# THE EFFECT OF CADENCE ON THE RATE OF NEUROMUSCULAR FATIGUE AND THE SLOW COMPONENT OF OXYGEN UPTAKE IN CYCLISTS DURING HEAVY EXERCISE

by

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## Abstract

This study was designed first to replicate previously published data which examined the effect of cadence on the rate of neuromuscular fatigue of the vastus lateralis muscle in cyclists during heavy exercise and how neuromuscular fatigue was incorporated in the selection of preferred cadence in cyclists. It was the intention of this study to expand previous work and include the examination of other lower limb musculature and detemine a global measure of neuromuscular fatigue for each cadence selected. In addition, the present study sought to assess the affect of cadence on the slow component of oxygen uptake ($VO_2$ SC) and attempt to establish a relationship between this slow component and a global measure of neuromuscular fatigue. Twelve cyclists performed four sessions of cycling exercise at a power output corresponding to their individual ventilatory thresholds (VT). Each criterion exercise session was performed at a different cadence, ranging from 55 to 100 RPM in 15 RPM steps. A measure of the rate of neuromuscular fatigue was calculated using both a previously published and a novel methodology. Neuromuscular fatigue was evaluated in vastus lateralis, soleus, gastrocnemius, biceps femoris, and gluteus maximus; a global representation was calculated as the sum of the rates of neuromuscular fatigue from all muscle groups. No level of neuromuscular fatigue was observed during the criterion exercise for any of the cadence conditions. There was no relationship between a global representation of neuromuscular fatigue and cadence. The $VO_2$ SC displayed a significant quadratic trend ($p < 0.05$) over the range of cadences, with a calculated minimum value at 80.8 RPM. No relationship between neuromuscular fatigue and the $VO_2$ SC was established, suggesting a different mechanistic basis for these two variables. These data suggested that cyclists do not use a measure of neuromuscular fatigue in the selection of preferred cadence at power outputs corresponding to VT. The cadence minimizing the $VO_2$ SC did not match the cyclists' preferred cadence, however did corresponded to the pedal frequency previously shown to maximize the efficiency of Type I muscle fibers.

I would like to acknowledge the contribution that many individuals have made not only to my Thesis but to my entire graduate experience. I would like to thank my family for their continued understanding and support throughout my Master's degree. I would like to thank my fellow grad students with whom I have spent so much of my time. Specifically Kelly Moore, Kevin McKim, Andrea Kalda, Mike Bodner, and Erica Sprules all deserve my highest regards for helping me get through the tough times and enjoy the good times. Thank you to all of my committee members for their support and assitance during the last year. I would like to specially thank my supervisor, David Sanderson. His guidance over the last 3 years has been an enriching experience.

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## 1. Introduction

During cycling exercise, it is possible to maintain a constant power output at a variety of pedaling rates through the manipulation of bicycle gearing. Generally, experienced cyclists prefer relatively high pedaling rates, approximating 90 RPM (43,77). These pedal rates do not correspond with the cadence eliciting the lowest metabolic demand, measured by oxygen consumption. Rather between a range of 50-60 RPM is consistently reported as the most economical or efficient (12,20,31,37,78) and is termed the optimal cadence. Researchers thus feel that there is some determinant for cadence selection other than lowest metabolic cost. This disparity between preferred and optimal cadence has been addressed in a number of studies, examining factors other than $\dot{V}O_2$ which may contribute to cadence selection including rate of perceived exertion (55,70), pedal forces (71), muscle blood flow (41), muscular or mechanical efficiency (31,81,99), force-velocity curves (83), fitness level (12,58), glycogen depletion rates and fibre type recruitment (1,38,87), muscular activity measured by EMG (40,60), and fatigue measured by EMG (88,89).

Recently, a series of papers has been published assessing the effect of cadence on the rate of neuromuscular fatigue (88,89), quantified as the measurable rate of increase in integrated EMG over time. The vastus lateralis muscle was selected to represent the lower limb muscular response to exercise. EMG signals were sampled from both non-cyclist and cyclists during high intensity cycling exercise, at 75% and 85% of $\dot{V}O_2$ max respectively. The authors demonstrated that a pedaling rate at which the minimal rate of neuromuscular fatigue could be obtained did exist and did not match the cadence eliciting the lowest average $\dot{V}O_2$. The cadence where minimal rate of neuromuscular fatigue was observed corresponded to the preferred pedaling rate of subjects, 70 RPM for non-cyclists and 80-90 RPM for cyclists. The speculated cause of the observed neuromuscular fatigue was an increase in the number of motor units which were firing as well as an increase in the firing rate of already active motor units (28,44,92,101).

With their results, Takaishi et al. have argued that the difference in the sensing of fatigue played an important role in deciding the preferred cadence for cycling exercise (89). They speculated that rate of neuromuscular fatigue was closely associated with perceived exertion. Preferred cadence has been investigated using a subjective rate of perceived exertion (RPE) scale. RPE studies on cadence manipulation have speculated that perceived exertion is peripheral in origin, meaning that information arising from muscular innervation and contraction are more

important than central variables such as heart rate and blood lactate (55). Pandolf and Noble found that RPE at 80 RPM was lower than at 40 or 60 RPM even though $\dot{V}O_2$ was higher (70). They concluded that RPE was most likely an interplay of a number of contributory factors; however, for cycling exercise, the local factor associated with feelings of strain in the working muscles and joints was most important. It has been suggested that peripheral mechanoreceptors, important in determination of perceived exertion, may have two sensory modes: tension and rate of change of tension (55). The combination of active or passive movements and of tension or limb position may induce the observed RPE-cadence relationship. A term which may be used to describe this perception of force is sense of effort. Sense of effort is a centrally mediated sensation, arising from internal neural correlates (corollary discharge) of the descending motor command (50). Sense of effort most likely reflects the magnitude of the voluntary motor command. The linear envelope of measured EMG from a muscle has been used as an indirect measure of the excitatory input or neural drive sent to a muscle, reflecting both the number of active motor units and their discharge rates (14). Neuromuscular fatigue may be a measure of the rate of increase of the sense of effort and could be used, as suggested by Takaishi et al., for pedaling rate selection during cycling exercise.

Takaishi et al. predicted that their subjects were exercising within the domain of heavy intensity (88). During heavy intensity exercise at a constant power output, there was an additional metabolic cost superimposed on the rapid $O_2$ kinetics associated with exercise onset, elevating $\dot{V}O_2$ above the predicted level based on the sub-lactate threshold (LT) $\dot{V}O_2$ –work rate relationship (76). This $\dot{V}O_2$ slow component ($\dot{V}O_2$ SC) has been widely studied; however, its mechanistic basis is poorly understood. There has been some debate regarding whether the observed increases in $\dot{V}O_2$ are central or peripheral in origin. Increases in oxygen uptake of working muscles have been observed in both static and dynamic work, suggesting that peripheral mechanisms are primarily responsible (76,94). One hypothesis accounting for $\dot{V}O_2$ slow component centres on fiber type recruitment and bases arguments on chemical and mechanical efficiency differences between Type I and Type II muscle fibers (74). Specifically, Type II fibers have a greater chemical energy cost for tension development, owing to lower chemical-to-mechanical coupling efficiency, faster actomyosin turnover rate, and increased calcium pump activity. If Type II are serially recruited during heavy exercise, to compensate for some loss in contractility of already active Type I fibers, this would be reflected as an increase in the oxygen uptake. Two recent publications have dealt with the effect of pedal rate on the slow component

of $\dot{V}O_2$. Gaesser et al. (32) found significant differences in the amplitude of the $\dot{V}O_2$ SC between pedaling rates of 50 and 100 RPM during supra-lactate cycling exercise, with $\dot{V}O_2$ SC at 50 RPM being higher. In addition, Bartow et al. (7) also examined this relationship, but over several cadences ranging from 45 to 90 RPM. While they reported no significant relationship between pedal frequency and $\dot{V}O_2$ SC amplitude, they did report a significant difference between the 45 and 75 RPM conditions. They admited that their exercise duration, 8 minutes, may not have been long enough to reflect differences in $\dot{V}O_2$ that might occur later in exercise and thus an overall relationship to cadence was not seen. If the mechanistic basis of the $\dot{V}O_2$ SC is a serial recruitment of less efficient Type II fibers then cadences which preferentially recruit Type II fibers may elicit a greater response of the $\dot{V}O_2$ SC. It has been observed that glycogen depletion patterns between Type I and Type II fibers differs during heavy exercise and can be altered through manipulation of cadence (1). It was suggested that Type II fibers may be preferentially recruited at both high and low cadences, in situations requiring either low tension/ high velocity contractions or high tension/ low velocity contractions. The relationship between cadence and the $\dot{V}O_2$ SC may be such that a minimization of the $\dot{V}O_2$ SC occurs at a cadence where Type I fibers are preferentially recruited, approximately 80 RPM.

There has also been limited research assessing the relationship between $\dot{V}O_2$ and iEMG in working muscles. Positive correlations between oxygen consumption and iEMG have been observed for both downhill running (29) and cycling (11). As well, Shinohara and Moritani have reported a significant correlation between $\dot{V}O_2$ and iEMG during cycling exercise within the heavy intensity domain (80). Because the $\dot{V}O_2$ SC and neuromuscular fatigue share a common speculated basis, Shinohara et al. suggested there may be a direct relationship between these two variables and Takaishi et al. (89) have speculated that cyclists may choose a cadence which minimizes increased $\dot{V}O_2$ during prolonged exercise.

The intent of the present study was to expand upon previous work assessing the relationship between neuromuscular fatigue, the $\dot{V}O_2$ SC, and pedaling frequency. For the present research, fatigue was defined as any exercise induced reduction in the ability to exert muscle force or power, regardless of whether the task can be sustained (10). Within this context, neuromuscular fatigue, quantified as an increase in iEMG over time due to a serial recruitment of motor units, was used as an index of fatigue. Additionally, the $\dot{V}O_2$ SC, quantified as any

increase in $\dot{V}O_2$ beyond 3 minutes of exercise, was used as a marker of fatigue. The extent of neuromuscular fatigue was examined in 5 muscles (vastus lateralis, biceps femoris, gluteus maximus, gastrocnemius, and soleus) and a global representation of neuromuscular fatigue was calculated as the sum of the neuromuscular fatigue rates for all muscles. Subjects cycled at an intensity corresponding to ventilatory threshold (VT) as the use of a relative measure of exercise intensity, such as VT, more accurately defines the boundaries of exercise domains than a percentage based intensity. This exercise intensity ensured subjects were within the heavy domain of exercise, above their lactate threshold (LT). The application of the present data in non-laboratory situations was also considered. Research suggests that cyclists typically choose to ride at or near their VT during prolonged bouts of intense exercise, such as racing (46).

## 2. Research Plan

*i. research questions*

1. **Is the rate of neuromuscular fatigue in selected lower limb musculature minimized at one selected pedaling rate?** The intent of this study was to examine the effect of pedal frequency on the rate of neuromuscular fatigue, quantified as an increase in the integrated EMG over time, in cyclists by replicating the work of Takaishi et al. (89), and to broaden the scope of this assessment through inclusion of a broader range of lower limb musculature. It was hypothesized that the minimized rate of neuromuscular fatigue for all selected muscular, represented globally by a sum of the rates for all selected muscles, would show a minimization at one of the criterion pedaling rates.

2. **If a cadence exists which minimizes the rate of neuromuscular fatigue, what is the relationship to steady-state metabolic cost?** It was hypothesized that the pedaling rates eliciting the minimal rate of neuromuscular fatigue would be higher than that of minimal metabolic cost. The cadence corresponding to the lowest rate of neuromuscular fatigue would correspond to subjects' preferred cadence.

3. **What is the relationship between the slow component of $\dot{V}O_2$ and cadence?** It was hypothesized that the $\dot{V}O_2$ SC, quantified as the increase in $\dot{V}O_2$ from the $4^{th}$ to $15^{th}$ minutes of exercise, would vary over cadence conditions, displaying a quadratic relationship with an observable minimum.

4. **If minimum rates exist for both the rate of neuromuscular fatigue and the $\dot{V}O_2$ SC at selected cadences, do they correspond?** It was hypothesized that the minimal rise in $\dot{V}O_2$ due to the slow component would match the pedaling rate at which neuromuscular fatigue was minimized.

*ii. methods*

Male cyclists (n = 12) were selected on the basis of their training history and level of aerobic fitness. Criteria for selection including active cycle training or participation in cycle racing events, and a maximal oxygen consumption of greater than 55 ml/kg/min or 4.5 l/min. The subject group consisted of both local and national level competitive cyclists, including mountain bikers, road racers and triathletes. While this sample was not truly be random, it adequately represented the cycling population in terms of physical characteristics, modes, timing, and intensity of training, and level of conditioning. All subjects read and signed an informed consent detailing all aspects of the protocol, which had been approved by the University of British Columbia Ethical Review Committee. Subjects were instructed to continue their present training regime during the testing period. In addition, subjects were asked to refrain from alcohol, caffeine, and high intensity exercise for 24 hours preceding each exercise session. Anthropometric measures, as well as current training and racing history, were recorded for each subject. No remuneration was given for participation in this study.

