

Oxygen pulse and heart rate recovery at maximal exercise are blunted in adults born preterm in normoxia and hypoxia

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INTRODUCTION: Preterm birth affects millions of infants each year, and most survivors go on to lead active lives, participating in sports and other athletic events without incident. However, recent evidence shows that there are some long term effects of preterm birth including right ventricular hypertrophy, reduced right ventricular ejection fractions, and lower blood pH during graded exercise. Heart rate recovery after maximal exercise has been used as a predictor of cardiovascular disease and mortality, and is known to be associated with negative prognostic indications in pulmonary arterial hypertension. Oxygen pulse (ml O₂ consumed/heart beat) has been shown to be a mirror of stroke volume (SV) during incremental exercise, and a blunted O₂ pulse has been shown to be reflective of an impaired left ventricular SV, ischemic heart disease and congestive heart failure, and a predictor of mortality in subjects with and without cardiopulmonary disease.

MATERIALS AND METHODS: Adults born preterm (preterms) and age-matched term-born controls (controls) subjects performed incremental exercise on a cycle ergometer to volitional exhaustion while breathing normoxic air (21% oxygen), followed by a second maximal exercise test in hypoxic air (12% oxygen) after a 45 minute rest. HR was recorded throughout exercise and every 10 seconds for two minutes of stationary recovery after each maximal exercise test. HRR, HR as a percentage of HR reserve, maximal oxygen consumption (VO_{2max}) and maximal O₂ pulse were compared using two-way ANOVA for each group and condition.

RESULTS: O₂ pulse was lower in preterms compared to controls in both normoxia and hypoxia (preterms: 13.4±2.1 and 9.1±1.8 ml/beat and controls: 19.9±4.6 and 15.5±3.2 ml/beat), and was lower in each group in hypoxia compared to normoxia (p<0.05 for all). Preterms showed significantly lower HRR in the first minute after maximal exercise in both normoxia and hypoxia compared to controls (preterms: 17.7±4.1 vs. 27.5±3.7 bpm and controls: 15.3±0.9 vs. 24.8±3.2 bpm, respectively, p<0.05 for all). Additionally, preterms recovered a lower percentage of their heart rate reserve at 60 seconds of recovery after maximal exercise compared to controls in both normoxia and hypoxia (preterms: 15.4±3.0 vs. 28.0±2.4% and controls: 23.5±2.4 vs. 34.0±2.3%, respectively, p<0.05 for all). HRR in hypoxia and normoxia were not significantly different in preterms at 60 seconds of recovery, though it was lower in controls in hypoxia. VO_{2max} was lower in hypoxia compared to normoxia in both groups, and was lower in preterms compared to controls in both normoxia and hypoxia (controls: 34.3±7.1 and 44.7±7.9 ml/kg/min and preterms: 23.9±7.3 and 35.3±6.2 ml/kg/min, respectively, p<0.05 for all).

CONCLUSIONS: O₂ pulse was blunted in preterms compared to controls in both normoxia and hypoxia at maximal exercise. HRR both absolutely and as a percentage of heart rate reserve is blunted in preterms after maximal cycling exercise in normoxia. As both O₂ pulse and HRR have been correlated with mortality risk and other cardiopulmonary diseases, this data suggests that the adult premature population has long term health consequences that go beyond diminished lung function.