Hypertension

Letter to the Editor: Response to Arterial Stiffness Index Is Not a Stiffness Parameter But a Ventriculo-Arterial Coupling Factor

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Letter to the Editor

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Response to Arterial Stiffness Index Is Not a Stiffness Parameter But a Ventriculo-Arterial Coupling Factor

We defined the ambulatory arterial stiffness index (AASI) as unity minus the regression slope of the brachial diastolic on systolic blood pressure.[1,2] AASI, therefore, models the dynamic relation between diastolic and systolic pressure throughout the day. We concur with Westerhof et al[3] that AASI is an indirect measure of arterial stiffness. Several hemodynamic factors influence AASI, including ventriculo-arterial coupling. In this regard, AASI does not differ from other measures of arterial stiffness, including pulse wave velocity, which many experts consider as the gold standard.

To further assess the physiological meaning of AASI, we partially implemented the proposal of Westerhof et al.[3] We computed in our 348 Chinese subjects[1] the decay time of aortic pressure during diastole (τ). We rewrote τ as [(60×mean arterial pressure)/(heart rate×pulse pressure)]. We averaged the τ values obtained from the blood pressure readings in each 24-hour ambulatory recording. Across our study sample,[1] τ was normally distributed (Shapiro–Wilk's W, 0.995; $P=0.34$), averaging (SD) 1.84 (0.29) seconds. We found an inverse association ($r=-0.21$; $P<0.0001$) between τ and AASI (Figure), which strengthens the concept that AASI is a measure of arterial stiffness. In the final formula, Westerhof et al[3] defined the slope of diastolic on systolic blood pressure as the ratio of diastolic to systolic blood pressure and, therefore, assumed an intercept of 0. We did not force the regression line through the origin,[1,2] because during diastole, when blood flow drops to 0, this is not the case for blood pressure.

The discussion on what AASI stands for should not detract attention from its prognostic significance. To date, 1 cross-sectional analysis[4] and 3 prospective cohort studies[2,5,6] demonstrated association of AASI either with signs of target organ damage in never-treated hypertensive patients[4] or with the incidence of cardiovascular mortality and morbidity.[2,5,6] AASI is particularly predictive of stroke,[2,5,6] even at levels of blood pressure within the normotensive range.[2,6] When adjusted for pulse pressure, AASI retained its predictive value.[2,5,6] Currently ongoing analyses of the Copenhagen cohort showed that AASI predicts stroke over and beyond aortic pulse wave velocity.

Because AASI reflects more than just arterial stiffness in the narrow sense of the word, some experts proposed a name change. AASI should not belie its name. The rationale for a name change, in that AASI reflects more than just arterial stiffness, is equally applicable to most other measures of arterial function, including pulse wave velocity.

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None.

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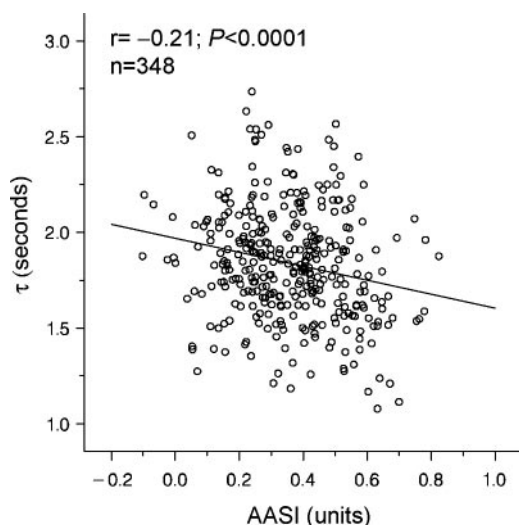*Copenhagen, Denmark*

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Relation between the decay time of aortic pressure during diastole (τ) and the AASI in 348 Chinese subjects.

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**Letter to the Editor: Ambulatory Arterial Stiffness Index Is Not a Stiffness
Parameter But a Ventriculo-Arterial Coupling Factor**

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Ambulatory Arterial Stiffness Index Is Not a Stiffness Parameter But a Ventriculo-Arterial Coupling Factor

To the Editor:

Recent hypertension research has shown that large artery compliance is an important determinant of systolic pressure, pulse pressure, and cardiovascular disease. Many methods exist to determine compliance or its inverse, arterial stiffness; the time constant of the aortic pressure decay in diastole, the ratio of stroke volume over pulse pressure, and pulse wave velocity are among the most used. These methods require either invasive measurements or 2 simultaneous measurements and are not practical to use in epidemiological studies or in night–day variations. Dolan et al[1] recently suggested the use of the Ambulatory Arterial Stiffness Index (AASI), defined as 1 minus the slope of the (linear) relation between diastolic and systolic pressure, as a measure of arterial stiffness. The AASI is easy to measure noninvasively and over long time periods. The AASI, although associated with pulse pressure, augmentation index, and other measures of arterial stiffness, was criticized by Laurent,[2] Benetos and Lacolley,[3] and Gavish[4] and defended by Dolan et al.[1] Thus, the question of whether AASI is a proper arterial stiffness parameter has not been answered.

Here we derive the AASI from basic principles. The ratio of stroke volume, SV, over pulse pressure, PP, is a measure of total arterial compliance: $C=SV/PP$, or $PP=SV/C$. The ratio of mean pressure, P_m, and cardiac output, Q, is a measure of systemic vascular resistance, R. With SV times heart rate, HR, being cardiac output, it follows that $P_m=Q\times R=SV\times HR\times R$ or $SV\times R/T$, with T heart period (R−R interval). Thus, $PP/P_m=T/RC=T/\tau$ with $\tau=RC$, the characteristic decay time of aortic pressure in diastole.

We approximate mean pressure by $P_m=(P_s+2P_d)/3$, and pulse pressure is $PP=P_s-P_d$, with P_s and P_d systolic and diastolic pressure. Inserting this into PP/P_m, we obtain the following: $(P_s-P_d)/(P_s/3+2P_d/3)=T/\tau$; rearrangement gives the following: $P_d=P_s\,(3-T/\tau)/(3+2T/\tau)$. Thus, 1 minus the slope of the relation between diastolic and systolic pressure equals the following:

$$AASI=1-(3-T/\tau)/(3+2T/\tau)=(T/\tau)/(1+2T/3\tau)$$

Taking $\tau=1.5s$ and $T=0.86s$ (70 bpm), we obtain $AASI=0.41$ (experimental data 0.33 to 0.56[1]). A stiffer arterial system, that is, decreased compliance and, thus, smaller τ, results in an increased AASI. If we assume that compliance depends on pressure, the ratio T/τ appears again in the formula, together with pressure, and, thus, the AASI depends not only on total arterial compliance or its inverse, arterial stiffness, but also systemic vascular resistance, heart period, and on pressure.

T is the characteristic time determined by the heart, and τ is the characteristic time of the arterial system.[5] Therefore, the AASI depends on both the heart and the arterial system. The ratio T/τ is a (temporal) ventriculo-arterial coupling factor; it has been shown that T/τ is similar in mammals at rest, resulting in similar systolic and diastolic pressures in mammals.[5] Thus, the AASI is a coupling factor as well.

Increased arterial stiffness (with R, T, and pressure constant) results in an increase in both AASI and pulse pressure, and, therefore, AASI correlates with indicators of arterial stiffness but is not a measure of arterial stiffness: it is a measure of ventriculo-arterial coupling.

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