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The effect of selective β1-blockade on EMG signal characteristics during progressive endurance exercise

Abstract This study analysed the effect of selective β1-blockade on neuromuscular recruitment characteristics during progressive endurance exercise. Ten healthy subjects ingested a selective β1-blocker, acebutolol (200 mg b.d.), for 7 days (for one of two cycling trials), with a 10-day wash-out period between trials. On the last day of acebutolol ingestion subjects performed three successive 15-min rides at 30%, 50% and 70% of their peak power output and then cycled at increasing (15 W min⁻¹) work rates to exhaustion. Force output, heart rate, submaximal \( \dot{V}O_2 \), rate of perceived exertion (RPE), electromyographic (EMG) data and blood lactate were captured during the cycling activity. Peak work rate \([270 (111) \text{ W vs } 197 (75) \text{ W, CON vs BETA, } P < 0.01]\), time to exhaustion \([49.7 (23.2) \text{ min vs } 40.3 (23.7) \text{ min, CON vs BETA, } P < 0.05]\) and heart rate \([\text{mean, for the full ride 135.5 (38.3) beats min}^{-1} \text{ vs } 111.5 (30.0) \text{ beats min}^{-1} \text{ CON vs BETA, } P < 0.05]\) were significantly lower for the group who ingested β1-blockade (BETA) compared to the control group (CON). Although not significant, submaximal \( \dot{V}O_2 \) was reduced in BETA during the ride, while RPE was significantly higher during the ride for BETA \((P < 0.01)\). Mean integrated electromyography was higher in the BETA group although these differences were not significant. Mean power frequency values of the BETA group showed a significant \((P < 0.05)\) shift to the upper end of the spectrum in comparison to the control group. Lactate values \([11.7 (3.5) \text{ mmol.l}^{-1} \text{ vs } 7.1 (4.1) \text{ mmol.l}^{-1} \text{ CON vs BETA}]\) were significantly lower \((P < 0.05)\) at exhaustion in BETA. Significant reductions in cycling performance were found when subjects ingested β1-blockers. This study has shown significant shifts to the upper end of the EMG frequency spectrum after β1-blocker ingestion, which could be caused by a change in neuromuscular recruitment strategy to compensate for the impaired submaximal exercise performance.

Keywords β1-Blockade · Fatigue · Integrated electromyography · Mean power frequency spectrum

Introduction

Individuals receiving β-adrenergic receptor blocking drugs often complain of muscle fatigue. Both non-selective β1- + β2-blockers and selective β1-blockers reduce most individuals' capacity to exercise (Hughson 1989; van Baak et al. 1987; Verstappen and van Baak 1987). β-Blockade either increases (Broberg et al. 1988; Macdonald et al. 1984) or has no effect on plasma lactate accumulation during exercise (Hughson 1989; Lundborg et al. 1981; van Baak et al. 1987), and it does not increase muscle glycogenolysis (Clerox et al. 1989; Kaiser et al. 1986). Plasma glucose concentration during exercise is either decreased (Lundborg et al. 1981; Macdonald et al. 1984; van Baak and Mooij 1994; Verstappen and van Baak 1987) or unaffected by β-blockade (Gullestad et al. 1989; Laustiola et al. 1983). Only the mobilisation of non-esterified fatty acids (NEFA) is consistently decreased by β-blockade (Lusti et al. 1983; Lundborg et al. 1981; Uusitupa et al. 1982; van Baak et al. 1987, 1993). However, whether lower plasma glucose or NEFA concentration is involved in the reduced exercise endurance capacity during β-blockade is open to question. Intravenous infusion studies have shown that reversal of the decreases in circulating glucose and NEFA concentrations with β-blockade do not improve exercise capacity (van Baak et al. 1993; van Baak and Mooij 1994).
Since changes in metabolism do not readily explain the impaired sub-maximal exercise capacity during β-blockade, other potential causes of early fatigue have to be considered. One possibility is that β-blockade may affect muscle recruitment activity from the central pathways of control. In cycle exercise the integrated electromyographic (IEMG) activity is known to increase with increased exercise intensity (Bigland-Ritchie and Woods 1974). During continual heavy work at a constant load, an increase in IEMG is observed and interpreted as a sign of muscle contraction failure (Lippold et al. 1960). More information detailing the motor unit action potentials can be gained by studying the mean power frequency spectrum (MPFS).

