Aortic stiffness as a predictor of coronary atherosclerosis

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In recent years, interest on arterial structure and function has increased exponentially, mainly because increased arterial stiffness is associated with, and independently predictive of, an increased cardiovascular morbidity and mortality in a variety of high-risk conditions (e.g. renal insufficiency, hypertension, diabetes and the elderly state) [1–5]. Concomitantly, this has also favoured the discovery of a variety of devices that have made it progressively easier to measure arterial stiffness in a quantitatively accurate non-invasive fashion, meaning that this assessment is available to a larger number of patients than was previously possible. Assessment of arterial stiffness is of special interest for coronary heart disease because, when large artery distensibility is reduced, there is: (i) an increase in systolic blood pressure, arterial impedance and left ventricular load; (ii) a reduction in diastolic blood pressure and thus of coronary perfusion; (iii) an acceleration (due to a greater traumatic effect of intravascular pressure on the vessel wall) of the cascade of events leading to the appearance and progression of atherosclerosis; and (iv) a reduced change in vessel volume in response to blood pressure changes and thus an impaired sensitivity of the baroreflex with a deranged neural cardiac control (increased sympathetic and reduced vagal tone) potentially dangerous for cardiac function, perfusion and rhythm [6–8]. On clinical grounds, this has received confirmation from studies demonstrating a positive relationship between the degree of arterial stiffness and a patient's ergometric performance, as well as prevalence and incidence of coronary events [9–11].

In this issue of the journal, van Popele *et al.* [12] measured aortic stiffness by pulse wave velocity from the carotid to the femoral artery whereas coronary atherosclerosis was assessed by quantifying, through electron beam tomography, the amount of coronary calcium that was expressed as total calcium score. In an unselected elderly population, there was a clear relationship between these two variables, even when data were adjusted for potential confounders (i.e. for other possible contributors to coronary atherosclerosis). This provides further evidence that arterial stiffness is associated with coronary vascular abnormalities, adding to previous data indicating that this is also the case in a non-selected elderly population.

The above results allow several considerations to be made. First, it should be emphasized that the study by van Popele *et al.* [12] has several merits, including the very large number of patients, their unbiased selection and the accuracy of the method employed to quantify, albeit in an indirect fashion, arterial stiffness. This places on solid ground the clinical implications of the results (i.e. that measuring aortic distensibility by a non-invasive device now in rather large use may provide physicians with a further element in favour of the possible presence of coronary disease and thus the need to proceed with further examinations). However, two considerations also need to be made. First, although of prognostic significance [13], the amount of calcium in the coronary vessels is not strictly specific for atherosclerosis, and its deposition is related to the ageing process to some extent. Second, as discussed by the authors [12], an association does not mean a cause–effect relationship, a simple explanation for the data being that the presence of coronary plaques is more likely to be accompanied by plaques elsewhere in the large artery tree, given that plaques have a direct stiffening effect on arteries [14].

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