

## Brachial artery diameter enlargement: a new marker of atherosclerosis?

M. Amato<sup>1</sup>, D. Sansaro<sup>1</sup>, A.L. Ravani<sup>1</sup>, B. Frigerio<sup>1</sup>, S. Castelnovo<sup>2</sup>, F. Veglia<sup>1</sup>, E. Tremoli<sup>1,3</sup>, D. Baldassarre<sup>1,3</sup>

<sup>1</sup> Centro Cardiologico Monzino IRCCS, Milan, Italy

<sup>2</sup> Centro Dislipidemie E. Grossi Paoletti, Ospedale Ca' Granda di Niguarda, Milan, Italy

<sup>3</sup> Dipartimento di Scienze Farmacologiche e Biomolecolari, Università di Milano, Milan, Italy

During the atherogenic process, the arterial diameter measured in plaque free areas tends to enlarge. This enlargement does not reflect the process defined 'vascular remodeling', which occurs primarily as a local response to the rheological changes induced by the presence of atherosclerotic plaques, but rather it occurs as simple compensatory response of arteries to the presence of atherosclerosis risk factors. Several studies suggested this arterial enlargement as a further surrogate marker of atherosclerosis. In a recent study (Baldassarre 2012), we have shown that the addition of the inter-adventitia common carotid artery diameter (ICCAD) measured in plaque-free areas to algorithms for the assessment of global cardiovascular risk improves the patient's risk stratification. However, carotid arteries are rarely free of atherosclerotic lesions, especially in adult or elderly subjects, and even if the measures are taken in plaque free areas, it can not be excluded the presence of plaques in the surroundings which might alter the vessel rheology, thus being the indirect responsible of the enlargement observed. Some studies have recently evaluated the diameter of other arterial districts known to be less prone to the development of atherosclerosis lesions. Most of these studies, focused on the brachial artery diameter (BAD), indicate that the arterial enlargement is a generalized phenomenon, and suggest that, as carotid diameter, also the enlargement of this arterial district may be useful to further improve the prediction of vascular events. All these studies, however, have been carried out in relatively small samples. In addition, limited information is available regarding the determinants of the enlargements evaluated simultaneously in different vascular districts.

**Aim of the study:** To validate, in a large sample, the role of BAD as an independent marker of atherosclerosis and to investigate whether the addition of BAD measurements to ICCAD measurements may actually offer additional information for the definition of patients' cardiovascular risk profile.

**Methods:** 4641 patients (44.6% women and 55.4% men; age (mean±SD) 58±13 and 55±13, respectively) have their BAD, ICCAD and carotid Intima media thickness (C-IMT) measured by B-Mode ultrasound. Measurements have been taken during the first visit at the Centro Dislipidemie E. Grossi Paoletti, (Ospedale Ca' Granda di Niguarda) or at the Centro Cardiologico Monzino, IRCCS. Both BAD and ICCAD were measured in plaque free areas. A total of 4271 subjects were asymptomatic, whereas 335 (64 women and 271 men) experienced a myocardial infarction and 35 (11 women and 24 men) a stroke.

**Results:** BAD was associated with the prevalence of vascular events in both women and men. After adjustment for age, traditional risk factors, C-IMT and ICCAD, this associations persisted in women (O.R and CI: 2.2 [1.1-4.4]; p<0.05) but not in men (O.R and CI: 1.1 [0.8-1.7]; p=ns). When the analysis was performed considering myocardial infarction and stroke separately, it becomes clear that the observed significant association was mainly due to association with myocardial infarction (O.R and CI: 2.6 [1.2-5.6]; p<0.05). BAD was closely associated with ICCAD (Beta of about 0.30±0.03; P<0.0001, in both sexes). Despite this, determinants of the enlargement of the two vascular districts were very different. For example, the relationship between BAD and the Framingham risk score was two times lower than that observed with ICCAD).

**Conclusions** The BAD is a independent marker of myocardial infarction, which, at least in women, may provide information which is complementary to that coming from vascular risk factors and ICCAD.

Baldassarre (2012). *J Am Coll Cardiol.*;60:1489-99