Maximal oxygen uptake ($\dot{V}O_2$ max) was defined as the highest 15 second average $\dot{V}O_2$ value attained during a maximal bicycle test. Subjects performed a ramped exercise test to point of exhaustion on an electronically braked cycling ergometer (Quinton Excalibur, Gronigen, The Netherlands). Following a warm-up of approximately 5 minutes in duration, the ramp test began. Beginning at 0 load, the test intensity increased by 30 Watts/min until subject exhaustion. For the ramped test, and all subsequent exercise sessions, subjects were cooled with a 50 Watt fan. Subjects were allowed to self-select cadence throughout the test. The average cadence chosen for the present study was 89.3 ± 5.0 RPM. While it has been published that $\dot{V}O_2$ max derived from a 80 RPM test is significantly higher than that derived from a 60 RPM test (57), the absolute differences are less than 2 ml/kg/min for a cadence range of 60 to 120 RPM. For a range of cadences between 60 and 120 RPM, the standard error of the mean (SEM) equals 1.83 ml/kg/min with an overall average of 58.7 ml/kg/min, thus a pre-selected cadence protocol was not deemed necessary. $\dot{V}O_2$, $CO_2$, and VE were measured using an automated gas analysis system (Rayfield, Waitsfield, VT) at 15 second intervals for all testing sessions. In addition, heart rate (HR) was recorded at 15 second intervals using a telemetric recording device (Polar Vantage, Kempele, Finland). Ventilatory threshold (VT) during the ramped protocol was determined for each subject

by VE/ $\dot{V}O_2$ method of visual inspection by an individual experienced in this protocol (79). Individual subject data for VT calculation can be found in Appendix II.

Four subsequent sessions of the criterion exercise, 55 RPM, 70 RPM, 85 RPM, and 100 RPM, were performed in a randomized order on separate days, with a minimum of 24 hours between tests. Each session consisted of a self-selected warm-up followed by a 15 minute ride at a calculated power corresponding to VT. The criterion exercise sessions were performed on a standard racing bicycle mounted on a Velodyne Trainer (Schwinn, Chicago, IL). The Velodyne Trainer can simulate both the inertial and non-inertial loading of riding on the road and is a valid and reliable alternative to the Monark bike ergometer whose use is standard for physiological testing (2). The manufacturers' reported accuracy for power output is within 5%. A Cateye cycling computer was mounted on the bike, and monitored by both subject and tester, to ensure the selected cadence was maintained within ± 1 RPM. Subjects used their own clipless pedals and shoes; seat height was standardized at 100% trochanteric length (12). VE, $\dot{V}O_2$, and HR measures were averaged over 1 minute intervals. A steady-state measure was calculated as the average response over the 4$^{th}$ minute to observe a baseline, and then compared to the average over the 15$^{th}$ minute of exercise. Physiologic differences, $\dot{V}O_2$ SC, ΔVE, and ΔHR, were calculated as the absolute differences between 4$^{th}$ and 15$^{th}$ minutes of exercise.

Physiological variables for each cadence condition were individually analyzed and any relationship over cadence was noted. All subjects did not all respond to the cadence conditions in an invariant manner. Some subjects had either a linear or quadratic response to cadence while others showed no apparent pattern; therefore, the average data were used rather than individual data for subsequent analysis. The average responses reported include data from all 12 subjects. The response to cadence conditions for each physiological dependent variable was fitted mathematically to a polynomial equation. For each significant quadratic trend, the differential equation was solved for 0 to calculate the cadence at which the variable was minimized.

Myoelectric signals were recorded during the four criterion exercise sessions using surface EMG. 10 subject data sets were evaluated for neuromuscular fatigue. Two subject data sets were discarded due either to loss of the ground electrode or a buffer overrun of the A/D recording software. A single pre-amplified bi-polar electrode (Ag- AgCl) was placed over the muscle belly of each of the vastus lateralis (VL), biceps femoris (BF), gluteus maximus (GM), soleus (SOL), and gastrocnemius (GAS) muscles (Therapeutics Unlimited, Iowa City, IA). Electrode placement followed site protocol outlined by Basmajian and Blumenstein (8) and care

was taken to minimize trial to trial variation in placement. A reference electrode was placed on the wrist. All electrode placements were preceded by removal of hair, abrasion of the skin, and alcohol swabbing. The RMS signal was sampled continuously at 150 Hz by a 12-bit analog-to-digital card (Data Translation DT2801, Marlboro, MA) and stored to disk using a data collection software package (LABTECH Notebook, Laboratory Collections Technologies Corporation, Wilmington, DE). The RMS time constant was set at 11.75 msec and the signal was high pass filtered at 20 Hz. The sampling frequency and time constant selected for the RMS EMG provided the closet match to the burst integral and duration of rectified and filtered (30 Hz) raw EMG sampled at 1000 Hz, collected over the same period.

For purposes of replication of Takaishi et al.'s work (88,89), neuromuscular fatigue of the VL, and of the SOL, GAS, BF, and GM, was calculated using the iEMG slope method. The RMS signal was integrated into 20 sec segments and plotted against time. The iEMG slope was calculated using linear regression and the slope of the line used as a relative measure of the rate of neuromuscular fatigue. Subsequent to this, all 5 muscles were analyzed by comparing differences in the average EMG bursts from the $4^{th}$ minute and the $15^{th}$ minute. This method had advantages over the iEMG slope method. It did not rely on a linear EMG fatigue model, thereby allowing evaluation of non-linear responses. It allowed a comparison of not only integral values but also of burst duration and mean amplitude of RMS signal. As well, this method isolated individual muscles bursts, filtering out the measurable signal between muscle bursts. An 'in house' program (Microsoft Excel, Visual Basic) was used to calculate the average traces. A baseline measure was calculated as the smallest average 100 msec window over 30 sec during the $10^{th}$ minute of exercise. A threshold was set as the baseline plus 4 standard deviations. This threshold level was found to be most accurate in differentiating between individual muscle bursts. Individual muscle bursts were defined as any above threshold EMG activity lasting between 25% and 100% of the time for a single crank revolution. Representative bursts were calculated as the average of 50 bursts beginning at both the $4^{th}$ and $15^{th}$ minute of exercise. From this representative burst, the duration, integral, and mean amplitude were calculated. A measure of neuromuscular fatigue was quantified as the percent difference between $4^{th}$ and $15^{th}$ minute values for the integral values ([$15^{th}$ min – $4^{th}$ min]/ $4^{th}$ min * 100%).

All dependent variables were analyzed using a single factor RM ANOVA model. SPSS statistical software was used for all analyses. Level of significance for all measures was set a priori at $P < 0.05$. In addition to reporting an F score and p value for each RM ANOVA,

polynomial orthogonal contrasts also assessed any significant trends and the grand mean of all conditions was tested for significant difference from 0.

## 3. Results

### *i. subject characteristics*

The physical characteristics of subjects are provided in Table I. The physiological profiles of the subject pool are within the range of well trained cyclists. Comparisons to reported values for elite road (USCF) and off-road cyclists (NORBA) revealed moderately lower scores for most criteria tested (100). $\dot{V}O_2$ max values are less than reported values (4.99 l/min NORBA; 5.09 l/min USCF) and % $\dot{V}O_2$ max at VT are also lower (77.1% NORBA; 80.1% USCF). Maximal power and power output at VT are consistent with reported values (420 W, 271 W NORBA; 470 W, 321 W USCF respectively). These comparisons suggest that the range of subjects used in the present study represent a continuum from moderately trained individuals to elite level cyclists.

| Subject # | Age (yrs) | Ht (cm) | Mass (kg) | $\dot{V}O_2$ Max (L/min) | $\dot{V}O_2$ Max (ml/kg/min) | Ventilatory Threshold (VT) (ml/kg/min) | Max Power (Watts) | Power at VT (Watts) | % $\dot{V}O_2$ Max at VT | Preferred Cadence (RPM) | Cycling Experience (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **1** | 21 | 182.6 | 75.5 | 4.79 | 63.4 | 47.0 | 453.0 | 300.0 | 74% | 98.9 | 4 |
| **2** | 30 | 171.6 | 64.4 | 4.24 | 65.8 | 50.9 | 416.0 | 275.0 | 77% | 96.5 | 10 |
| **3** | 26 | 181.2 | 76.3 | 4.68 | 61.3 | 42.2 | 457.5 | 275.0 | 69% | 104.5 | 6 |
| **4** | 25 | 191.2 | 88.9 | 5.02 | 56.5 | 40.9 | 487.5 | 350.0 | 72% | 81.3 | 6 |
| **5** | 23 | 186.1 | 71.7 | 4.49 | 62.6 | 41.3 | 420.0 | 250.0 | 66% | 91.4 | 3 |
| **6** | 25 | 177.6 | 71 | 4.16 | 58.6 | 39.9 | 450.0 | 275.0 | 68% | 75.9 | 4 |
| **7** | 28 | 188.3 | 77.5 | 5.10 | 65.8 | 45.4 | 510.0 | 325.0 | 69% | 87.1 | 8 |
| **8** | 21 | 173.3 | 61.1 | 3.58 | 58.6 | 47.8 | 367.5 | 250.0 | 82% | 79.8 | 2 |
| **9** | 39 | 186 | 85 | 4.77 | 56.1 | 40.1 | 450.0 | 300.0 | 71% | 86.6 | 7 |
| **10** | 23 | 177.7 | 77.7 | 5.46 | 70.3 | 49.7 | 502.5 | 325.0 | 71% | 94.1 | 6 |
| **11** | 24 | 179.8 | 71.5 | 4.74 | 66.3 | 49.4 | 427.5 | 275.0 | 74% | 90.0 | 3 |
| **12** | 28 | 179.6 | 74.4 | 4.93 | 66.3 | 40.5 | 422.5 | 300.0 | 61% | 85.8 | 10 |
| Average | 26.1 | 181.3 | 74.6 | 4.66 | 62.6 | 44.6 | 447.0 | 292.0 | 71% | 89.3 | 5.75 |
| SD | 4.9 | 5.9 | 7.7 | 0.5 | 4.5 | 4.2 | 40.3 | 30.8 | 5% | 5.0 (8.3*) | 2.7 |

Table I - Summary Characteristics of Subjects. Note EMG data includes subjects 1-10 while physiologic data includes subjects 1-12. * = standard error of mean

*ii. neuromuscular fatigue – iEMG slope method*

Examples of RMS EMG data from each muscle group are shown in Figure 1A-E. 20 seconds of RMS EMG are shown at the beginning of minute 3 (i) and minute 14 (ii) for the SOL, GAS, BF, VL, and GM muscles. For replication of Takaishi et al.'s work (89), RMS EMG was segmented into 20 second blocks and integrated. The 20 second integral values were plotted from minute 3 till minute 15 for calculation of the iEMG slopes. The average traces were calculated from the first 50 bursts beginning at minute 3 and minute 14, for comparison of integral values, burst duration, and mean amplitude.

The calculated iEMG slopes showed at least two different response patterns. Figure 2A shows a single subject data set for the VL in which all cadence conditions elicited positive iEMG slopes. The more common response, however is shown in Figure 2B, where 1 or more cadence conditions had negative iEMG slopes, indicating a progressive decrease in integrated RMS EMG over the exercise duration. The fit of a linear regression to the RMS data was both observed visually and by examination of the R value. There were several cases were R values were low and/ or a linear regression did not seem to fit the RMS data. Low R values were a result of any of 3 reasons: 1) The RMS data visually displayed a linear relationship but the regression slope was close to 0, 2) The 20 sec iEMG intervals were dispursed about the linear regression line, and 3) The EMG response over time was not a linear relationship. Figure 2C displays an example of each case.

The average iEMG slopes for the SOL, GAS, BF, VL, and GM are shown in Figure 3. Comparision of the present data for the VL with that of Takaishi et al. (89), revealed considerable differences in both the amplitude of the iEMG slopes and their relationship to cadence. The present data showed smaller, or negative, iEMG slope values with much greater intersubject variability than in Takaishi et al.. Calculations of the present data for VL yielded no significant differences between means ($F_{3,24} = 0.216$, $p = 0.884$) and the grand mean of cadence conditions showed no significant difference from 0 ($F_{1,8} = 0.596$, $p = 0.462$). This indicated that for the VL muscle, no level of neuromuscular fatigue, as defined by Takaishi et al. (89), occured. Visual inspection of the remaining muscle groups revealed similar trends of small, or negative, average iEMG slopes and large intersubject variability. No statistical evaluation was completed for these remaining muscles as an alternate method for evaluation of neuromuscular fatigue was used.