To the authors’ knowledge, the only experiment that has been conducted to study the influence of β-blockers on the EMG pattern under exercise conditions was by Tesch et al. (1984), who discovered no differences in IEMG or MPFS during cycling, but did find a decrease in MPFS during β-blockade in comparison to the control group. This could be indicative of muscle fatigue or decreased muscle fibre conduction velocity (v_{MFC}) (Merletti and Roy 1996; Mortimer et al. 1970). MPFS was calculated by describing data captured at 90 W initial work rate, and all subsequent data was normalised against it. However, this normalisation technique can be questioned as Ebenbichler et al. (1998) concluded that maximal voluntary contraction (MVC) is the most reliable form of reference when conducting MPFS analyses. This has to be considered throughout investigation of biological signals in which the electrical and mechanical activities of the recruited motor units are summed (Esposito et al. 1998). Derman (1993) examined IEMG patterns of exercising subjects ingesting β-blockade and found no difference in MVC but significantly higher IEMG activity.He concluded that the higher IEMG was due to additional recruitment of non-fatigued skeletal muscle fibres to maintain the same work rate. However, in this study MPFS was not determined. Therefore, the hypothesis for this study is that during submaximal exercise to exhaustion, β₁-blocker ingestion will result in an altered neuromuscular recruitment pattern, shown by both the IEMG and MPFS signal. Accordingly, we examined the effects of acebutolol, a selective β₁-blocker with intrinsic sympathetic activity on IEMG and MPFS during successive cycle rides at 30%, 50% and 70% of peak work rate and at fatigue.

### Methods

#### Subjects

Ten healthy males who were physically active on a regular basis volunteered for the study. Three subjects were unable to complete all experiments due to adverse effects such as headaches, dizziness and nausea whilst ingesting beta blockade. The mean age (SD) of the remaining subjects was 26.1 (2.1) years (range 23–30 years), height 181 (9) cm (range 169–194 cm), mass 78.6 (9.7) kg (range 62–94 kg) and percent body fat 14.8 (2.7)% (range 10.6–17.7%). The mean lean thigh volume (V_{LT}) was 6,492 (928) cc (range 4,629–7,381 cc). All subjects were well-informed about possible risks associated with the experiment and gave their informed consent before participation.

#### Preliminary testing

To determine peak power output (W_{peak}), a modified protocol as described by Hawley and Noakes (1992) was used. Subjects performed a 10-min warm-up on an electrically braked cycle ergometer (Lode, Groningen, Netherlands). The starting power output was determined by multiplying the subject's body mass by 2.5 W. The load was subsequently increased every 150 s by, first, 50 W and then by 25 W until the subjects were unable to maintain force output, or pedalling frequency dropped from 90 to < 50 rev min⁻¹. W_{peak} was defined as the last completed work rate in watts plus the fraction of time spent in the final non-completed work rate, multiplied by 25 W.

#### Tablet ingestion

Following the progressive exercise tests, the subjects ingested acebutolol for one of the two phases of the trial in a random order. The trials took place over 1-week periods with a 10-day wash out in between trials. Subjects were instructed to consume two 200 mg capsules between 0700 and 0900 hours before breakfast for a period of 7 days.

#### Blood sampling

On the last day of each phase the subjects were instructed to report to the laboratory. An 18-gauge Teflon cannula (Jelco, Johnson and Johnson, Halfway house, South Africa) was positioned in an antecubital vein and connected to a three-way stop cock (Uniflex, Mallinckrodt, Hennef-Seig, Germany). This cannula was flushed periodically with 2–3 ml of sterile saline containing heparin (5 IU ml⁻¹) and was used for the collection of venous blood samples (10 ml) at rest and during exercise. Venous blood samples (10 ml) were drawn at rest, at the end of each 15 min work rate and at exhaustion. The samples were then divided into aliquots, which were put into an ice-cold tube containing potassium oxalate and sodium fluoride for later determinations of lactate concentrations. The tubes were centrifuged at 3,000 g for 10 min at 4 °C immediately after the completion of the trial and the supernatants were stored at –20 °C for later analyses of plasma lactate. Plasma lactate concentrations was measured with spectrophotometric (Beckman model 35; Beckman Instruments, Fullerton, Calif, USA) enzymatic assays (Lactate PAP, BioMieux, Lyon, France; NEFA half-micro test, Boehringer Mannheim, Germany).

#### Maximal isometric voluntary contraction

To normalise EMG recordings during cycling it was first necessary to perform maximal isometric force output testing (Hunter et al. 2002), which is not affected by β-blocker ingestion (Derman et al. 1993). The strength of the subjects’ right knee extensors was measured on an isokinetic dynamometer (Kin-Com Chattanooga Group Inc., USA). Subjects sat on the dynamometer and their hips, thighs and upper bodies were firmly strapped to the seat. In this position their hip was at 100° angle of flexion. The right lower leg was then attached to the arm of the dynamometer at a level slightly above the lateral malleolus of the ankle joint and the axis of rotation of the dynamometer arm was aligned with the lateral femoral condyle. The dynamometer arm was then set so that the knee was at a 60° angle from full leg extension. Each subject performed four sub-maximal familiarisation contractions prior to performing two maximal MVCs, the latter of which was used for subsequent analyses. All subjects were encouraged verbally to exert maximal effort during both MVCs.