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COMMENTARY

Ambulatory arterial stiffness index: a marker of subclinical organ damage in treated and untreated dipper hypertensive patients

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Cardiovascular risk assessment is a fundamental step in the management of hypertensive patients. It is not difficult to determine the degree of hypertension and the presence of co-existing risk factors, but detecting subclinical organ damage (SOD) is more complicated, and its success depends on the diagnostic test chosen.

Large-artery stiffness is recognized as an indicator of SOD in hypertensive patients and as an important predictor of cardiovascular morbidity and mortality in subjects with essential hypertension.[1] Although systemic arterial stiffness can only be estimated from models of circulation, regional and local arterial stiffness can be measured directly at various sites along the arterial tree. One major problem with arterial stiffness determination is that accurate measurement of a given artery's stiffness necessarily requires simultaneous, accurate measurements of the local arterial cross-sectional area (or diameter) and pressure, which are difficult to obtain *in vivo*. Thus, in a compromise between simplicity and accuracy, indexes of arterial stiffness are used as surrogates of the 'true' stiffness. A number of surrogate measures of large-artery stiffness have been proposed, but each has important limitations.[2,3] The dynamic relationship between systolic and diastolic blood pressure (BP) provides a means of investigating arterial stiffness. For a given increase in diastolic BP, systolic BP is expected to increase to a limited extent in a compliant artery, whereas the increase will be greater in a stiff artery. The opposite holds for an increase in diastolic BP for a given increase in systolic BP; this relationship can be considered a measure of arterial compliance.

These principles are the basis of the ambulatory arterial stiffness index (AASI).[4] Individual values of systolic and diastolic BP measurements obtained through 24-h non-invasive ambulatory BP monitoring are plotted, and the linear regression slope of diastolic BP *vs.* systolic BP is assumed to be a global measure of arterial compliance. The complement of the slope (1 minus the slope)—the AASI—has been accepted as a measure of arterial stiffness.[4]

The AASI is associated with preclinical target organ damage in subjects with hypertension[5,6] and with an increased risk of cardiovascular mortality in hypertensive patients.[7] Its use does not require specialized equipment or dedicated personnel, and it is expected to be widely applied in the scientific and medical communities.

The AASI is not appropriate for use in non-dipper hypertensive patients. Dipper subjects have nocturnal systolic and diastolic BP values that are much lower than the corresponding daytime values, resulting in an increase in the regression coefficient of diastolic BP *vs.* systolic BP. Non-dipper subjects, on the other hand, tend to have a narrower range of diastolic BP values over a 24-h period. As consequence, for non-dipper subjects the correlation coefficient of the regression of diastolic *vs.* systolic BP over 24 h tends to decrease and that of its complement, the AASI, tends to increase.[8]

Ángel García-García *et al.*[9] found that an increased AASI was associated with the presence of vascular, cardiac and renal SOD in primary hypertensive patients, regardless of whether they were taking blood-pressure-lowering drugs. In our opinion, these results are relevant because the AASI was calculated in a population of dippers independent of the use of blood-pressure-lowering drug treatment. In conclusion, we believe that before calculating the AASI in a hypertensive population, it is important to take into account that there might be false-positive results for non-dipper subjects.

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