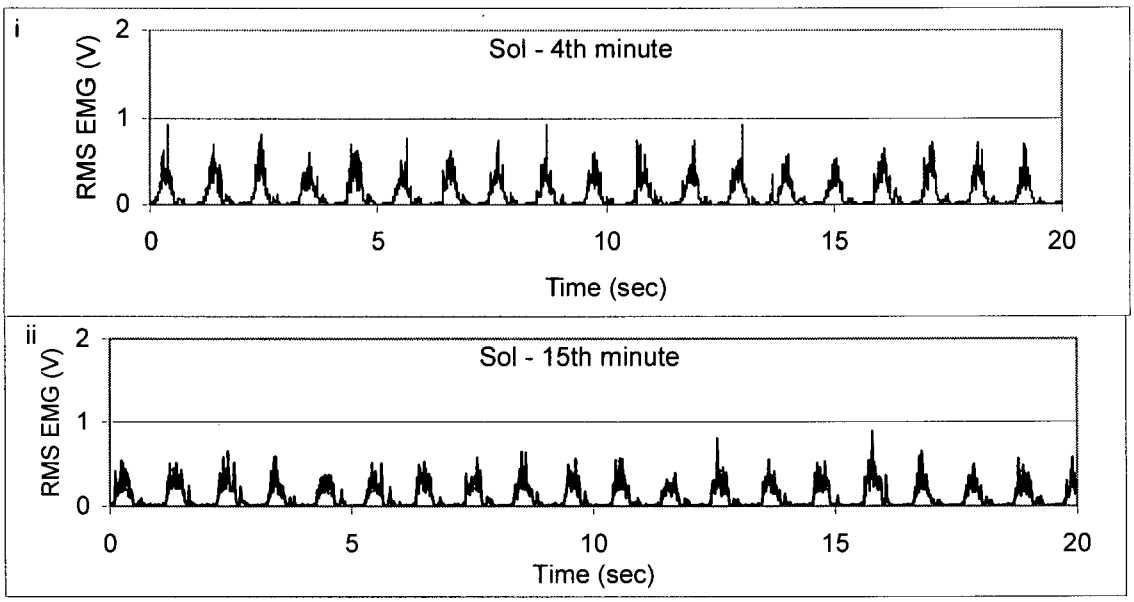


Figure 1A

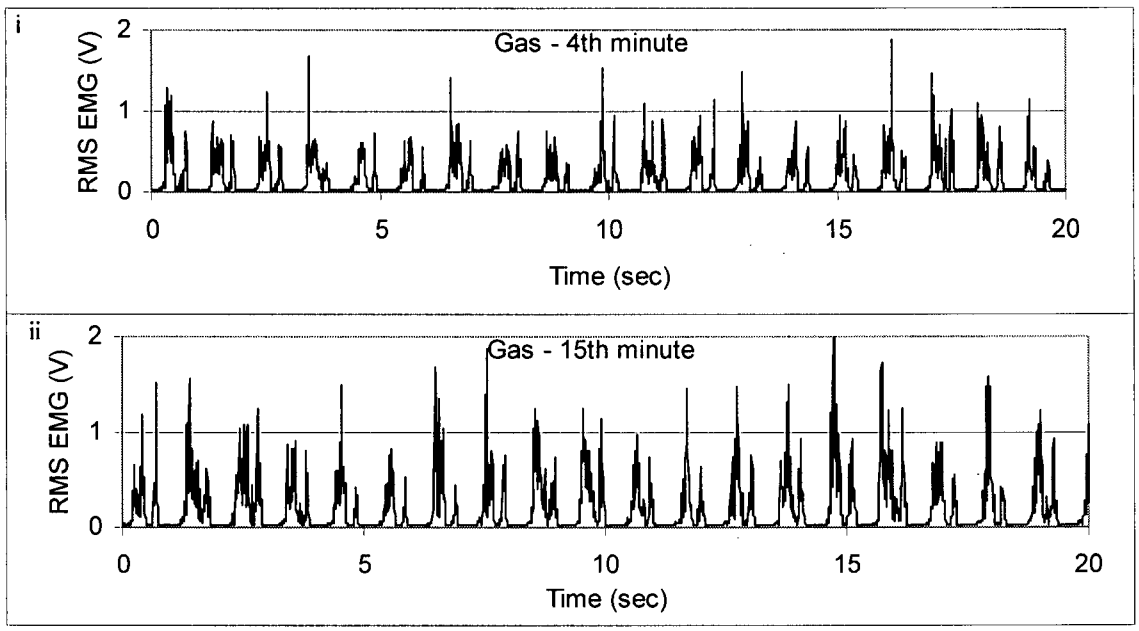


Figure 1B

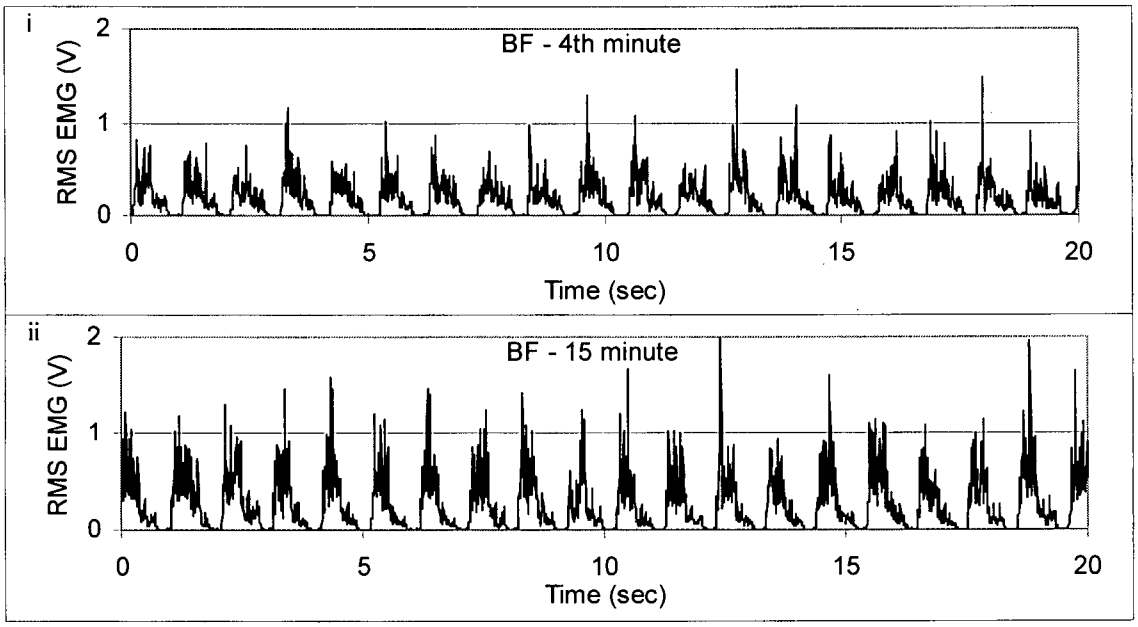


Figure 1C

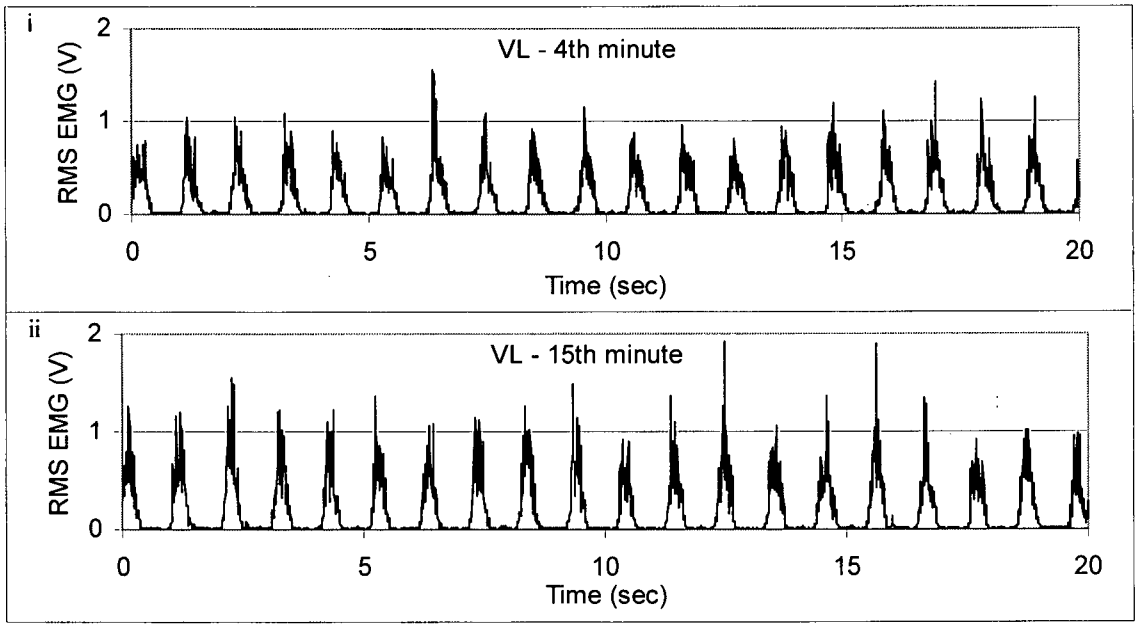


Figure 1D

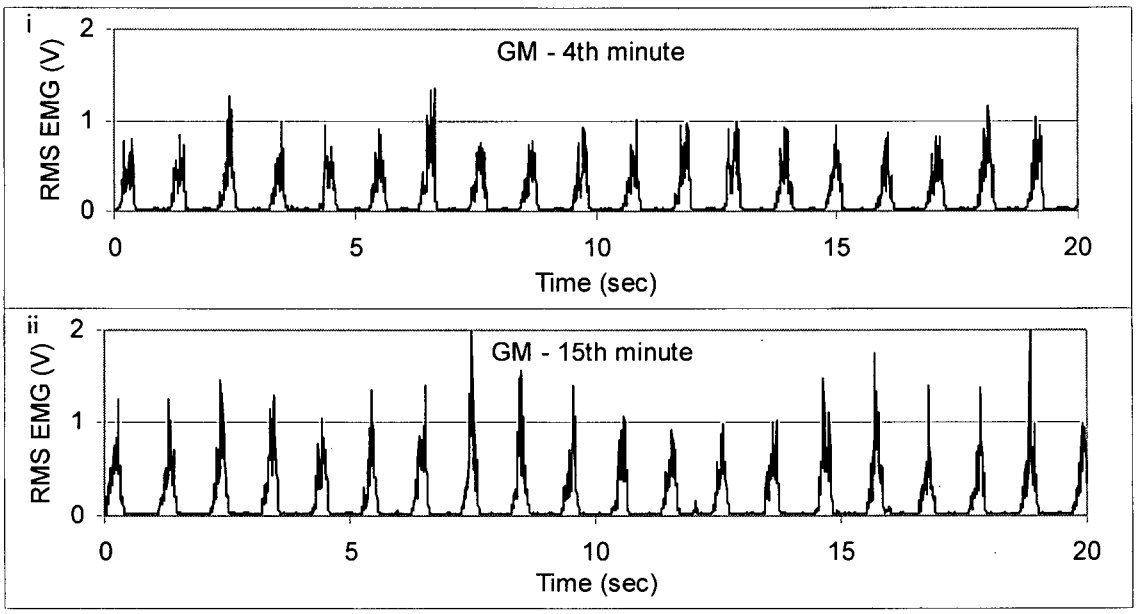


Figure 1E

Figure 1 - RMS EMG for (A) – soleus, (B) – gastrocnemius, (C) – biceps femoris, (D) – vastus lateralis, and (E) – gluteus maximus muscle from single subject. 20 seconds of EMG activity displayed from beginning of 4$^{th}$ minute (i) and 15$^{th}$ minute (ii). iEMG slopes calculated from 20 sec blocks of integrated RMS EMG from 4$^{th}$ min to 15$^{th}$ min. Average trace comparisons calculated from 1$^{st}$ 50 bursts beginning at 4$^{th}$ min and 15$^{th}$ min.

