Response to Central Pulse Pressure: Is It Really an Independent Predictor of Cardiovascular Risk?

We thank Kopec and Podolec1 for their interest in our recent study. They correctly noticed that aortic stiffness is one of the major determinants of central pulse pressure. However, they asked if central pulse pressure “only reflects” aortic stiffening or “adds additional cardiovascular risk” independently of aortic stiffness. Well, it is known that a pulsatile component of blood pressure (BP) takes part in the pathogenesis of atherosclerosis, as well as in the pathogenesis of acute coronary syndromes.2 It could be even considered an indispensable factor to the atherosclerosis development. Indeed, in our article we were able to show that central BP cyclic changes are better predictors of cardiovascular events (the majority of which are complications of atherosclerosis) than left ventricular ejection fraction, revascularization procedures, and the extent of coronary atherosclerosis, diabetes, or renal disease. On the other hand, aortic stiffness just reflects the “disease” of arterial wall. The only known connection (other than the probable correlation between the disease of aortic and coronary artery walls) between the stiffness of the aortic wall and progression (or rupture) of an atherosclerotic plaque in a coronary artery is the pulsatile component of BP.

Another important finding of our study was that cyclic, relative changes of central BP are at least as important as absolute changes. Indeed, this confirms that cyclic tensile stress of the arterial wall, irrespectively of the absolute value of BP, is one of the most important factors in the development of atherosclerosis and its complications.2

Finally, taking all we know into account, it is reasonable to conclude that central BP measurements are very useful tools both in research and in clinical practice. In this regard, it should not be forgotten that the most important factors influencing the height of the pressure wave are stroke volume and the speed of blood flow from the left ventricle to the ascending aorta. This explains why intra-aortic pressures are correlated with the extent of coronary atherosclerosis in patients with preserved but not in those with impaired left ventricle systolic function.3,4 The pathophysiological basis for this phenomenon can be found in the literature.4–6 The conclusion was drawn from these studies that subjects with low and normal left ventricular ejection fraction should be investigated separately when he BP waveform-coronary atherosclerosis relationship is analyzed. Otherwise, the correlation between pulse pressure and coronary atherosclerosis is artificially diminished.

Disclosures

None.

Piotr Jankowski
Kalina Kawecka-Jaszcz
Department of Cardiology and Hypertension
Jagiellonian University, Medical College
Kraków, Poland


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Piotr Jankowski and Kalina Kawecka-Jaszcz

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