Effects of Ventricular Premature Stimulus Coupling Interval on Blood Pressure and Heart Rate Turbulence

To the Editor:

The recent article by Watanabe et al.1 addresses the important issue of heart rate turbulence (HRT) as a potential noninvasive index for mortality after myocardial infarction. In attempts to investigate underlying mechanisms, the study contains a potentially serious flaw that should be considered by others who might wish to pursue the subject. The authors investigated effects of baroreceptors on HRT by use of measurements of arterial pressure by intravascular catheters. They do not describe the site of measurement, but if it is a peripheral location (e.g., radial or brachial artery), the large change in heart rate (40 to 150 beat/min) used in the study can produce substantial errors in using the peripheral pulse as a surrogate of central (carotid or aortic) pulse. Wilkinson et al.2 have shown that such a change in heart rate can increase the amplification of the arterial pulse almost 2-fold, such that at higher heart rate, peripheral systolic pressure is grossly exaggerated. This is important in baroreflex assessment, because baroreceptors respond to central and not peripheral systolic pressure.3 With maneuvers such as those applied by Watanabe et al.,1 it is desirable that some correction be made4 so that the baroreflex mechanisms of heart rate change associated with a premature ventricular contraction can be elucidated with precision.

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Response

We appreciate the interest shown by Drs Avolio and O’Rourke in our recent article1 on the relationship between ventricular premature coupling (VPC) interval and heart rate turbulence. They make the important point that pulse pressure measured in the periphery can be much greater than that measured near the heart. We used 5 to 8 Fr arterial sheaths 12 cm long inserted into the femoral artery just below the inguinal ligament. The tip of the sheath would have extended to the common iliac artery in most patients, a fairly central location.

For arguments’ sake, we consider whether our conclusions would have differed had our blood pressure measurements been made from a peripheral location. The amplitude transfer function given in the article by Avolio et al.2 indicates that pulse pressure amplification is minimal (1.0) at frequencies lower than 1 Hz (VV interval of 1000 ms), whereas the amplification smoothly increases to 1.4 at 2 Hz (VV interval of 500 ms). In the example we gave in Figure 4, dividing our systolic or pulse pressure values by the amplification factor would have resulted in a steeper slope relating blood pressure value to VPC interval (division of blood pressure at shortest VV interval by 1.4, longest interval by 1), with no change being seen for the relation of blood pressure to compensatory pause interval. For patients with faster baseline heart rate, blood pressure after the compensatory pause would have been affected by a factor close to 1, resulting in a small increase of slope value. Therefore, our published conclusion drawn from the qualitative aspects of behavior such as those seen in Figure 4 would have been unaffected.

Finally, transfer functions relating central to peripheral pressures have not been validated for large beat-to-beat RR interval change like that seen with a VPC. Although transfer functions may be a rigid function determined by the structure of the arterial tree and unaffected by stroke volume, this hypothesis should be validated so that central pressures can be derived with confidence in extreme cases, such as transient arrhythmias. It is not self-evident to us that a transfer function validated at a steady state heart rate of 120 should apply equally to a low-pressure wave produced at a VPC interval of 500 ms (heart rate of 120) in a patient with baseline heart rate of 60.

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