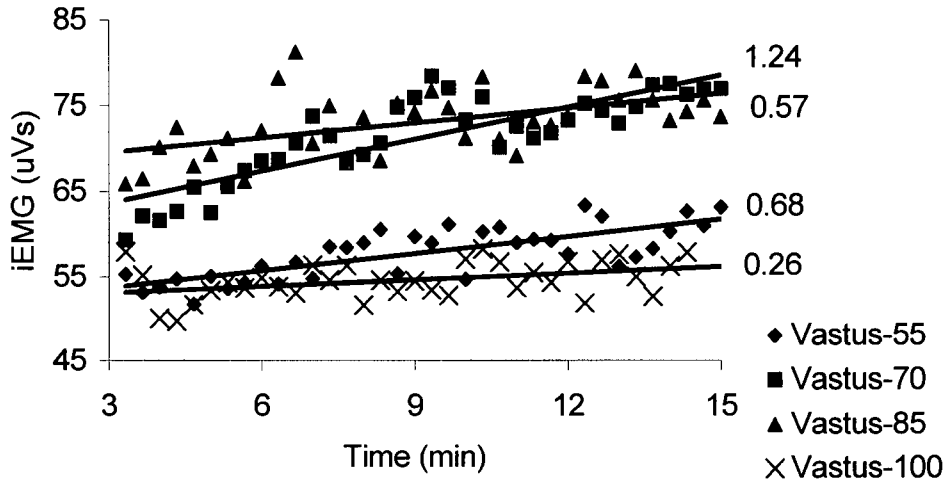


Figure 2A

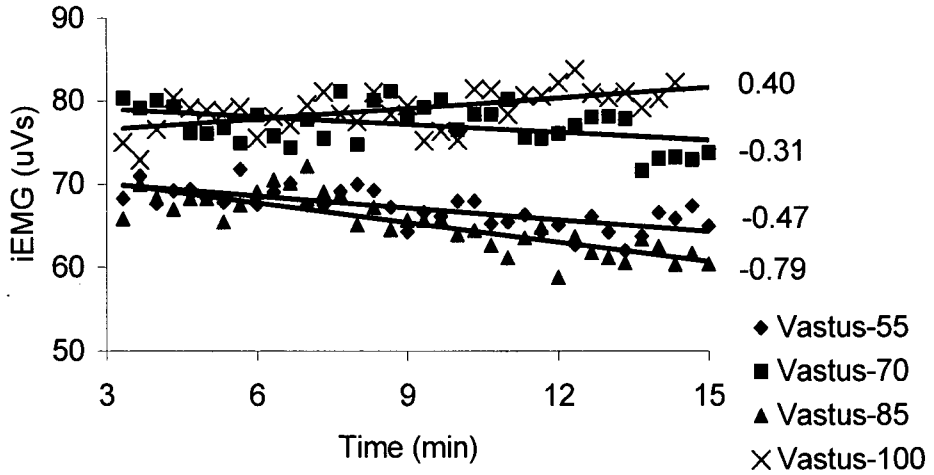


Figure 2B

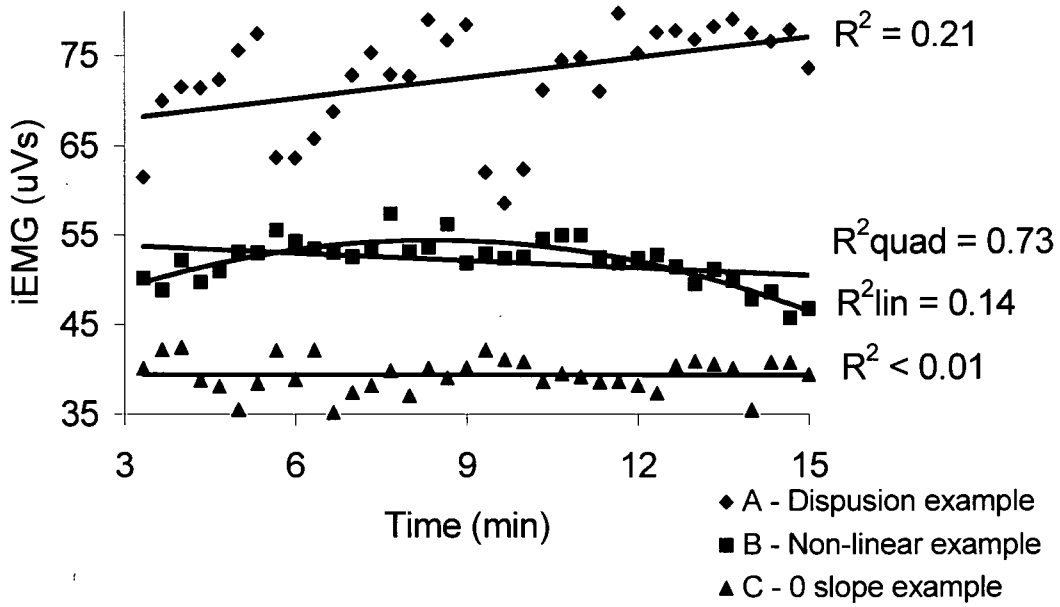


Figure 2C

Figure 2 (A) – Data set showing individual cadence conditions for vastus lateralis of a single subject. 20 second integrated RMS EMG blocks are plotted beginning from the 4th min until end of exercise, at 15 min. iEMG slope through each set of symbols is fitted as a linear regression line. iEMG slope is noted beside each condition. Note all condtions yield position iEMG slopes. (B) – Typical data set for vastus lateralis. iEMG slopes calculated similarly to Figure 1A. 1 or more cadence conditions elicits negative iEMG slope response. iEMG slope is noted beside each condition. (C) – Examples of low R values with linear regression for calculation of iEMG slope. Not all iEMG slopes fit a linear regression model. Some iEMG slopes had large dispursion of data points about the iEMG slope (A), some iEMG slope fit a curvilinear response (B), while other iEMG slopes equaled 0 (C) indicating no change in iEMG over time.

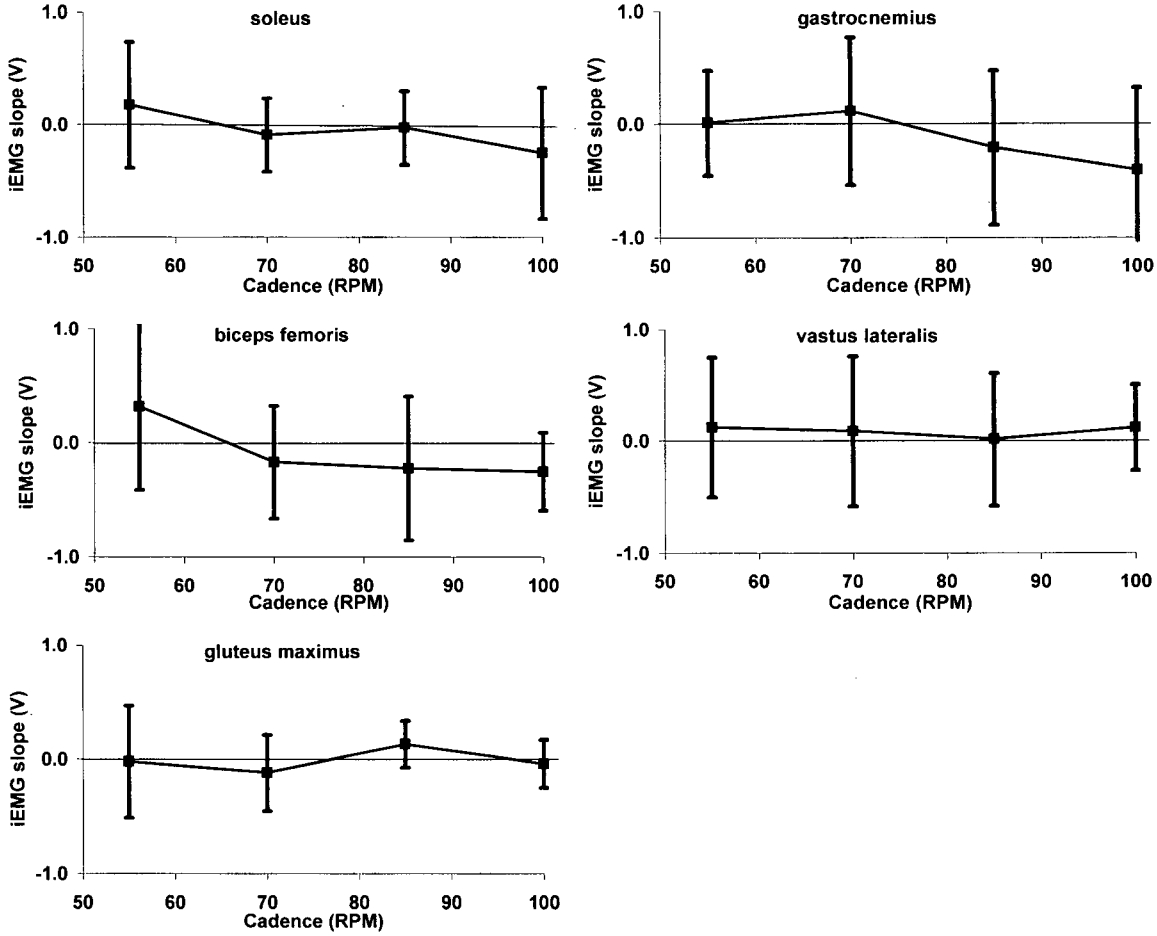


Figure 3 – Average iEMG slopes for SOL, GAS, BF, VL, and GM muscles. Positive slopes indicate a progressive increase in iEMG over time. Error bars represent ± 1 SD.

*iii. neuromuscular fatigue – average burst differences in integral, duration, and mean*

Figure 4 shows the percent change in iEMG of individual muscle bursts between the 4th minute of exercise and the 15th minute of exercise over the cadence conditions for each muscle. There were no significant differences in percent difference of integral values from minute 4 to minute 15 between cadence conditions for any of the 5 muscles tested ({VL $F_{2.3,20.6} = 579$, $p = 0.592$}, {SOL $F_{3,27} = 0.827$, $p = 0.491$}, {GAS $F_{3,18} = 1.713$, $p = 0.2$}, {BF $F_{2.4,21.7} = 0.706$, $p = 0.529$}, {GM $F_{3,24} = 1.43$, $p = 0.258$}); nor were any of the grand means over cadence conditions different from 0 ({VL $F_{1,9} = 0.127$, $p = 0.73$}, {SOL $F_{1,9} = 0.029$, $p = 0.868$}, {GAS $F_{1,6} = 0.011$, $p = 0.919$}, {BF $F_{1,9} = 0.124$, $p = 0.733$}, {GM $F_{1,8} = 0.247$, $p = 0.633$}). BF was the only muscle group which showed a significant linear trend ($F_{1,9} = 10.651$, $p = 0.01$), decreasing as cadence increased.

Muscle burst duration and mean amplitude were also calculated for each muscle group. The purpose of these calculations was to differentiate each average muscle burst into components of time and intensity. Burst duration and mean amplitude for each muscle group is shown in Figure 5 and Figure 6 respectively. Similar to the iEMG calculations, no significant differences between cadence conditions were found for any muscles groups for either burst duration or mean amplitude. In addition, the grand means for all muscles were not significantly different from 0 for either duration or mean amplitude.

*iv. physiological variables*

$\dot{V}O_2$, seen in Figure 7A, showed a significant cadence condition effect for the 4th minute of exercise ($F_{2.6,29} = 5.186$, $p = 0.007$) and a significant linear trend ($F_{1,11} = 14.918$, $p = 0.003$), with $\dot{V}O_2$ increasing as cadence increased. 7 of 12 subjects showed some response, either linear or quadratic, over the cadence conditions. While trend analysis revealed a significant linear trend, observation of the average data and polynomial regression reveals a higher $R^2$ value for a quadratic curve than a linear ($R^2 = 0.99$ and $R^2 = 0.87$ respectively). A non-significant quadratic trend was probably due to the lack of values for cadence conditions lower than 55 RPM. The equation of best fit for 4th minute $\dot{V}O_2$ value minimized at 60.3 RPM. There were 3 different

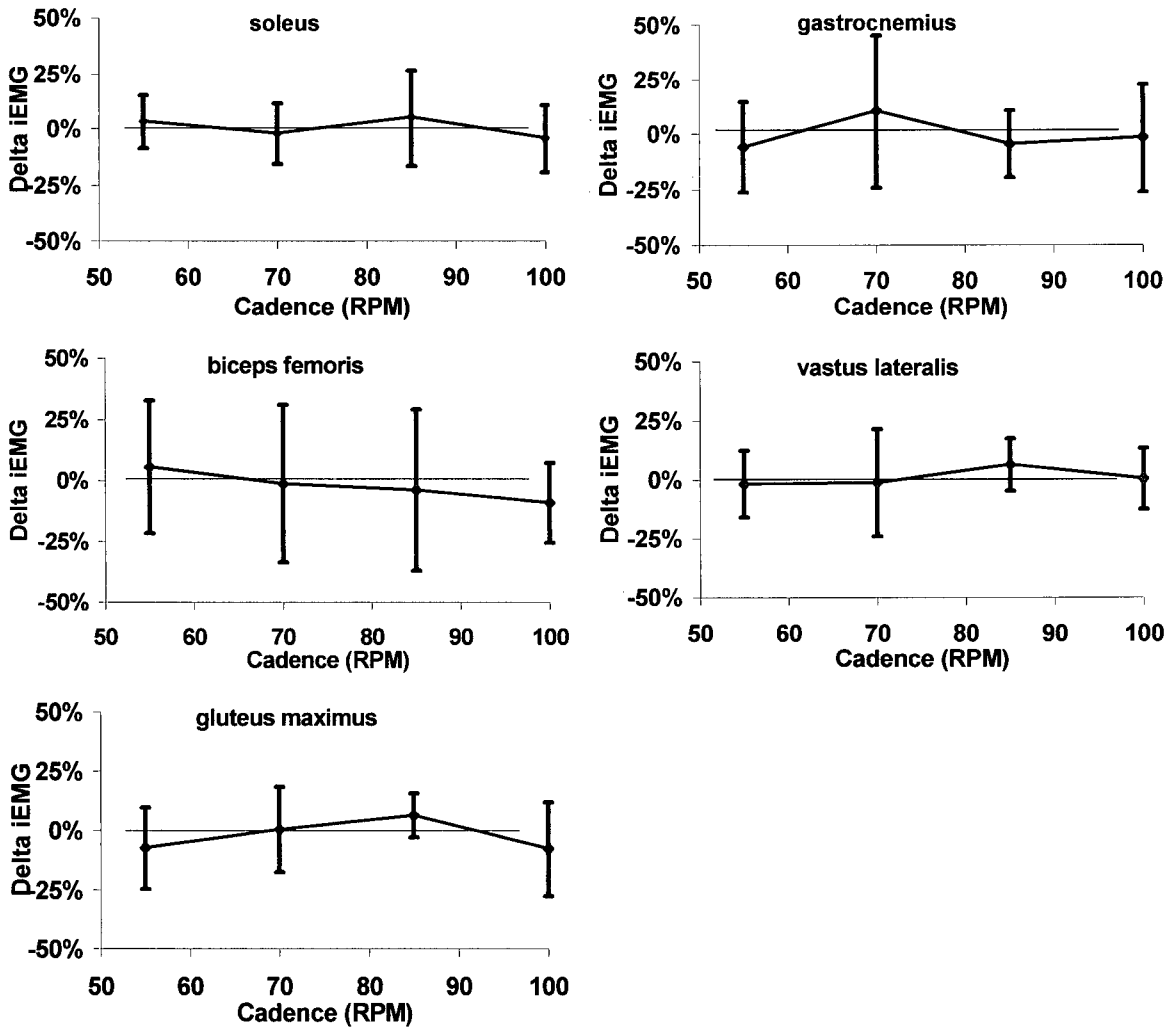


Figure 4 – Percent differences between 15th and 4th minute iEMG of average muscle bursts during exercise for 5 lower limb muscles, calculated as (15th min – 4th min)/4th min (positive value = increase in iEMG over exercise duration). Error bars represent ± 1 SD.

