Aspirin augments carotid-cardiac baroreflex sensitivity during muscle mechanoreflex and metaboreflex activation in humans.

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Abstract
Muscle mechanoreflex activation decreases the sensitivity of carotid baroreflex (CBR)-heart rate (HR) control during local metabolite accumulation in humans. However, the contribution of thromboxane A2 (TXA2) toward this response is unknown. Therefore, the effect of inhibiting TXA2 production via low-dose aspirin on CBR-HR sensitivity during muscle mechanoreflex and metaboreflex activation in humans was examined. Twelve young subjects performed two trials during two visits, preceded by 7 days' low-dose aspirin (81 mg) or placebo. One trial involved 3-min passive calf stretch (mechanoreflex) during 7.5-min limb circulatory occlusion (CO). In another trial, CO was preceded by 1.5 min of 70% maximal voluntary contraction isometric calf exercise to accumulate metabolites during CO and stretch (mechanoreflex and metaboreflex). HR (ECG) and mean arterial pressure (Finometer) were recorded. CBR function was assessed using rapid neck pressures ranging from +40 to -80 mmHg. Aspirin significantly decreased baseline thromboxane B2 production by 84 ± 4% (P < 0.05) but did not affect 6-keto prostaglandin F1α. Following aspirin, stretch with metabolite accumulation significantly augmented maximal gain (GMAX) and operating point gain (GOP) of CBR-HR (GMAX; -0.71 ± 0.14 vs. -0.37 ± 0.08 and GOP; -0.69 ± 0.13 vs. -0.35 ± 0.12 beats·min(-1)·mmHg(-1) for aspirin and placebo, respectively; P < 0.05). CBR-HR function curves were reset similarly with aspirin and placebo during stretch with metabolite accumulation. In conclusion, these findings suggest that low-dose aspirin augments CBR-HR sensitivity during concurrent muscle mechanoreflex and metaboreflex activation in humans. This increased sensitivity appears linked to reduced TXA2 production, which likely plays a role in metabolite sensitization of muscle mechanoreceptors.

KEYWORDS: aspirin, baroreflex, mechanoreflex

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