Blood vessels are exposed to two kinds of dynamic mechanical forces. One of them is shear stress and the other is the cyclic strain of the vascular wall, which, according to the Laplace's law, is mainly determined by cyclic change of blood pressure (BP). While shear stress directly affects endothelial cells, BP changes (and the resulting changes in arterial wall strain) influence all structures of the arterial wall. Arteries are permanently exposed to a basal stretch, which is related to mean BP, and to a pulsatile stretch owing to pulse pressure (PP). Cyclic changes in the intramural tension have recently been recognized as an important factor in the pathogenesis of atherosclerosis and acute coronary syndromes. The study by Khoueiry et al. fits well into list of recent research investigating the relation between BP, arterial stiffness, and atherosclerosis.

Nowadays, there is no doubt about the presence of significant relation between the pulsatile BP component and atherosclerosis, but these links are in fact bidirectional. On one hand, diffused atherosclerotic plaques impair the elastic properties of the arterial wall (although unstable, soft, and lipid-rich plaques do not impair arterial compliance), whereas on the other hand, an increased stiffness enhances the pulsatile component of BP, leading to the progression of atherosclerotic alterations. Taken together, these relations are responsible for a vicious circle. It should be, however, emphasized that low values of PP are required for the development of a differentiated and fully functional phenotype of vascular wall cells, as well as for the regulation of migratory properties, proliferation, and matrix turnover.

A number of studies have shown the correlation between central PP and the extent of coronary atherosclerosis. In the population studied by Khoueiry et al., aortic PP was higher in subjects with right coronary artery (RCA) stenosis compared to those with stenosis in the left coronary artery (LCA). This could be due to the difference in blood flow pattern between LCA and RCA (as suggested by Khoueiry et al.) or earlier (at lower level of BP) atherosclerotic plaque development in LCA compared to RCA. Although the former explanation cannot be fully excluded due to cross-sectional nature of the study it should be underscored that systolic, diastolic, and mean pressure were similar in the study groups as was the mean number of antihypertensive drugs used per one participant (unfortunately the doses of drugs are unknown). Another elucidation could be stiffer aorta in RCA diseased patients as opposed to subjects with stenosed LCA, which, however, could not be explained on the basis of the current knowledge. Although the authors performed multivariate analysis the reader should remember that the study groups differed in respect of several important variables, including heart rate, which in turn is one of major determinants of PP. The selection bias or an unrecognized difference in the studied groups should be also taken into account. Several phenomena could induce bias. The most important is that both high PP as well as stenosis in left main or left anterior descending artery (as compared to stenosis in RCA) are related to higher cardiovascular risk. Therefore, it is possible that some subjects with high PP and stenosis in LCA could not be included in the study because they had died earlier. In this respect, it should be underscored that blood flow through LCA depends on diastolic pressure which is low in subjects with high PP.

Laminar flow in the arteries and high shear stress are believed to protect against atherosclerosis development. One could argue that the present results at least partly disagree with this conception. Indeed, as the blood flow is more pulsatile in LCA than in RCA one could expect rather closer relation between PP and the extent of atherosclerosis in LCA than in RCA. However, one should not forget that change in BP not always leads to change in blood flow. Indeed, increase in systolic pressure leads to increased systolic blood flow in RCA, but its effect on blood flow in LCA is diminished by the parallel increase in systolic pressure in left ventricle. Moreover, it seems that changes in flow patterns can be more important than the flow patterns themselves in producing potentially deleterious effects on vascular biology. The present study has shown that PP may be higher in patients with stenosed RCA as compared to those with stenosed LCA, which could suggest that the difference in hemodynamic conditions in RCA and LCA may influence the atherosclerosis development. These results, if confirmed in prospective studies using more precise methods of the atherosclerosis extent assessment, may have deep influence on not only understanding of the factors influencing atherosclerosis development but also might potentially have clinical implications.

Disclosure: The authors declared no conflict of interest.