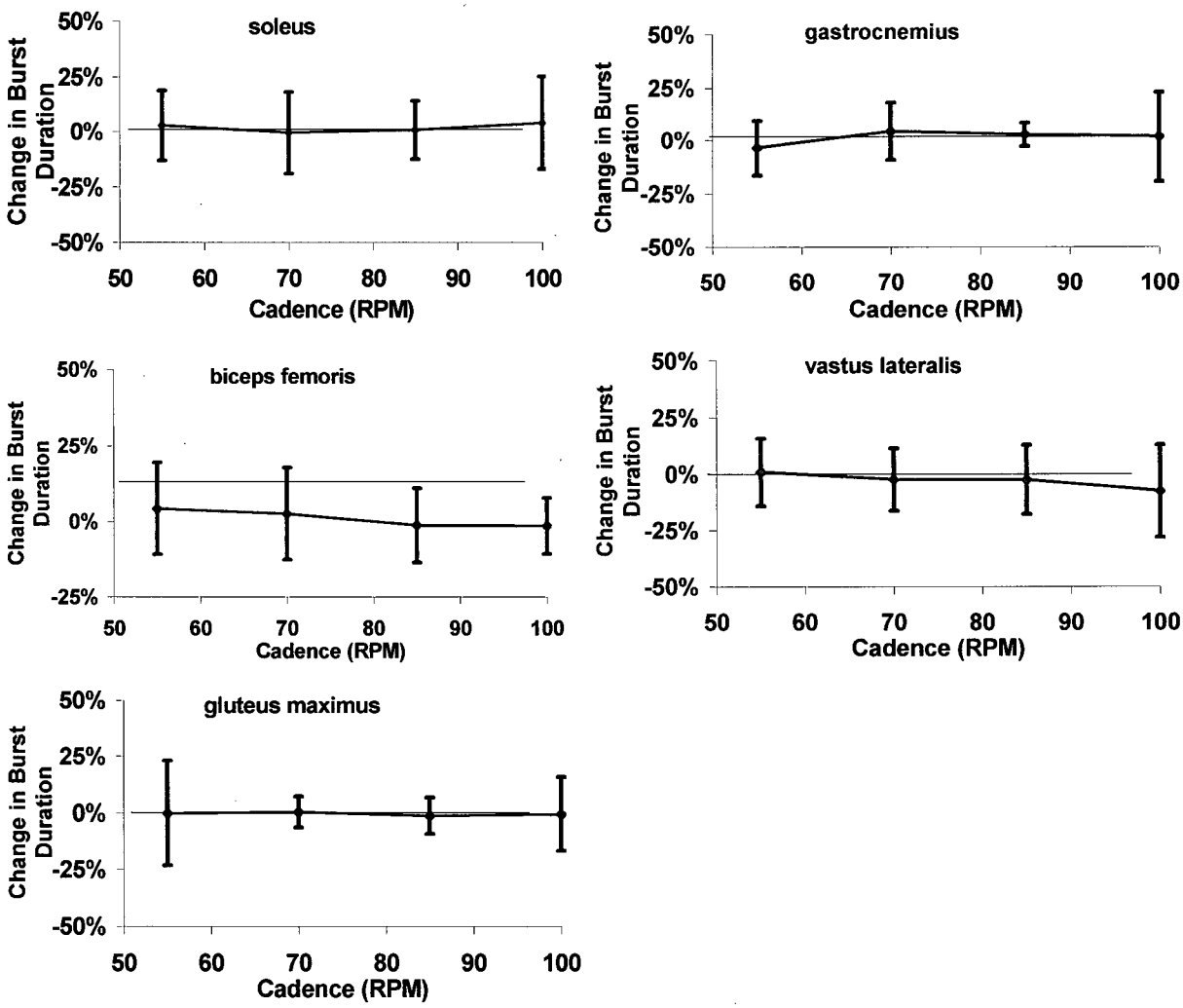


Figure 5 – Percent differences between 15th and 4th minute durations of average muscle bursts during exercise for 5 lower limb muscles, calculated as (15th min – 4th min)/4th min (positive value = increase in duration over exercise period). Error bars represent ± 1 SD.

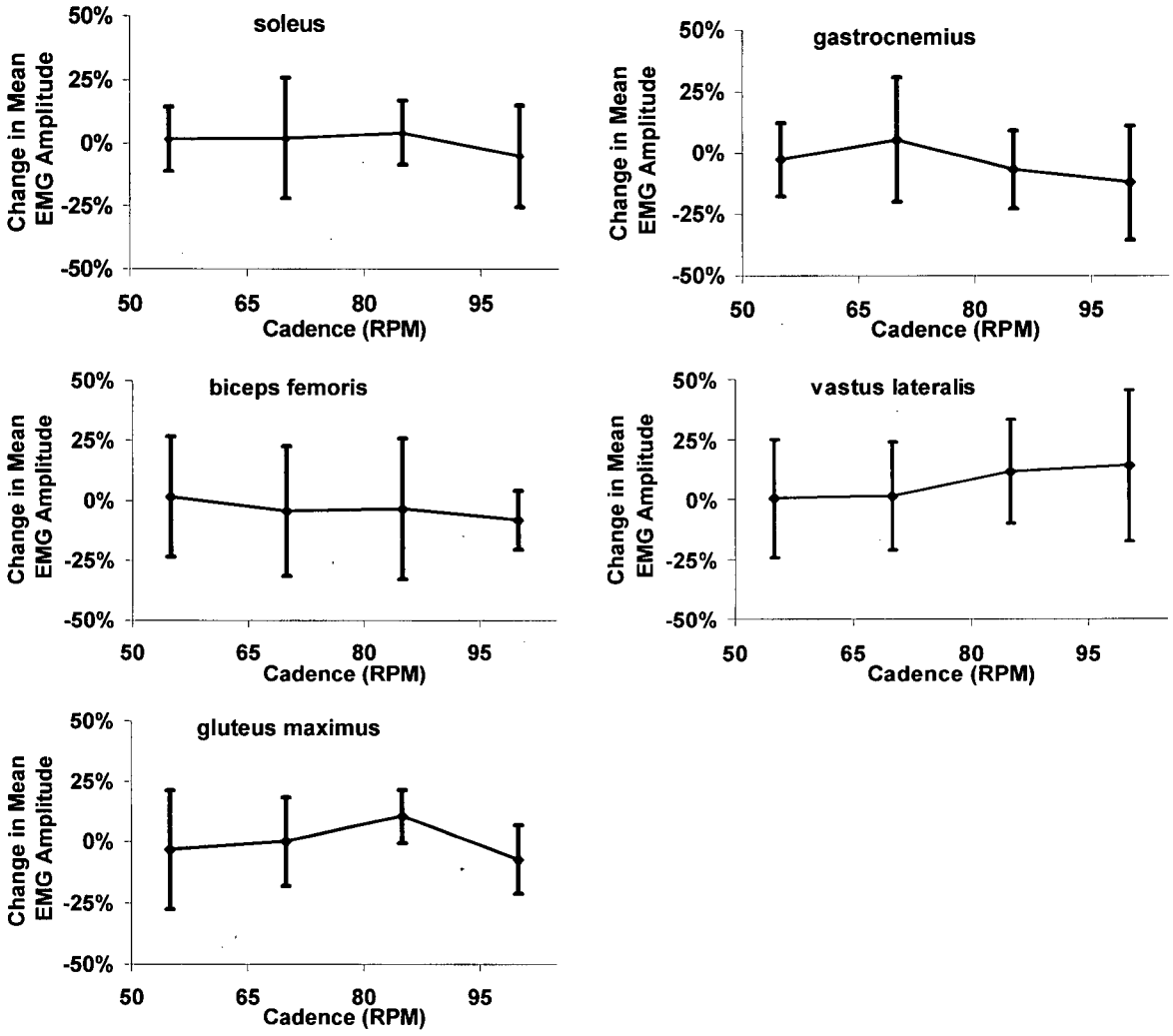


Figure 6 – Percent differences between 15th and 4th minute average amplitude of single muscle bursts during exercise for 5 lower limb muscles, calculated as (15th min – 4th min)/4th min (positive value = increase in amplitude over exercise duration). Error bars represent ± 1 SD.

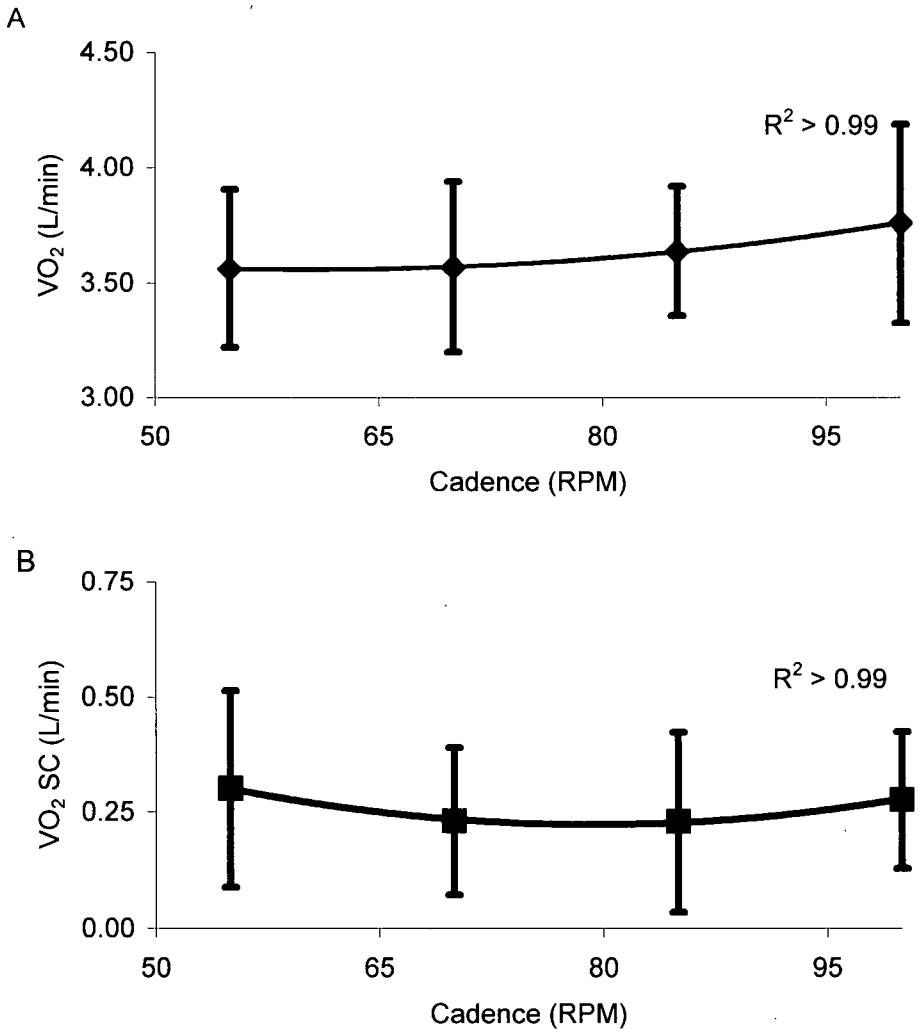


Figure 7 – $VO_2$ response to cadence conditions. (A) – Absolute $VO_2$ response during 4$^{th}$ minute of exercise. (B) – $VO_2$ SC measured as difference from 4$^{th}$ to 15$^{th}$ minute. Error bars represent ± 1 SD.

response patterns for the $\dot{V}O_2$ SC over cadence conditions. Upon visual inspection, 3 of 12 subjects showed no apparent trend, while 7 of 12 appeared to have a quadratic trend and the remaining 2 displayed a linear trend which increased as cadence increased. The $\dot{V}O_2$ SC, Figure 7B, did not show a significant cadence condition effect, however there was a significant quadratic trend ($F_{1,11} = 5.252$, $p = 0.043$). The equation of best fit for the mean $\dot{V}O_2$ SC over cadence displayed a minimum at 80.8 RPM. Table II displays the optimal and preferred cadence conditions for individual subjects. 11 of 12 subjects preferred cadence conditions higher than their optimal condition.

| **Subject** | **Optimal Cadence Condition (RPM) and {Corresponding $\dot{V}O_2$ (l/min)}** | **Preferred Cadence Condition (RPM) and {Corresponding $\dot{V}O_2$ (l/min)}** |
|---|---|---|
| **1** | 70 {3.54} | 85 {3.75} |
| **2** | 100 {3.30} | 85 {3.45} |
| **3** | 55 {3.48} | 85 {3.53} |
| **4** | 55 {3.99} | 100 {4.29} |
| **5** | 55 {3.15} | 70 {3.16} |
| **6** | 70 {3.21} | 85 {3.45} |
| **7** | 70 {3.73} | 85 {3.91} |
| **8** | 55 {3.05} | 70 {3.15} |
| **9** | 55 {3.68} | 85 {3.79} |
| **10** | 70 {3.94} | 100 {4.26} |
| **11** | 55 {3.25} | 85 {3.42} |
| **12** | 70 {3.59} | 85 {3.64} |

Table II – Optimal and Preferred Cadence Conditions for Individual Subjects.

