Central Aortic Pressure Influences Pulse
Wave Velocity

To the Editor:

The study reported by Lantelme and colleagues\(^1\) suggests that increasing heart rate is independently associated with higher aortic pulse wave velocity (PWV). We are concerned that the authors of this study have neither addressed the impact of central aortic pressure changes on PWV nor the presence of potential bias in their study.

In Lantelme’s study, PWV represented the average wave speed from the carotid artery, through the aorta, and down to the common femoral artery. Local wave speed varies between different arteries and along the length of the aorta,\(^2\) and, furthermore, may alter in response to changes in local pressure.\(^3\) Knowledge of central aortic pressure is therefore an essential requirement for the interpretation of studies using PWV.

Central aortic pressure is influenced by left ventricular ejection and the phenomenon of wave reflection.\(^3\) Systolic pressure in peripheral muscular arteries tends to be higher than in those measured centrally,\(^4\) particularly in younger subjects, and amplification of the central pressure pulse toward the periphery may be influenced by a number of factors, including heart rate.\(^5–7\) Previous work using similarly aged subjects\(^5,6\) suggested that, over a similar range of heart-pacing rates, there was a significant increase in both diastolic and mean central pressures. Only one study measured PWV,\(^5\) where it was noted to increase nonsignificantly with increasing heart rate. There is also evidence that in younger subjects increased heart-pacing rates are associated with elevation of central diastolic blood pressure.\(^7,8\) It is possible therefore that central pressures, and particularly diastolic blood pressure, which were not controlled for in Lantelme’s study, contributed to the change in PWV seen with different pacing rates.

In this small group of elderly subjects, Lantelme et al found no significant changes in brachial pressures over paced heart rates of 60 to 100 bpm. In contrast, however, the 2 studies\(^5,6\) that recruited subjects of similar age to those in Lantelme’s group showed that both brachial systolic and diastolic pressures increased significantly over a range of physiological heart rates. A potential contributing factor to this important difference between the studies is the method used to measure brachial blood pressure. Lantelme’s use of a mercury sphygmomanometer exposes the results to significant operator bias. On the other hand, the use of a validated semiautomated blood pressure monitor, as used in the other studies, removes this problem and may explain the differences in peripheral blood pressure observed in these studies.

There is further evidence for potential bias in this study supported by the data presented as a Bland–Altman plot. There is a discernible trend for the difference between the 2 measurements of PWV to increase with increasing values of PWV. This is further supported by the correlation data, where the slope of the regression line is approximately 0.7.

Although epidemiological and observational data have suggested that heart rate is an independent determinant of PWV, few conclusions can be made from Lantelme’s study because of potential methodological bias and the failure to address the effects of central arterial pressure.

Response: Pulse Wave Velocity, Heart Rate, and Blood Pressure

In their letter, Zambanini et al raised concerns about the scope and the interpretation of our recently published results concerning the effect of heart rate (HR) on pulse wave velocity (PWV).\(^1\) First of all, we would like to emphasize the fact that our study sought to assess the value of PWV from a clinical angle. In this respect, its robustness was tested in the presence of a common hemodynamic variation, that is, HR change.

The major concern of Zambanini et al is the lack of brachial blood pressure (BP) variations during cardiac pacing at variance with 2 previously published studies.\(^2,3\) Brachial BP was measured with a mercury sphygmomanometer, which may represent a source of bias in case of systematic under- or overestimation.

However, this would not necessarily prevent the disclosure of a relationship between BP and HR. A likely hypothesis relies on the effect of HR on cardiac output, which is a major determinant of systolic BP. This effect might have been more negative in our study\(^1\) than in the 2 others\(^2,3\) because of differences in age or in the mode of stimulation. Contrary to what is indicated in their letter, age was clearly different in our group of patients (77.8 years) when compared with that in the aforementioned studies\(^2,3\) (69 and 63 years, respectively). Being older, our subjects likely had a greater prevalence of cardiac dysfunction. It has been shown that increasing HR leads to a decreased cardiac output in
patients with cardiomyopathy. Therefore, cardiac index probably decreased more with pacing in our study than in the 2 others. Regarding the mode of stimulation, all pacemakers but 2 were set in the VVI mode. It has been shown that lengthening atrioventricular delay was an important way to increase cardiac output, especially in dilated cardiomyopathy. Having used a different mode of stimulation, ie, atrial or atrioventricular pacing, probably helped maintain a better cardiac output during HR increases.

Central aortic BP was not evaluated in our study, but a rise was very unlikely because of the stable brachial systolic BP. Accordingly, in Wilkinson’s study, it was shown that central systolic BP remained stable during pacing, whereas central diastolic BP paralleled brachial diastolic BP. Even if an increase of central diastolic BP cannot be thoroughly ruled out, it is very unlikely in our experimental conditions.

Finally, the authors raised concerns about the reproducibility of our measurements. This deserves several comments. Firstly, our reproducibility was found to be very close to the values reported in the literature. Secondly, our reading of the Bland-Altman plot is that there was no relation between the value of PWV and the difference between the 2 measurements, except for one outlier.

As a conclusion, we believe that the merit of our experiment is to question the robustness of PWV for clinical use. We conclude that HR has a significant effect on PWV. The magnitude of this effect has, however, to be put in balance with the changes that have a significant impact on cardiovascular risk.

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*Hypertension*. 2002;40:e10-e11; originally published online October 28, 2002;
doi: 10.1161/01.HYP.0000041883.60004.7B
*Hypertension* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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