Postural influences on the mechanical and neural components of the cardiac baroreflex

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The cardiac baroreflex is a fundamental mechanism of blood pressure (BP) regulation. It has been suggested that quantification of baroreflex gain in healthy subjects may reflect only the state of the mechanical component, (*i.e.* barosensory vessel stretch in response to changes in BP), found to be influenced by posture (Saeed *et al*., 2009). However, these conclusions are based upon spontaneous methods of baroreflex assessment, the accuracy of which has been questioned (Lipman *et al*., 2003). Closed-loop approaches do not allow the investigator to determine the input and output signals of the physiological response, and studies have shown that much of the variability can be explained by respiratory sinus arrhythmia (Lipman *et al*., 2003; Tzeng *et al*., 2009). Therefore, the aim of this study was to actively perturb BP in order to engage a more open-loop approach with which to explore the influence of posture on the mechanical and neural components of the baroreflex.

In 9 participants (5 male, mean ± SD age 25.0 ± 3.7y) we measured continuous BP, R-R intervals and carotid artery diameter during intravenous bolus injections of sodium nitroprusside (SNP) followed by phenylephrine (PE) (modified Oxford method) during supine and standing postures. This technique quantifies baroreflex gain for falling BP *via* SNP (G-down) and rising BP *via* PE (G-up). Integrated gain was determined by plotting beat-to-beat R-R intervals against systolic BP. The mechanical component was diameter plotted against systolic BP, and the neural component was R-R intervals plotted against carotid artery diameter. Linear mixed models were employed to compare the integrated, mechanical and neural gains between supine and standing postures.

In response to rising pressures, there was an attenuated ($P<0.05$) baroreflex gain in the standing position (G-up ± SE = 5.1 ± 0.4 ms/mm Hg) compared with supine (G-up = 12.8 ± 0.5 ms/mm Hg). The attenuation was explained by a diminished ($P<0.05$) neural gain whilst standing (G-up = 264.9 ± 25.7 ms/mm) compared with supine (G-up = 617.5 ± 44.6 ms/mm). This neural response to a change in posture was consistent in all participants. There was no significant difference in mechanical gain between the two postures in response to rising pressures ($P=0.19$). Baroreflex gain in response to falling pressures was not significantly different between standing (G-down = 6.2 ± 0.3 ms/mm Hg) and supine (G-down = 6.4 ± 0.4 ms/mm Hg), with no significant effects of posture on either the mechanical or neural component ($P>0.05$). Analysis using heart rate, in place of R-R interval, lead to the same results for G-up. However, a significant increase was found in neural gain for G-down ($P<0.05$), although this increase was not observed in all participants.

Our findings indicate that when responding to rising pressures baroreflex gain is reduced in the standing position. In contrast to the study by Saeed *et al*., (2009) our results suggest that the reduction in baroreflex gain associated with an upright posture is explained by the neural component, and therefore may be due to parasympathetic withdrawal resulting in reduced vagal nerve activity with which to regulate heart rate.

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Saeed NP, Reneman RS, Hoeks AP. (2009) *Journal of Vascular Research* **46:** 469-77.
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