In Figure 8A, the ventilatory response during the 4$^{th}$ minute revealed a significant effect over cadence conditions ($F_{2.27,24.5} = 4.335$, $p = 0.021$) and displayed both significant linear ($F_{1,11} = 5.135$, $p = 0.045$) and quadratic trends ($F_{1,11} = 5.038$, $p = 0.046$). 7 of 12 subjects showed either a linear or quadratic response over the cadence conditions. 6 of those 7 subjects matched those who responded, linearly or quadratically, for $\dot{V}O_2$ . The increase in ventilation from the 4$^{th}$ to

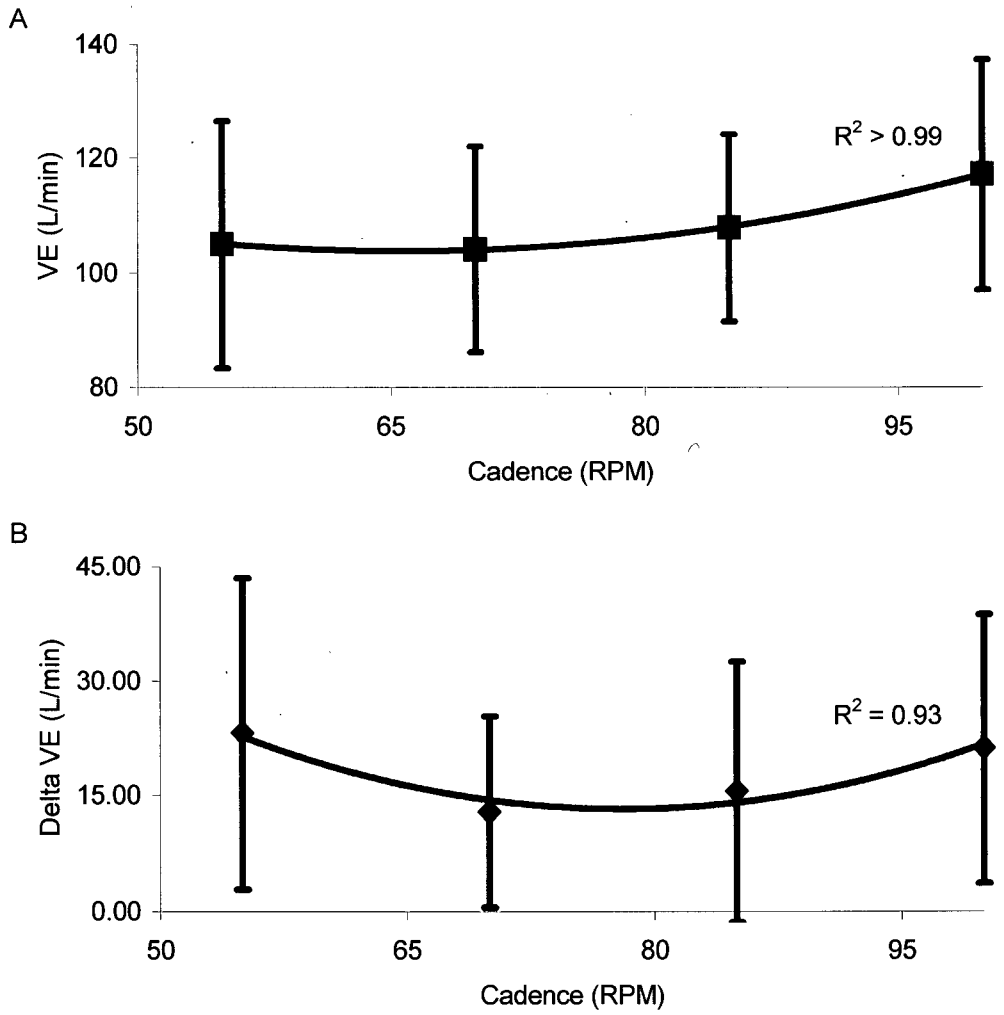


Figure 8 – Ventilatory response to cadence conditions. (A) – Ventilation during 4th minute of exercise. (B) – Delta VE - Increase in ventilation from 4th to 15th minute of exercise. Error bars represent ± 1 SD.

$15^{th}$ minute of exercise, Figure 8B, was non-significant over cadence conditions but revealed a significant quadratic trend ($F_{1,11} = 5.175$, $p = 0.044$). Similar to $\dot{V}O_2$ SC response, there were more than 1 response patterns for change in ventilation. Upon observation, 5 subjects displayed a quadratic relationship across cadence conditions, 1 displayed a decreasing linear relationship, and the remaining 6 showed no apparent pattern over cadence 4th minute. 4 of the 5 subjects with quadratic responses matched subjects who also had quadratic $\dot{V}O_2$ SC response. HR response during the $4^{th}$ minute, seen in Figure 9A, displayed a significant cadence effect ($F_{2.9,31.8} = 8.096$, $p < 0.001$) and trend analysis revealed both significant linear ($F_{1,11} = 14.56$, $p = 0.003$) and quadratic ($F_{1,11} = 7.335$, $p = 0.02$) trends. The change in HR from $4^{th}$ to $15^{th}$ minute, Figure 9B, was non-significant over cadence conditions.

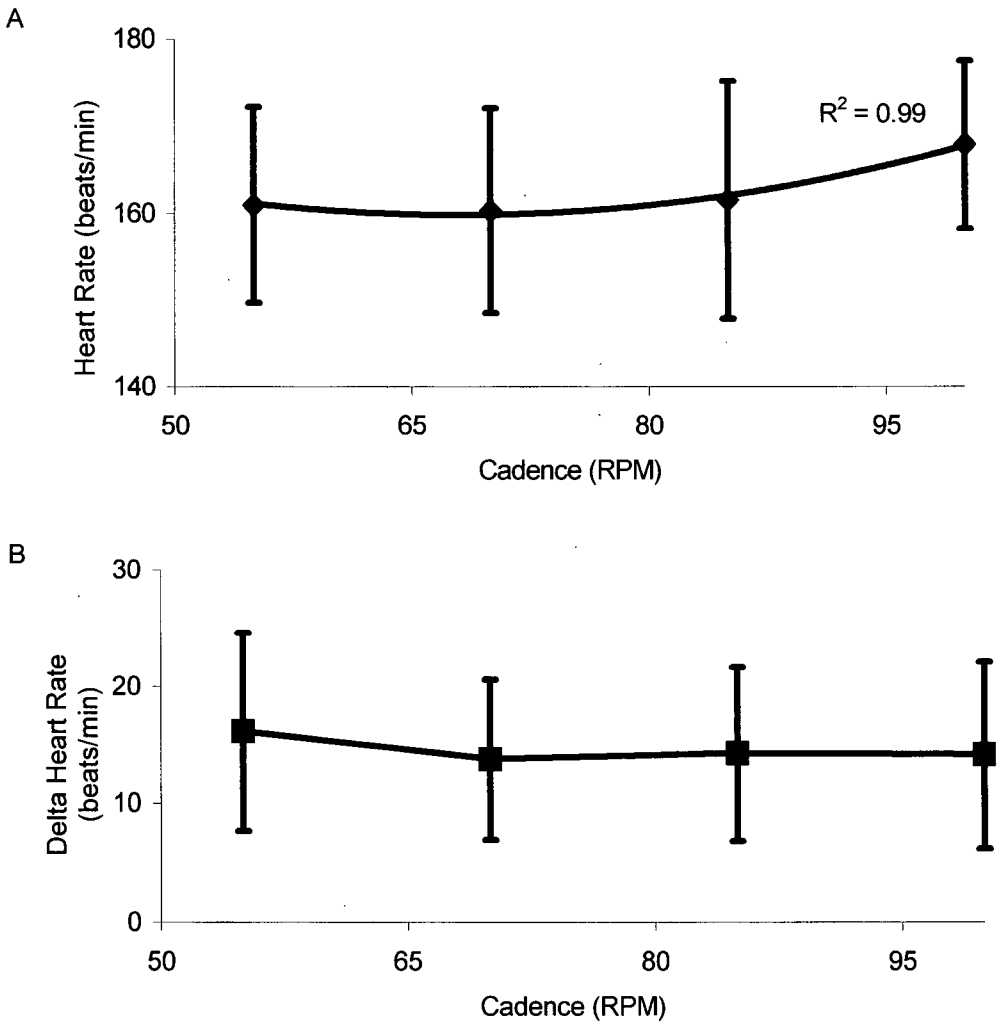


Figure 9 – HR response to cadence conditions. (A) – HR response during 4$^{th}$ minute of exercise. (B) – Delta HR - Increase in HR from 4$^{th}$ to 15$^{th}$ minute. Error bars represent ± 1 SD.

## 4. Discussion

### *i. replication of previous literature*

Before expanding Takaishi et al.'s (87,88) work, one of the initial objectives of the present work was to replicate their previously published studies. In their work, a model for neuromuscular fatigue was used to evaluate cycling cadence in both cyclists and non-cyclists. Neuromuscular fatigue was quantified as the slope of the integrated EMG (iEMG) signal averaged over 20 second intervals for the duration of exercise. Takaishi et al. cited previous studies which found a progressive increase in iEMG during both static and dynamic contractions (60,63,72). This increase in iEMG was attributed to a progressive recruitment of additional motor units and/or an increase in the firing rates of already active motor units. Takaishi et al. hypothesized that there would be differences in the extent of neuromuscular fatigue during prolonged pedaling against a given exercise intensity at different pedaling rates. Their results showed that neuromuscular fatigue of the VL was minimized at cadences corresponding to subjects' preferred pedaling rate. The results of the present study were not in agreement with those of Takaishi et al. (87,88). The most obvious discrepancy between the present study and Takaishi et al.'s works is that the criterion exercise did not invariantly produce increases in iEMG over the exercise duration. In fact, nearly 50% (17/39) of the VL trials for individual subject cadence conditions produced negative iEMG slopes. There were a number of factors which may have influenced either the muscular response or the recorded EMG signal. In light of the differences observed in iEMG response for the vastus lateralis between this study and Takaishi et al.'s (87,88), it was necessary to reconsider the changes made in the methods for the present study.

The subjects which participated in the present study were trained cyclists similar to those used by Takaishi et al. (88) in 1996. However, the average relative and absolute $\dot{V}O_2$ max values for the present study (62.6 ± 4.5 ml/kg/min, 4.66 ± 0.5 l/min) are higher than Takaishi et al.'s subject pool (57.7 ± 1.2ml/kg/min, 3.55 ± 0.38 l/min). This difference in aerobic capacity may affect the neuromuscular response to the criterion exercise. It has been reported that aerobic training diminishes the $\dot{V}O_2$ SC (15,35,101) and that $\dot{V}O_2$ max is negatively correlated with $\dot{V}O_2$ SC (7). If $\dot{V}O_2$ kinetics and iEMG during fatiguing exercise are related, as suggested by Shinohara et al. (79), then the effect of increased aerobic fitness may be to attenuate the rate of

neuromuscular fatigue. Previous to their examination of trained cyclists, Takaishi et al. (87) has published an additional study, similarly designed, examining neuromuscular fatigue in healthy, but non-trained individuals which also found increases in iEMG at varying cadences during heavy exercise. These results indicated that a measure of neuromuscular fatigue during cycling activity may be relatively robust across individuals with varying levels of fitness, at least up to some level of aerobic conditioning, such as characterized by Takaishi et al.'s 1996 subject pool.

The single greatest difference in protocols between the present study and Takaishi et al.'s work (87,88) is the exercise intensity for the criterion exercise sessions. Takaishi et al. rode subjects at 85% and 75% of their power output at $\dot{V}O_2$ max for cyclists and non-cyclists respectively. The current study chose to use an exercise intensity equaling the power output achieved at ventilatory threshold (VT). This criterion power output equaled an average of 71 ± 5% of their maximal oxygen uptake. However, at steady-state, over the 4$^{th}$ minute of exercise, the average $\dot{V}O_2$ relative to $\dot{V}O_2$ max was 78 ± 5% during the cadence conditions. Over the 15$^{th}$ minute, the average response was 84 ± 7% of $\dot{V}O_2$ max. While the 4$^{th}$ minute $\dot{V}O_2$ outputs are considerably higher than the estimated VT from subjects' initial maximal $\dot{V}O_2$ test, the disparity between calculated VT and the average oxygen cost over cadence conditions may be partially explained by the observation that $\dot{V}O_2$ kinetics during ramp protocols are significantly slower than those found in step tests (66). Takaishi et al. states that some subjects in their study reached values of almost 95% of their $\dot{V}O_2$ max during criterion exercise sessions at extreme high or low cadences (88). In the present study, 2 subjects reached 97% of their $\dot{V}O_2$ max at either the 55 or 100 RPM cadence conditions. In addition, all subjects in the present study rode at higher power outputs than in Takaishi et al.'s trained cyclists study (88). Power outputs in Takaishi et al.'s study ranged from 200-240 Watts, while the range in the present study ranged from 250-350 Watts, with an average of 292 ± 30.8 Watts. While still unclear, it seems unlikely that differences in exercise intensity protocol between the 2 studies would result in such dramatic differences in results, with a complete absence of neuromuscular fatigue observed in the VL for the present study.

The goal of Takaishi et al. in choosing 75% and 85% for untrained non-cyclists and cyclists respectively was to exercise individuals at an intensity at which Type II muscle fibres should be recruited in addition to Type I fibres (87,88). They cited earlier works assessing glycogen depletion patterns (1,38,92) which found that exercise intensities between 85 and 90%

resulted in significant Type II fibre depletion. The present study used the relative measure of power output at VT as the criterion exercise intensity because of its physiological significance. VT, shown to be temporally linked to lactate threshold (LT) (6), defines the lower end of the heavy exercise domain (34). It is within this domain of exercise intensity which the linearity of the $\dot{V}O_2$-work rate relationship breaks down and an additional oxygen cost is superimposed during the Phase 3 kinetics. The underlying cause for both neuromuscular fatigue and the slow component of $\dot{V}O_2$ was proposed to be a serial recruitment of Type II fibres to compensate for the deficit in contractility of already active fibers (79,87,88). The work assessing glycogen depletion patterns cited by Takaishi et al. (88) examined the Type IIa fiber glycogen depletion at varying exercise intensities (92). Vollestad et al. (92) found that 60 minutes of exercise at 61% $\dot{V}O_2$ max resulted in a 38% reduction of glycogen in the Type IIa fiber population compared with an 80% reduction for exercise to exhaustion at 91% of $\dot{V}O_2$ max. They estimated that 68% of the Type IIa fibers had lower glycogen levels after the 61% exercise intensity while after the 91% intensity exercise, 72% of Type IIa fibers had some level of glycogen depletion. An additional study (47) found that exercise intensities as low as 52% of $\dot{V}O_2$ max recruit all fibers, regardless of type, and that all fibers share in the contraction to a substantial degree, even at moderate workloads. These results indicate that lower exercise intensities recruit similar populations of Type IIa fibers as higher intensity exercise, although their absolute contribution to exercise may be less. While all subjects, in both the present study and Takaishi et al.'s, may have been exercising within a domain strenuous enough not to allow achievement of steady-state $\dot{V}O_2$ throughout the criterion exercise periods, there may exist a true physiologic difference in exercise response between the present study's exercise intensity and those of Takaishi et al.(87,88).

ii. *neuromuscular fatigue*

In addition to the vastus lateralis, EMG from 4 other muscles of the lower limb was also measured. The original purpose of the present study was to assess neuromuscular fatigue in these muscles in order to assess a systemic or global response. It was felt that VL, in isolation, may not provide an adequate representation of the entire lower limb and that the evaluation of the combined effect of other muscles may be a more accurate correlate to other fatigue variables,

such as the $\dot{V}O_2$ SC. This study's lack of replication of the VL iEMG response to cadence published by Takaishi et al. (87,88) is a limitation to further evaluation of other musculature of the lower limb. In light of the VL iEMG slope response in the present study, all 5 muscles were then analyzed as average discrete muscle bursts at steady-state and end of exercise. If any differences in iEMG were found over time, then the content of the integral could be partitioned into burst duration and average RMS amplitude. No such differences were found for any muscle. The lack of fatiguing EMG response for any muscle group demanded a critical evaluation of the concept of neuromuscular fatigue and its measurement.

