Effect of prolonged exercise in a hypoxic environment on cardiac function and cardiac troponin T

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METHODS

Eight trained male volunteers completed the study (mean (SD) age 33.5 (8.8) years, height 1.79 (0.08) m, body mass 77.7 (8.3) kg. VO2max, 67.4 (6.3) ml/kg/min). After ethical approval from the universities’ ethics committees and before the start of the study, each subject provided written informed consent. The subjects completed two 50 mile cycle trials on a Kingcycle training rig (Kingcycle, High Wycombe, Buckinghamshire, UK), randomly assigned from normobaric normoxia and normobaric hypoxia (15% FIO2). Echocardiographic assessment and whole blood collection was completed before, immediately after, and 24 hours after exercise. Left ventricular systolic and diastolic functional variables were calculated, and serum was analysed for cardiac troponin T. Results were analysed using a two way repeated measures analysis of variance, with α set at 0.05.

RESULTS

No significant differences were observed in either systolic or diastolic function across time or between trials. Cardiac troponin T was detected in one subject immediately after exercise in the normobaric hypoxic trial. A 50 mile cycle trial in either normobaric normoxia or normobaric hypoxia does not induce exercise induced cardiac fatigue. Some people, however, may exhibit minimal cardiac damage after exercise in normobaric hypoxia. The clinical significance of this is yet to be elucidated.

Exercise induced cardiac fatigue (EICF) has recently been described by many authors as has minimal cardiac damage after prolonged exercise. Further, it has been suggested that acute altitude exposure may exacerbate the incidence of EICF because of the increased physiological strain associated with exercising at altitude. Stimulated by the increased participation in endurance events at moderate to high altitude and the adoption by many athletes of hypoxic trial was completed in a commercially available Kingcycle training rig (Kingcycle, High Wycombe, UK), randomly assigned from normobaric normoxia and normobaric hypoxia (15% FIO2). Echocardiographic assessment and whole blood collection was completed before, immediately after, and 24 hours after exercise. Left ventricular systolic and diastolic functional variables were calculated, and serum was analysed for cardiac troponin T. Results were analysed using a two way repeated measures analysis of variance, with α set at 0.05.

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Methods:

Eleven trained male triathletes volunteered for the study. Each completed two 50 mile cycle trials, randomly assigned from normobaric normoxia and normobaric hypoxia (15% FIO2). Echocardiographic assessment and whole blood collection was completed before, immediately after, and 24 hours after exercise. Left ventricular systolic and diastolic functional variables were calculated, and serum was analysed for cardiac troponin T. Results were analysed using a two way repeated measures analysis of variance, with α set at 0.05.

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Abbreviations: cTnT, cardiac troponin T; EICF, exercise induced cardiac fatigue; FS, fractional shortening; Q, cardiac output; SV, stroke volume
Cardiac function after exercise in a hypoxic environment

The results of this study suggest that 50 miles of cycling at an intensity equivalent to lactate threshold in either normobaric normoxia or normobaric hypoxia does not induce reductions in either left ventricular systolic or diastolic function. The impact of altered heart rates on serial measurements of diastolic function has been debated.15,16 In the present study, minimal release of cTnT after prolonged exercise has been shown in a limited number of subjects in previous studies.5,11,20–22 The rapid return to baseline cTnT in the one positive subject in our study coupled with the minimal concentration attained may suggest a transient cytosolic leakage propagated by membrane damage, as opposed to cardiomyocyte necrosis.23,24 It is possible that such cytosolic leakage may be caused by free radical mediated injury,11 and as such may explain why the cTnT release in this study was only observed in the normobaric hypoxic trial where free radical production would be increased.25 Currently, however, any suggestions of the potential mechanisms responsible for such cTnT release remain speculative. It is noteworthy that exercised induced troponin release in this study if a less well trained subject pool had been used. At present, however, the precise mechanisms for and clinical significance of minimal cTnT release after prolonged exercise cannot be elucidated.

CONCLUSIONS

A 50 mile cycle trial at lactate threshold in either normobaric normoxia or normobaric hypoxia does not induce cardiac dysfunction or evidence of cardiac damage in most subjects. Some, however, may show evidence of minimal cardiac damage. Further work is warranted into the factors that may interact to induce minimal cardiac damage in certain people.

Take home message

Two hours of vigorous exercise in either a normobaric hypoxic or normobaric normoxic environment in trained subjects does not produce exercise induced cardiac fatigue. Minimal cTnT release may, however, be observed in some subjects, the long term implications of which are yet to be elucidated.
REFERENCES