Effects of Heart Rate Changes on Arterial Distensibility in Humans

To the Editor:

Dependence of arterial stiffness on heart rate, claimed by Giannattasio et al., runs counter to classic studies, which were previously discussed in relation to conflicting data obtained with one method used to measure aortic pulse wave velocity. There is a potential flaw in the method applied by Giannattasio et al in their determination of carotid and radial artery distensibility at different heart rates. They measured diameter change of the target artery, but pressure pulsation at a distal site (brachial pressure for carotid, finger pressure for radial artery). Errors inherent in distal pressure measurement have been stressed in a recent consensus document but were considered by authors to be minimal. We disagree. In similar studies by Wilkinson et al., there was an average 35% fall in central pulse pressure, compared with brachial, when heart rate was increased from 60 to 110/min by pacing. Giannattasio et al quoted an early evaluation of the Finapres system and considered this accurate for their purposes, but this study did not test response to change in heart rate. In a later manuscript by the developers of Finapres, a marked heart rate difference was noted for systolic pressure between noninvasive finger and brachial intraarterial pressure (20 mm Hg difference for heart rate change of 40 bpm). The differences in distensibility calculated by Giannattasio et al were at the margin of statistical significance (P<0.05). Given the problems in estimating pulse pressure at the site of diameter measurement and the likelihood that this was overestimated, we continue to rely on the previous work, which showed no significant dependence of arterial stiffness on heart rate.

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Response

We have discussed the issue raised by Michel O’Rourke and Audrey Adji concerning our paper in which we outlined several arguments in favor of the conclusion that the difference between the blood pressure and arterial distensibility measuring sites did not detract from the observation that an increase in heart rate was accompanied by arterial stiffening. We wish to further emphasize the following, however. (1) Increasing heart rate reduced not only distensibility in the carotid but also in the radial artery, i.e., a vessel close to the finger where blood pressure was measured. We have previously shown that finger blood pressure measure-ments correspond to measurements made intraarterially from the contralateral radial artery at rest, over 24 hours, and during a variety of laboratory maneuvers that lead to marked heart rate changes. (2) We have shown an increase in heart rate by pacing to cause carotid artery stiffening in rats, in which blood pressure was measured intraarterially on the contralateral vessel at the same level where diameter changes were assessed. (3) In the paper quoted by O’Rourke and Adji as classical, the conclusion that an increase in heart rate leads to an increased arterial distensibility was based on indirect data, i.e., on the fact that a heart rate increase was accompanied by a reduction in aortic pressure, which, in turn, was indirectly estimated by the augmentation index. We doubt this approach to be superior to the more direct measurements we have used.

Finally, other studies have examined the relationship between heart rate and arterial distensibility in acute and chronic conditions and have also concluded that in humans an increase in the former has a stiffening effect on the latter. Interestingly, we have seen a heart rate-related effect on carotid artery distensibility also in sympathectomized rats that led us to suggest that the viscous property of the vessel wall may be involved. That is, that viscosity of the medium makes vessel distension dependant on time, leading to an “operative” stiffening when time is reduced, as when heart rate increases.

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