DeVries, 1968, was one of the premier investigators of the EMG-time relationshipship during fatiguing contractions (28). He attempted to establish the linearity of iEMG time plots for varying levels of isometric contraction. DeVries' general results indicated that iEMG fatigue curves were generally linear, with linearity increasing with increased contraction strength. However, at 30% MVC, 4 out of 15 subjects displayed some degree of curvilinearity for contractions lasting approximately 3 minutes. In a separate investigation, DeVries et al. (27) found that 3 out of 14 subjects did not display linear iEMG plots at various power outputs during cycling. In this study, EMG from the VL was measured during cycling bouts of various non-maximal power outputs for durations of less than 5 minutes. DeVries et al. chose to exclude these 3 subjects from subsequent data analysis. Shinohara and Moritani (79) have reported decreases in iEMG at the onset of exercise, during cycling exercise at an intensity above VT. They found an average of 7% reduction in iEMG from exercise onset to the $4^{th}$ minute of exercise. They concede that "the large decrease of iEMG in (their) study could not be fully explained." No other studies investigating iEMG fatigue make mention of linearity of their iEMG curves. A non-linear EMG fatigue response has also been reported during isometric and dynamic exercise (44). Hagberg found that exponential fitting of RMS EMG fatigue curves fit better than linear regression.

The underlying cause of neuromuscular fatigue, quantified as an increase in iEMG over time, is presently still unclear. The most common explanation of this observed increase is due to an increase in the number of motor units which are firing as well as an increase in the firing rate of already active motor units (28,44,91,100); however, there is little direct evidence to support this claim. Maton, 1981, has reported MU recruitment and some degree of increases in firing frequency during isometric contractions (60). Moritani et al. (64) have limited evidence of MU recruitment from fine wire electrodes during cycling; however, they were unable to isolate single MU spikes due to movement artifacts. DeLuca (25) has alternately proposed that observed

increases in EMG signals during fatiguing contractions are due to filtering properties of the muscle and tissue. During a sustained contraction, the conduction velocity along muscle fibers decreases, increasing the duration of individual MU action potentials. This change in the MU action potentials is reflected in a lowering of the mean frequency in the power density spectrum. It has been theorized that muscle tissue and differential electrodes act as low-pass filters; as the distance between the active fibers and the electrodes increases, the bandwidth of the tissue filter decreases. Thus the shift to a lower mean frequency in EMG signal would increase the measurable signal at the electrode.

Another variable which has been shown to affect EMG over time is muscle temperature. An increase in muscle temperature from 34-39 °C resulted in a 4% reduction in the RMS amplitude (72). DeVries et al. (27) acknowledged that changes in muscle temperature may be problematic when using EMG, but stated that short work bouts, less than 5 minutes, may be a reasonably valid method for estimation of neuromuscular fatigue. Subsequent works using the iEMG slope, or neuromuscular fatigue, model do not mention this limitation. Muscle temperature was not measured in the present study but effort was made to cool subjects adequately during exercise, as outlined in the methodology. It is unknown to what extent temperature affected the RMS signal in the present study.

In DeVries original investigation (28), he evaluated the test- retest reliability for isometric contractions of the biceps brachii. He found reliability to be $r = 0.934$. No such reliability evaluation has been performed for dynamic contractions; therefore, the test-retest reliability is unknown. It may be erroneous to assume an equal degree of reliability between isometric and dynamic contractions. Another component of DeVries original investigation was a direct comparison between endurance time at MVC and iEMG slope (28). This correlationship was used to assess the validity of the iEMG slope measure. No direct measure of fatigue has been used in the evaluation of neuromuscular fatigue for dynamic contractions; therefore, the validity of this measure is unknown. While it is generally accepted that the amount of measurable electrical activity within a muscle increases over time for both isometric and dynamic exercise, when measured with surface electrodes (28,44,91), the cause of this response is still not fully understood. Some studies report considerable variation in EMG responses between subjects during fatiguing contractions and question its use during dynamic exercise (95). Takaishi et al.'s work (87,88), as well as the present study, failed to independently evaluate fatigue outside of the measure of neuromuscular fatigue or $\dot{V}O_2$ kinetics. The presence of

fatigue must only be assumed and the mechanistic basis for observed responses can only be speculated.

The use of iEMG slopes, or neuromuscular, fatigue has yet to be proven a valid and reliable measurement of muscular fatigue for dynamic contractions. Several studies have shown increases in iEMG during fatiguing exercise (27,64,79,87,88) but have yet to provide evidence of the underlying mechanism of observed increases. As well, increases in iEMG during fatiguing contractions do not seem to be an invariant response across all subjects. It has even been reported that observed increases in iEMG during fatiguing contractions are an artifact of the EMG recording methods which mask the true MU action potential response, a decrease in firing rate during fatiguing contractions (25).The present study failed to show any systematic increases in iEMG of the course of exercise for any muscle group regardless of cadence. Without an independent measure of fatigue, such a MVC, it is difficult to gauge the level of fatigue over the exercise period and therefore little can be concluded about electromyographic response seen in the present study.

iii. *preferred and optimal cadence*

The purpose of Takaishi et al.'s work (87,88) was to quantify the extent of neuromuscular fatigue during prolonged pedaling at different rates and to elucidate the possible mechanism for the known discrepancy between the most efficient pedaling rate and the preferred pedaling rate. They chose to ride their subjects at a relatively high power output, corresponding to the power output at 75% and 85% of their $\dot{V}O_2$ max for non-cyclists and cyclists respectively. They were able to show a match between subjects' preferred cadence and the cadence which elicited the lowest level of neuromuscular fatigue at a given level of intensity. The present study failed to replicate these findings using a power output in the same relative domain of intensity. It is of interest to examine the nature of preferred cadence and its relationshipship to exercise intensity and also examine the nature of neuromuscular fatigue and its relationshipship to power.

At present, there is conflicting evidence regarding the relationshipship between most economical cadence and power output. It has been reported that there is a linear (19,77) or non-linear (55) increase in most economical cadence with increase in power output, and also that there is no change in most economical cadence (57). Regardless, the most economical cadence at any submaximal power output is significantly lower than preferred cadence. Marsh and Martin (57) have recently examined the relationshipship between aerobic power and the power output

on preferred and most economical cadences. They found that preferred cadence for cyclists was consistent across a range of power outputs from 75-250 Watts and was always significantly higher than the most economical cadence. Their cyclists' preferred cadence averaged 93.1 RPM across all power outputs. This value is similar to the preferred cadence reported elsewhere (43,70), to values reported by Takaishi et al. for cyclists (88), and to the average preferred cadence in the present study. With the cumulative data from all of these studies, it is arguable that preferred cadence is reasonably robust across varying power outputs. This range, reported in either power output or percentage of $\dot{V}O_2$ max, is inclusive of all power outputs up to near maximal effort.

Takaishi et al. (87,88) concluded from their study of both cyclists and non-cyclists subjects select relatively higher cadences according to the minimization of neuromuscular fatigue, rather than the metabolic economy for pedaling exercise. In their conclusions they do not reference what, if any, effect manipulation of power output has on cadence self-selection and neuromuscular fatigue. However, in their 1994 paper pp.156, Takaishi et al. do report that their results indicated "that the exercise intensity adopted in individuals was high enough to elicit neuromuscular fatigue." This statement is an acknowledgment that neuromuscular fatigue is only observed at or above a given intensity of exercise, yet in their 1996 paper, pp.1492, they hypothesize that "because experienced cyclists generally prefer and use higher pedaling rates in daily training, it (was) expected that the pedaling rate of the minimal neuromuscular fatigue for cyclists (was) different from that for non-cyclists." The latter statement implies that neuromuscular fatigue should be observable at exercise intensities used in daily training, significantly lower than the 85% $\dot{V}O_2$ max used in their study for cyclists. A previous investigation by Moritani et al. (64) examined the relationshipship between a neuromuscular fatigue threshold ($EMG_{FT}$) and anaerobic threshold (determined from VT). They found a highly significant correlationship between $EMG_{FT}$ and AT ($r = 0.92$), yet the mean $EMG_{FT}$ $\dot{V}O_2$ (1.84 $\pm$ 0.55 l/min) was significantly higher than $\dot{V}O_2$ at VT (1.72 $\pm$ 0.54 l/min). Moritani et al. also assessed the level of neuromuscular fatigue at power outputs above and below $EMG_{FT}$. The iEMG slopes for power outputs at $EMG_{FT}$ or below approximated 0. Thus a subject's cadence selection may only be influenced by neuromuscular fatigue at exercise intensities significantly above anaerobic threshold.

It has been shown that a subject's preferred cadence is relatively robust over a range of moderate exercise intensities (57) and that cyclists' prefer cadences of approximately 90 RPM

(43,57,58,70). While coincident with preferred cadence, Takaishi et al.'s research (87,88) cannot explain cadence selection at power outputs lower than that corresponding to some level above anaerobic threshold. Marsh and Martin found that the preferred cadence of untrained individuals was dependent on power output, with an inverse relationshipship to power (57). As well, untrained subjects elicited a greater cadence effect on $\dot{V}O_2$ than trained subjects. Marsh and Martin suggested that economy considerations may be a more important factor for untrained individuals at higher relative power outputs (57). They speculated that perhaps some combination of several factors may be integrated for cadence selection and that a tradeoff may exist in that the reduction of other factors, such a pedal forces, joint moments, or muscle stresses, are offset by an increase in aerobic demand at higher cadences. It may be argued, by Takaishi et al.'s (87,88) results, that at intensities significantly above anaerobic threshold, neuromuscular fatigue is also an important determinant of preferred cadence.

A limitation of Marsh and Martin's study is that trained individuals did not ride at similar relative intensities to untrained individuals; therefore, no evidence exists that trained individuals would respond in the same nature as untrained. Whether a similar tradeoff between aerobic demand and other variables would be observed is questionable. Marsh and Martin (57) and Takaishi et al. (89) both argued that the similar preferred cadences observed between trained cyclists and trained non-cyclists may be explained by the similarities in mode of training. High repetition, relatively low forces, and relatively fast joint angular velocities are shared between running and cycling. This pattern of stress may be responsible for adaptations in the force-velocity properties of the lower extremity muscles. Such adaptations may act to minimize cadence effects on aerobic demand and reduce the speculated trade-off proposed for untrained individuals.

In an earlier investigation, Marsh and Martin (59) tested the hypothesis that electromyographic activity of selected lower limb muscles was minimized at subjects' preferred cadence. They found little evidence of systematic changes in EMG over varying cadences, with only gastrocnemius being substantially and systematically altered. Secondary to their null findings, they proposed an alternate hypothesis that EMG changes over cadence may parallel those of aerobic demand. They showed that a global representation of EMG, the sum of the normalized average EMG for 5 muscles of the lower limb, matched well with the average aerobic demand over cadences ranging from 50 to 110 RPM. Both measures were significantly lower than subjects' preferred cadence. In light of the close match between a global EMG measure and aerobic demand, it could also be hypothesized that a measure of global

neuromuscular fatigue may parallel the $\dot{V}O_2$ SC as cadence was manipulated. These data are displayed in Figure 10. In contrast to the hypothesized relationshipship, the global neuromuscular fatigue displayed an inverse relationshipship with the $\dot{V}O_2$ SC over cadence, with both the global 55 RPM and 100 RPM conditions having negative overall responses. The significance of the negative global neuromuscular fatigue for certain cadence conditions or of the overall global neuromuscular fatigue pattern over cadence is unknown, but it is apparent from the present results that no direct relationshipship exists between a global neuromuscular fatigue response and the $\dot{V}O_2$ SC.

It has been suggested that RPE is closely associated with feelings of strain in working muscles (69). While the present study did not evaluate RPE, upon completion of all conditions each subject was asked which cadence they preferred during the criterion exercise. Individual subject data, shown in Table II in the results section, indicates that no subject chose the 55 RPM condition even though $\dot{V}O_2$ values were generally lower during this cadence condition. The failure to elicit systematic neuromuscular fatigue during the criterion exercise sessions in the present study did not support the hypothesis that subjects are sensing a rate of fatigue, possibly through an indirect measure of the excitatory input or neural drive sent to a muscle, reflecting both the number of active motor units and their discharge rates. A majority of subjects did prefer the cadence condition (85 RPM) that most closely match their preferred cadence (89.3 RPM); however, some determinant other than neuromuscular fatigue was incorporated into this selection.

*iv. physiological variables*

The exercise intensity used in the present study was sufficient to evoke a slow component during Phase 3 of $\dot{V}O_2$ kinetics. While the average $\dot{V}O_2$ SC did not differ over cadence conditions, there was a significant quadratic trend indicating cadence may have some affect on the $\dot{V}O_2$ SC. This finding is similar to other results reported in the literature (7,32,42). These results support Hypothesis 3; the slow component of $\dot{V}O_2$ displays a quadratic relationshipship to cadence.

