Muscle fatigue during intermittent exercise in individuals with mental retardation

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1. Introduction

Strength and muscular fatigue are integral components of physical fitness and overall health. Fatigue originates in peripheral and central cites. Peripheral fatigue has been linked to absolute strength (Nordlund, Thorstensson, & Cresswell, 2004), substrate depletion, and/or metabolite accumulation in muscles (St Clair Gibson, Lambert, & Noakes, 2001) while the contribution of the central nervous system to fatigue depends on neural processes that govern voluntary activation of a muscle (Enoka & Stuart, 1992; Gandevia, 1998, 2001). Indeed, it has been documented that individuals with lower levels of strength, lower glycolytic capacity, lower ability of maximal voluntary activation, as well as lower voluntary activation of motor units during exercise demonstrate lower fatigue during sustained high-intensity muscular contraction (Enoka & Stuart, 1992; Hicks, Kent-Braun, & Ditor, 2001; Nordlund et al., 2004; Strekis, Skurvydas, & Ratevicius, 2007; Yamada, Kaneko, & Masuda, 2002).

Numerous studies have shown that sedentary adults and elite athletes with mental retardation demonstrate lower strength measures compared to individuals without mental retardation (Croce, Pitetti, Horvat, & Miller, 1996; Horvat, Croce, Pitetti, & Fernhall, 1999; Horvat, Pitetti, & Croce, 1997; van de Vliet et al., 2006). There is also evidence that individuals with mental retardation may have disturbed central and peripheral processing components as indicated by the longer reaction times.
and motor times (Davis, Sparrow, & Ward, 1991; LeClair, Pollock, & Elliot, 1993); and the associations of intelligence and/or general mental ability with peripheral (Tan, 1996) and brain (Reed, Vernon, & Johnson, 2004) nerve conduction velocities and with neural transmission (McRorie & Cooper, 2003, 2004). Finally, individuals with mental retardation demonstrate damage in the integrity of white matter tracts that are responsible for the processing and control of sensory and motor information controlling arousal and motor function (Yu et al., 2008) that may affect motoneuronal recruitment and movement control during sustained effort. Thus, the lower strength, the impaired central nervous system, and their association with fatigue suggest that fatigue profile during exercise may be different between individuals with and without mental retardation.

While several studies examined muscular fatigue during exercise in individuals with typical development (Clark, Collier, Manini, & Ploutz-Snyder, 2005; Hicks et al., 2001; Hunter, Griffith, Schlachter, & Kufahl, 2009; Kamehisa, Okuyama, Ikegawa, & Fukunaga, 1996; Pincivero, Gandaio, & Ito, 2003; Russ, Towse, Wigmore, Lanza, & Kent-Braun, 2008; Wüst, Morse, de Haan, Jones, & Degens, 2008), information on fatigue profile during intermittent exercise in adults with mental retardation is lacking. Muscular fatigue that occurs during intermittent exercise is a characteristic of many exercise training programs and sports. Exercise training has essential implications for individuals with mental disabilities since they demonstrate low physical fitness that has been directly related to their productivity in the work settings (Fernhall, 1993). Furthermore, in the last years individuals with mental retardation are regularly involved in intensive training to achieve a high level of physical fitness in order to participate in athletic competitions. Thus, the knowledge on the ability of the muscles to resist fatigue in individuals with mental retardation should be of great interest to rehabilitation and sport professionals because it provides a more complete profile of their neuromuscular function. In addition, understanding the ability to repeat exercise bouts during intermittent exercise (fatigue resistance) in these individuals is essential for planning and designing exercise programs for recreational and social purposes, as well as for the development of motor skills and physical fitness in rehabilitation and sport settings.

Therefore, the aim of this study was to examine the fatigue profile during intermittent exercise in individuals with mental retardation vs. that in individuals without mental retardation. Furthermore, we investigated the ability of individuals with mental retardation to produce work during short-term continuous muscular effort (anaerobic capacity).

2. Methods

2.1. Participants

Ten healthy men with typical development and 10 healthy individuals with mental retardation participated in the study. All volunteers engaged in recreational physical activities three times per week. The institutional review board approved the experimental protocol and the participants with typical development provided written consent; the informed consent for the individuals with mental retardation was provided by their parents or legal guardians. The IQ of participants with mental retardation was determined by a Weschler Intelligence Scale test. Participants with mental retardation had intelligence quotients (IQ) that was within a range for individuals with mild-moderate mental retardation (51 ± 5).

2.2. Study design

Each participant reported to the exercise laboratory in the morning of the testing. Following orientation and assessment of physical characteristics, they performed an intermittent exercise protocol that involved 4 sets of 30 s (18 maximal flexions and extensions of the knee joint), with a 60 s rest interval between sets. Peak torque of knee flexors (PTFL) and extensors (PTEX), and total work (TW) produced were measured at each set and fatigue was calculated. Pre-motor time was measured before the fatiguing protocol and blood lactate concentrations were measured at rest and 5 min after the completion of the exercise protocol. The participants were asked to abstain from exercise activity for 48 h before the study, to have sufficient rest the night prior the experiment, and to refrain from caffeine ingestion the day of the experiment.

2.3. Testing procedures and instrumentation

Upon arrival to the laboratory height and body weight (Seca, Hamburg, Germany) were measured. Following a 10-min rest, a blood sample (5 µL) was obtained from the fingertip for the determination of baseline lactate concentration (LACTATE PRO, Akray, Japan). Next, the participants underwent a 5-min warm up on a bicycle ergometer at a heart rate of 120–130 bpm, and 5 min of stretching exercises. Following warm-up, the skin was shaved and cleaned with alcohol and bipolar surface electrodes (Model SS2, Biopac Systems, Inc., Goleta, CA, bipolar silver/silver chloride electrodes, center-to-center interelectrode distance = 2 cm) were applied to the belly of vastus medialis and on the patella for bipolar EMG recording (MP100, Biopac System, Inc.) in order to measure pre-motor time. The electrodes were interfaced to a portable amplifier/transmitter (Model TEL100M, Biopac Systems, Inc., Goleta, CA, Common mode rejection ratio > 110 db at 50/60 Hz, bandwidth = 10–500 Hz; gain = 1000). The unit was interfaced to a Biopac MP100 Data Acquisition unit (Biopac Systems, Inc., Goleta, CA) and converted into digital form at a rate of 1000 Hz. Pressing the PC mouse a lamp was turned on and at the same time a signal was given to initiate the EMG recording. The participants were asked to perform a knee extension movement as fast as possible after the visual signal to assess pre-motor time. Pre-motor time (ms) was defined as a period between the onset of a visual stimulus and the beginning of muscle activity as recorded by change in EMG (Horvat, Ramsey, Amestoy, & Croce, 2003).
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