no reflections, can be expressed by the Moens-Korteweg equation (16,17): $C0= \sqrt{Eh/2\rho R}$ (E: Young's modulus of elasticity, h: wall thickness, R: mean radius, ρ: blood density). Following Bramwell and Hill, equation of Moens-Korteweg[16,17] also can be expressed as $C0= \sqrt{dP\ V/dV\ \rho}$ (P: pressure, V: volume of tube per unit lenght, dV/VdP: volume compliance of the tube).[18] In this equation, the square of the wave velocity is associated with the inverse of the volume compliance that represents the total arterial stiffness. Since heart rate and blood pressure influence PWV, the measurement has to be adjusted according to these variables.[1] PWV is an easy and reproducible method for clinical practice.[1-15] Assessment of central arterial stiffness is more appropriate for the risk stratification of cardiovascular diseases rather than peripheral arterial stiffness.[1] Carotid-femoral PWV is the gold standard test for assessing central arterial stiffness;[1-15] however, it is valuable under experienced hands, so its applicability is mostly limited to research institutes.[19] Brachial-ankle PWV measurement is another method that is simple enough to use in clinical practice, as it only involves wrapping a pressure cuff around each of the four extremities.[20] It reflects muscular arterial stiffness besides elastic arterial stiffness.[20] Although the usefulness of the brachial-ankle PWV was demonstrated in some small-scale studies,[21-23] and further studies are needed to confirm the usefulness of the brachial-ankle PWV, the brachial-ankle PWV shows close correlation with aortic PWV.[20] Aging (decreases elastin fiber, increases collagenous material and causes loss of arterial elasticity), high blood pressure (increases at high blood pressure and decreases at low blood pressure) and other atherogenic factors cause vascular injury, which in turn triggers structural stiffening of the arteries.[1] Moreover, increased blood pressure is associated with functional stiffening of the arteries by causing increased arterial wall tension.

AUGMENTATION INDEX AND CENTRAL BLOOD PRESSURE

The carotid artery and/or radial artery blood pressure waveforms used for determining total systemic artery compliance were analyzed to identify the shoulder and peak of the waves.[24] The augmentation point was reliably identified mathematically as the first zero-crossing from positive to negative, after the beginning of systole, of the third derivative of the pressure waveform. The AI reflects stiffness of the systemic arterial tree.[24] It is defined as Dp/pp (where Dp is the difference between the late systolic peak and the mid-systolic peak and pp is the amplitude of the pulse pressure wave), and it allows the estimation of the effect of the reflected wave on the amplitude of the pulse pressure wave. Macrovascular functions and microvascular functions affect the AI. Increased central arterial stiffness is associated with an increase in the propagation velocity of waves and a shift of the reflection point to proximal sites in the arterial tree, with augmentation of the interaction of the incident and reflected pulse waves. Carotid artery AI and radial artery AI are the two available methods used in the clinical settings to measure AI and central blood pressure could be calculated from them.[25] Central pressures are pathophysiologically more relevant than peripheral pressures in the pathogenesis of cardiovascular diseases.[26] Although carotid artery AI derived central blood pressure is reliable, there is still controversy on the validation of the central blood pressure estimated from the radial artery.[25,27] However, radial AI measurement and central blood pressure estimated from the radial AI are easy to perform and are used in a number of large scale studies.[28,29] Increased stiffness in the arterial tree is suggested to enhance the traveling speed of the incident pulse wave and to shift the reflection point to a proximal site in the arterial tree.[30]

WHICH METHOD IS THE BEST?

Central blood pressure and pulse pressure, the AI and PWV, which are often misused as interchangeable indexes of arterial stiffness, increase with age, hypertension, diabetes mellitus, chronic renal failure, chronic inflammation and hypercholesterolemia and are associated with target organ damage (left ventricular hypertrophy, microalbuminuria, carotid intima-media thickness, and endothelial dysfunction) and clinical outcomes. Velocity of the pulse wave, the amplitude of the reflected wave, the reflectance point and the duration and pattern of the left ventricle ejection are major determinants of central blood pressure, central pulse pressure, and the AI with respect to change in heart rate and ventricular contractility,[31] whereas aortic PWV, which is defined as the velocity of the arterial pulse for moving along vessel wall, represents intrinsic arterial stiffness, according to the Bramwell-Hill Formula.[18] By influencing reflecting wave, heart rate and ventricular ejection, pathophysiological conditions and drugs may change central pulse pressure and the AI without changing aortic PWV. The AI is much more sensitive to the effects of heart rate than aortic PWV.[32,33] Finally, although the carotid-femoral PWV is the gold standard test for assessing central arterial stiffness, the AI reflects stiffness of the systemic arterial tree.

In conclusion, although various arterial parameters can be used for the non-invasive assessment of arterial stiffness and wave reflections, carotid-femoral (aortic) PWV is the gold standard for the assesment of arterial stiffness,[26] as it has the largest amount of epidemiological evidence for its predictive value for cardiovascular events and requires technical studies.

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