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Cardiovascular Effects of Cadence and Workload.

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Increases in cadence may augment SV during submaximal cycling (> 65 % V.O (2max)) via effects of increased muscle pump activity on preload. At lower workloads (45 - 65 % V.O (2max)), SV tends to plateau, suggesting that effects of increases in cadence on pump activity have little influence on SV. We hypothesized that cadence-induced increases in CO at submaximal workloads, where SV tends to plateau, are due to elevations in HR and/or O₂ extraction. SV, CO, HR, V.O₂, and Deltaa - vO₂ were assessed at 80 and 100 rpm during workloads of 50 % (LO) or 65 % (HI) of V.O₂ (2max) in 11 male cyclists. No changes in SV were seen. CO was higher at 100 rpm in 10 of 11 subjects at LO (18.1 +/- 2.7 vs. 17.2 +/- 2.6 L/min). V.O₂ at both workloads was greater at 100 than 80 rpm as was HR (LO: 129 +/- 11 vs. 121 +/- 10 beats/min; HI: 146 +/- 13 vs. 139 +/- 14 beats/min) (p < 0.05). Deltaa - vO₂ was greater at HI compared to LO at 80 (15.1 +/- 1.6 vs. 13.6 +/- 1.3 ml) and 100 rpm (16.0 +/- 1.7 vs. 15.1 +/- 1.6 ml) (p < 0.05). Results suggest that increases in O₂ demand during low submaximal cycling (50 % V.O (2max)) at high cadences are met by HR-induced increases in CO. At higher workloads (65 % V.O (2max)), inability of higher cadences to increase CO and O₂ delivery is offset by greater O₂ extraction.