To the editor:

The study by Horvath et al [1] presents a very good correlation (n=22, R=0.91) between invasively measured aortic PWV on the one hand, and PWV obtained by the Arteriograph on the other hand. In this paper, the authors refer to our previous work [2] as follows:

‘The importance of the stop flow, caused by the occlusion, to detect a clear, pronounced late systolic wave ... was proven by Segers et al. by a mathematical circulatory model.’

‘Segers et al. validated the operation principle of the Arteriograph (completely occluded brachial artery, stop flow) by an elegant mathematical model. They found that the time interval between the early and late systolic peaks, used by the Arteriograph to determine aortic PWV, shows a very strong (R²=0.9739) linear correlation with the change of the aortic stiffness, namely with the true aortic PWV.

The authors may, however, have missed our follow-up study [3] in which we used the same model to demonstrate that the aforementioned correlation between Arteriograph and true aortic PWV is driven by changes in brachial artery stiffness. Changing the stiffness of all non-brachial arterial model segments did not affect the time interval between early and late systolic peaks in any way, whereas changing only brachial artery stiffness did have a significant effect (while aortic properties had remained the same). The only possible conclusion from these observations is that the parameter provided by the Arteriograph is a measure for brachial stiffness alone. We therefore hypothesize that the relatively good correlations observed in the study of Horvath et al, like the previously reported correlations between the Arteriograph and other non-invasive devices [4-6], are due to the fact that brachial and aortic PWV are intrinsically connected to each other [7]. We do acknowledge that this hypothesis is based on the results of a mathematical model and should therefore be confirmed by an invasive validation. Unfortunately, in real life it is not as trivial to selectively change brachial and aortic stiffness independently of each other as in a computational model.

Using wave intensity analysis, we also provided a physical explanation as to why the Arteriograph is only sensitive to brachial stiffness: our data suggest that the measured wave in stop-flow conditions is predominantly the result of waves traveling back and forth in the brachial artery, reflected distally on the occluded cuff, and proximally on the open-end reflection of the aortic junction. In an editorial comment published in the same edition of the journal [8], this hypothesis was termed highly speculative. Although we admit that the waves observed in wave intensity analysis can be subject to interpretation, they are simply the result of physical laws imposed in our model, and should therefore not be dismissed without providing an alternative explanation.

In summary, we remain skeptical towards data measured by the Arteriograph. If the authors really want to prove the working principle of the Arteriograph, we suggest them to measure high-fidelity invasive pressures and flows in both the proximal and distal brachial artery and aorta, both in control and brachial stop flow conditions. This should allow to study the actual wave reflections in the arteries of interest and will hopefully allow deeper insight into what the Arteriograph is actually measuring.


