Editorial

Arterial hypertension is defined by a stable increase in systemic arterial blood pressure (BP) values, i.e. systolic value of 140 mmHg or more and/or diastolic one of 90 mmHg or more. Its prevalence is about 30–45% of the general population; representing a well-known cardiovascular (CV) risk factor [1]. In addition to BP values, the assessment of target organ damage has a pivotal role in stratification of total CV risk of patients. Current guidelines for the management of arterial hypertension suggest several tools for evaluating hypertension-related asymptomatic organ damage, such as electrocardiography, echocardiography, vascular ultrasound examination (carotid wall related asymptomatic organ damage, such as electrocardiography, arterial hypertension suggest several tools for evaluating hypertension-of total CV risk of patients. Current guidelines for the management of arterial hypertension suggest several tools for evaluating hypertension-related asymptomatic organ damage, such as electrocardiography, echocardiography, vascular ultrasound examination (carotid wall related asymptomatic organ damage, such as electrocardiography, arterial hypertension suggest several tools for evaluating hypertension-related asymptomatic organ damage, such as electrocardiography, echocardiography, vascular ultrasound examination (carotid wall related asymptomatic organ damage, such as electrocardiography, arterial hypertension suggest several tools for evaluating hypertension-related asymptomatic organ damage, such as electrocardiography, echocardiography, vascular ultrasound examination (carotid wall related asymptomatic organ damage, such as electrocardiography, arterial hypertension suggest several tools for evaluating hypertension-related asymptomatic organ damage, such as electrocardiography, echocardiography, vascular 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causes its dysfunction and promotes atherosclerotic process [20]. In male subjects elevated systolic BP in adolescence can predict the reduction of FMD 21 years later independently of other CV risk factors [21]. These studies suggest that hypertension may cause endothelial dysfunction, but since both conditions share some pathogenetic mechanisms, it is interesting to highlight whether the impaired endothelial function precedes the development of hypertension or vice versa.

Some evidences support the role of endothelial dysfunction in promoting the onset of arterial hypertension. In offspring of essential hypertensive patients a reduced vasorelaxant response to acetylcholine was found compared with that of offspring of normotensive subjects [22]. In a cohort of 952 healthy postmenopausal women with normal BP levels an increase in the risk of developing arterial hypertension, correlated with the grade of endothelial dysfunction, was demonstrated, independently of age and baseline pressure values. In particular, a 16% increase in the relative risk of hypertension for each unit decrease of FMD was found [23]. However, a study carried out in 3500 participants from the Multi-Ethnic Study of Atherosclerosis did not show a role of endothelial dysfunction assessed with FMD as independent risk factor for hypertension [24]. Although results are conflicting and this issue remains open, these data suggest that endothelial dysfunction is a consequence of arterial hypertension rather than a primary abnormality. Further studies are needed in order to better understand this relationship.

In the future, the knowledge of exact mechanisms underlying such a linkage may lead to new insights, i.e. in the drug development, offering to patients more effective treatments for management of arterial hypertension. As an early marker of atherosclerosis it is desirable that the assessment of endothelial function may be widely used by physicians as screening tool to identify subjects with asymptomatic and initial arterial damage at higher CV risk. Finally, future studies aimed to highlight whether the recovery of endothelial function is associated with better prognosis of hypertensive patients are warranted.

References
