

## POSTER 62

**SELECTIVE ATTENUATION OF CAROTID-CARDIAC RESPONSES TO HYPERTENSION AT THE ONSET OF STATIC HANDGRIP IN HUMANS**

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Previous studies have indicated that at the onset of exercise cardiac baroreflex function is reduced in an intensity-dependent manner, which appears to be mediated by a blunted ability to buffer hypertensive challenges. However, whether cardiac baroreflex responses to a hypotensive stimulus are altered at exercise onset is unclear. To examine this, ten subjects ( $25 \pm 1$  years) performed multiple 1-min bouts of static handgrip (HG) at 15 and 60% of maximal voluntary contraction (MVC), while breathing to a metronome set at eupneic frequency. Neck pressure (NP +40 Torr) or neck suction (NS -60 Torr) was applied for 5 s at end expiration, to simulate carotid baroreflex (CBR) hypotension and hypertension, respectively at rest, at the onset of HG ( $< 1$  s), and at  $\sim 40$  s of HG. Cardiac responses to NP at the onset of exercise were maintained at both HG intensities, whereas heart rate responses to NS were blunted at exercise onset during 60% MVC HG ( $-16 \pm 2$  rest vs.  $-9 \pm 3$  bpm 60% MVC;  $P < 0.05$ ) but preserved at 15% MVC. The response range of the carotid-cardiac baroreflex at the onset of 60% MVC was reduced ( $25 \pm 1$  rest vs.  $18 \pm 3$  bpm 60% MVC;  $P < 0.05$ ) compared to rest and 15% MVC. Heart rate responses to NP and NS applied at  $\sim 40$  s of 15 and 60% MVC were similar to rest. These results suggest that carotid-cardiac responses to hypertension are selectively contributing to an impaired CBR control of heart rate at the onset of static HG in humans.

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