T2 recovery unable to distinguish anaerobic exercise from aerobic

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Purpose: To evaluate T2 recovery rate following anaerobic skeletal muscle exercise using Wingate cycling.

Introduction: Short-time high-intensity exercise is known to induce marked metabolic effects in contracting skeletal muscles. Anaerobic metabolism is especially present during a short supramaximal exercise, such as Wingate test (1). The high rate of ATP production required for such an exercise is derived primarily from anaerobic sources such as PCr and glycogen breakdown to lactate. Anaerobic exercise can therefore be characterized by high intramuscular lactate production that exceeds lactate elimination rate with very high muscle H^+ and lactate levels. The monoexponential transverse relaxation time (T2) of skeletal muscle is known to increase linearly by exercise, and suggested to be mostly related to increased water content and mechanisms related to relative aerobic capacity of the muscle, e.g. net accumulation of osmoles (2). It is not settled to what degree factors derived from anaerobic metabolism, such as lactate and H^+, would contribute to the altered T2. However, accumulation of tissue lactate is closely related to accumulation of extravascular water and theoretically an effect on T2 could be possible.

Materials and Methods: 10 healthy volunteers were studied prior to and immediately after a 30-second Wingate test and repeatedly during 25-min of recovery. Power output and venous lactate were measured as well as mean blood flow velocity in right common femoral artery by ultrasound. Cross-sectional area (CSA) of the right thigh and T2 from both vastus lateral muscles were calculated using a 1.5 T magnet and a multiple spin echo sequence (TR/TE 1500/15-30-45-60 ms), exemplified by pre- and post-exercise images at TE 60 ms in Figures 1 and 2. All values are presented as mean±SE.

Results: Peak power value (W/kg) was 10.8±0.3 with a power decrease of 46.3±3.7 %. The lactate recovery curve (Fig. 3) showed peak level of 12.8 mmol/l obtained 6 min after cessation of exercise. The mean blood flow velocity in common femoral artery increased 8 times from 13.9±3.5 cm/s to 100.1±21.1 cm/s (Fig. 4), with still elevated velocities by 2.6 times the resting value at the end of recovery. The cross-sectional area of the right thigh increased by 5.1±1.0 % from 210±34 cm^2 to 220±32 cm^2 with a very slow recovery as shown in Fig 3, still elevated 23 min after exercise by 3.9±1.0 %. T2 increased from 25.2±1.2 ms by 16.5±1.4 % as shown in Fig. 3 with a continuous decline throughout recovery. Calculated half-time of CSA recovery was 57.7 min and of T2 recovery 11.4 min.

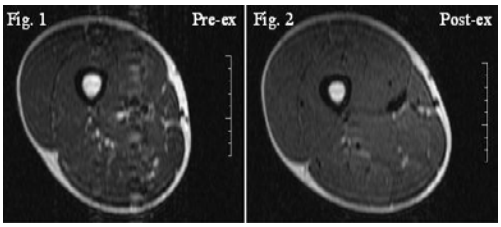


Fig. 3

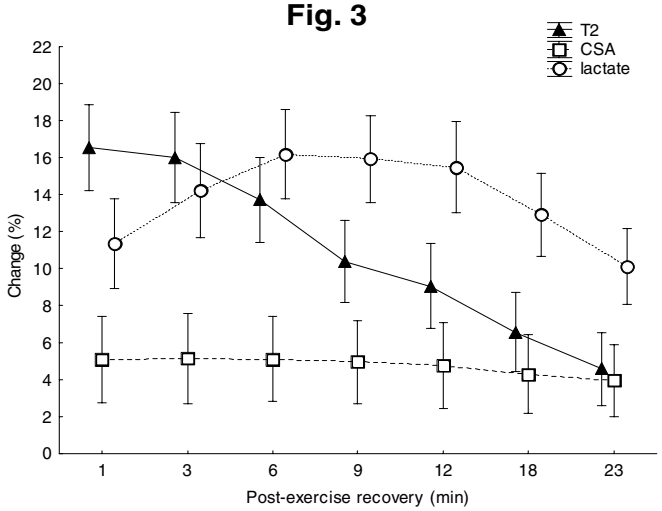
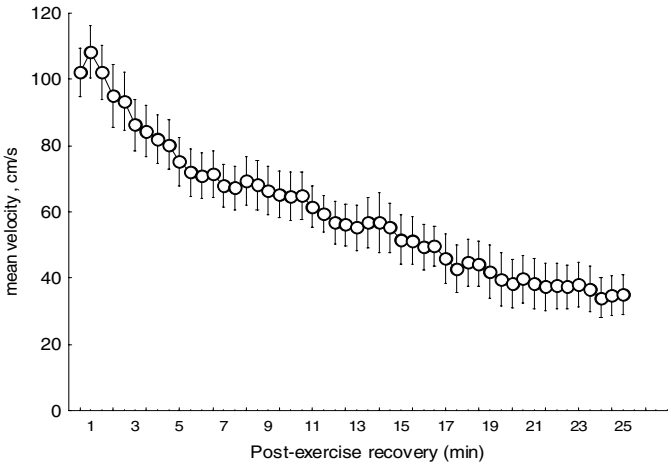


Fig. 4



References: 1) Beneke et al. EJAP 2002; 87: 388-392. **2)** Reid et al. JAP 2001;90: 897-902. **3)** Hussain et al. Exp Physiol 1996; 81: 173-187.

Conclusion: A deviant response of T2 and CSA recovery is found, with slow CSA and blood flow recovery after wingate, contrasting a previously shown parallel recovery of T2 and CSA after high-intensity endurance exercise which also goes with a more rapid blood flow recovery (3). Both the maximal T2 level and its recovery after wingate are comparable with recovery after high-intensity endurance exercise. Consequently, the measured T2 recovery seems unable to distinguish the pronounced anaerobically driven metabolism and vaso-active metabolites accumulated during the anaerobic Wingate test.