The precise cause of the SC of $\dot{V}O_2$ is not fully understood. In light of recent publications which refute proposed mechanisms for the slow component of $\dot{V}O_2$, such as

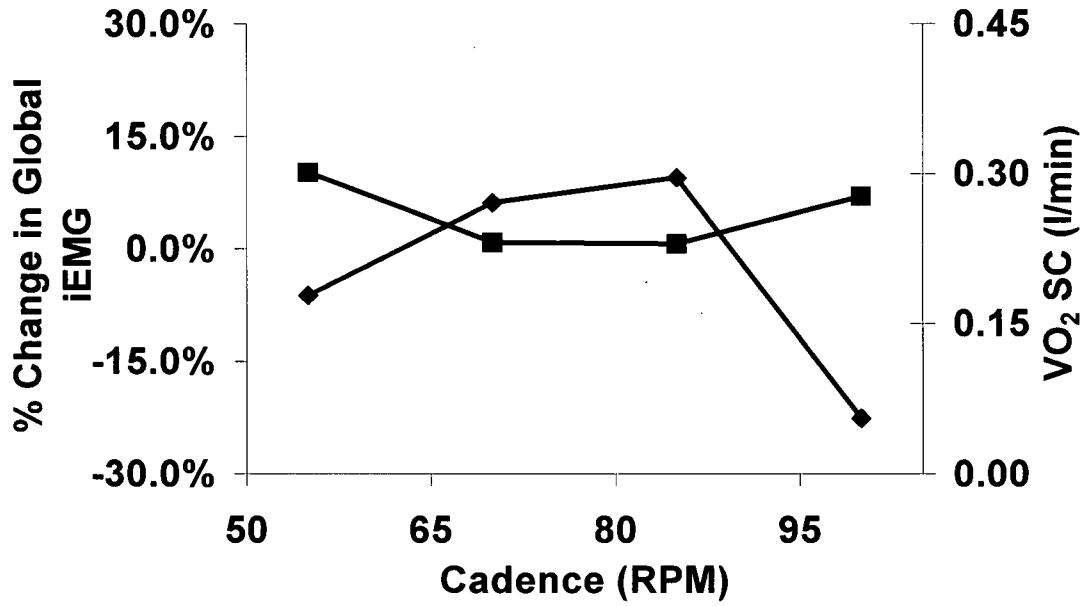


Figure 10 – $\dot{V}O_2$ SC (■) and global neuromuscular fatigue (♦) response to cadence. Global neuromuscular fatigue calculated as the sum of percent difference between 4$^{th}$ minute and 15$^{th}$ minute average iEMG traces for VL, BF, GM, SOL, GAS response to cadence.

increases in blood lactate concentrations, increased cost of ventilation, increases in core and muscle temperature, and increases in plasma epinephrine levels, many researchers have suggested that motor unit recruitment patterns may be responsible for observed increases in $\dot{V}O_2$ seen during heavy exercise (7,29, 34,35,73,75, 79 ,102). The idea that fiber type recruitment patterns may account for the slow component of $\dot{V}O_2$ is an attractive hypothesis. It provides a mechanistic basis for many of the parameters associated with this metabolic response including: temporal profile, lactate-$\dot{V}O_2$ relationshipship, and training adaptations. The central paradigm is that during heavy intensity exercise, where a slow component of $\dot{V}O_2$ is recognized, there is a serial recruitment of less efficient Type II fibers to replace the fatiguing Type I that were initially recruited.

It has been established that Type II fibers are less efficient energetically than Type I fibers; the high energy phosphate produced per molecule of oxygen for Type II fibers is less than for Type I. The overall phosphate/oxygen (P/O) ratio of fast glycolytic fibers (Type IIb) is 18% lower than Type I in isolated mitochondria (73). The lower P/O ratio is likely due to heavier reliance on the α-glycerophosphate shuttle in Type II fibers rather than the malate-aspartate shuttle which is primarily used in Type I fibers (24). Because mitochondria are impermeable to NADH, the normal pathways for transferring reducing equivalents from cytoplasmic NADH formed during glycolysis into the mitochondria is the α-glycerophosphate and malate-aspartate shuttles. When the α-glycerophosphate shuttle is used, $H^+$ is transferred to intramitochondrial FAD rather than to the malate-aspartate linked NAD, thus bypassing one phosphorylative site (97). In addition, Type II fibers have 5-10 times greater calcium pump activity, which is ATP driven, and also produce between 50% and 600% more heat per unit of tension development (24).

While the majority of people have approximately 50% Type I and 50% Type II fibers, endurance athletes tend to have a higher percentage of Type I fibers and sprinters tend to have a higher percentage of Type II fibers (39). Percentage of Type I fibers have been correlated with efficiency in both constant workload cycling and a novel leg extension task (22,23). Such work has led to speculation that training may induce conversion of muscle fiber types from Type I to Type II or vice versa. There is no direct evidence which indicates that such a phenomenon occurs, rather a high percentage of one fiber type may precipitate selection of a particular sport. However, in highly endurance trained men it is often impossible to detect any Type IIb fibers which may be a result of conversion of Type IIb fibers to Type IIa (45). Recently, it has been

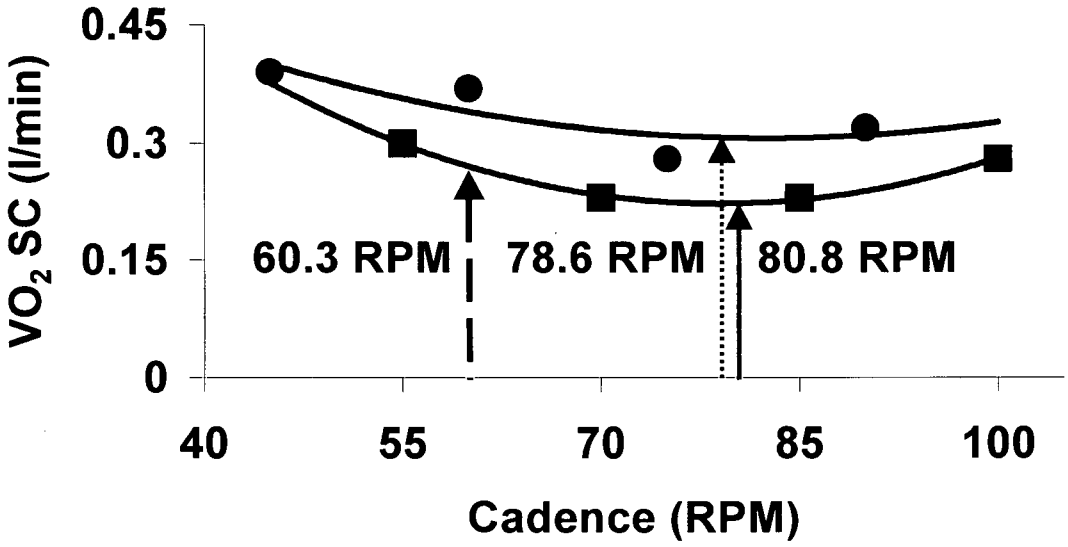


Figure 11 – $\dot{V}O_2$ SC over cadence conditions. ■ - present study; • - Barstow et al. (1996). ——▶ - calculated minimum SC for present study; ·····▶ - calculated minimum SC from Barstow et al. (1996); – – –▶ - calculated minimum $\dot{V}O_2$ over 4th minute, present study.

reported that the $\dot{V}O_2$ kinetic response to heavy exercise is related to percentage of Type I fibers (7). Percentage of Type I fibers were found to be significantly correlated with both the gain of the primary fast component and with the relative size of the $\dot{V}O_2$ SC. Subjects with a larger percentage of Type I fibers had a greater amplitude for the fast component and a reduced contribution of the SC of $\dot{V}O_2$. These results are similar to those found after endurance training (15).

It has been well documented that there is a reduction in the slow component of $\dot{V}O_2$ after an endurance training protocol (15,35,101). However, Casaburi et al. (15) are the only investigators who have published definitive results concerning the effects of endurance training on the slow component. Subjects followed an 8 week training program with supra-LT workloads resulting in a significant reduction in $\dot{V}O_2$ SC. However, results from an elderly exercise study may indicate that training intensity need not be high in order to diminish the slow component (35). As well, a significant attenuation in the slow component may be apparent after just 2 weeks of training (35). The above mentioned papers all speculated on the possible cause of the slow component, drawing correlationships between such factors as $[L^+]$ or ventilation and the slow component (101). To date, however, there has been no publication attempting to establish a temporal relationship between specific adaptations of skeletal muscle and attenuation of the slow component of $\dot{V}O_2$ after endurance training. Associated with endurance training are increases in mitochondrial content (both size and number), respiratory capacity, mitochondrial enzymes, ability to oxidize fatty acids, ketones, and pyruvate, and changes in mitochondrial composition (45). There is some diversity in the specific alterations in mitochondrial enzymes in response to endurance training, with enzymes increasing between 30% and 300% or not at all (45). One such enzyme in which no appreciable increases are seen in α-glycerophosphate dehydrogenase while large increases are seen in the enzymes of the malate-aspartate shuttle. It has been speculated that altered proportions between the malate-aspartate and α-glycerophosphate shuttles may account for the slow component of $\dot{V}O_2$ (97). Whipp reasoned that increased reliance on the α-glycerophosphate shuttle may be due to saturation of the malate-aspartate shuttle or dominant use of Type IIb fibers. In light of enzymatic changes which occur with endurance training in the malate-aspartate shuttle but not the α-glycerophosphate shuttle, it is probable that training induced changes are reflective of a greater reliance on Type I fibers due to increases in their respiratory capacity.

In basing arguments for the motor unit recruitment theory, several authors have cited glycogen depletion patterns as evidence for increased Type II recruitment eliciting a slow component of $\dot{V}O_2$. A study by Ahlquist et al. examining the effect of pedaling frequency on glycogen depletion rates in Type I and Type II quadriceps muscle fibers during heavy exercise found that cycling at 50 RPM rather than 100 RPM resulted in greater Type II fiber glycogen depletion (1). Subjects rode at approximately 85% of their $\dot{V}O_{2\,max}$ for 30 minutes at both 50 RPM and 100 RPM on separate days. Glycogen depletion was quantified via microphotometry of periodic acid-Schiff stain. Ahlquist et al. proposed that their result indicated that Type II fiber were recruited not only for high velocity work but also for low velocity, high tension work. The main limitation to this study is the selection of only 2 cadences for study. The cadences picked represent the ends of the spectrum of cadences studied and do not allow a relationshipship between glycogen depletion and cadence to be established. It remains presently unclear how this data fits into a logical argument concerning the cause of the $\dot{V}O_2$ SC. Ahlquist et al.'s work does not provide any direct evidence of serial recruitment of Type II fibers, rather it shows only that Type II fibers were active for some portion of the exercise duration.

It is likely that both Type I and Type II fibers are initially recruited during heavy exercise (73). It has been speculated that the slow component of $\dot{V}O_2$, with a separate time constant from the initial rise in $\dot{V}O_2$ may be an artifact of the slower kinetics of Type II fibers (73). The time constant for $\dot{V}O_2$ rise in mouse extensor digitorum longus muscle, which is primarily Type II fibers, is approximately 138 seconds while the time constant for the mouse soleus muscle, primarily Type I, is only 36 seconds (24). These time constants, measured in isolated muscle, correlate well with time constants seen for pulmonary $\dot{V}O_2$ kinetics during heavy exercise. The delayed onset of the $\dot{V}O_2$ SC suggests that it is not a fundamental component of the initial response to exercise. If true, it remains unclear how differences in fiber type percentage would affect the gain of the primary fast component of $\dot{V}O_2$ kinetics (7), and if a relationshipship exists between this finding and the concomitant decrease in $\dot{V}O_2$ SC observed with increased Type I fibers.

In Coyle et al.'s work (22), correlating percentage of Type I fibers to cycling efficiency, subjects rode at 80 RPM. This cadence was thought to represent a contraction velocity for the VL that was close to the velocity of peak efficiency of Type I fibers. This optimal rate of pedaling was calculated using data from *in vitro* experiments and extrapolating to a *in vivo*

model. He estimated the velocities that are predicted to elicit peak efficiency in Type I compared with Type II fibers and also the relative shortening velocity of the VL at 80 RPM. Using published values for $V_{max}$ of Type I and Type II fibers in human deltoid muscle (30), peak efficiency was estimated as 1/3 of $V_{max}$. This value was approximately 1 fiberlength/sec. He then determined that a cadence of 80 RPM would require the VL to contract at approximately 200°/sec, which corresponds to approximately 1.2 fiberlengths/sec. Coyle et al. cautioned that his calculations were speculative; however, they presented a working hypothesis.

The incorporation of Coyle et al.'s work (22) in the support of serial recruitment of Type II fibers as the mechanistic basis of the $\dot{V}O_2$ SC may gain more validity in light of recent findings that percentage of Type I fibers were significantly correlated to amplitude of $\dot{V}O_2$ SC (7). If a direct and casual relationshipship exists between percentage of Type I fibers and the $\dot{V}O_2$ SC then manipulation of contraction velocity should elicit changes in the amplitude of the $\dot{V}O_2$ SC. A significant amplitude difference for the $\dot{V}O_2$ SC has been reported between 50 and 100 RPM for supra-lactate cycle exercise 18 minutes in duration (32). Barstow et al. (7) also examined the possible effect of cadence on the $\dot{V}O_2$ SC and reported no systematic differences between cadences ranging from 45 to 90 RPM. However, they do report a significant difference between the $\dot{V}O_2$ SC at 45 RPM and that at 75 RPM. Close examination of their data reveals that a quadratic relationshipship between cadence and $\dot{V}O_2$ SC ($R^2 = 0.85$). Figure 11 plots data from both the present study and Barstow et al.'s. The calculated minima for $\dot{V}O_2$ SC for both data sets match closely, even considering there is significant differences in methodology between the studies. Barstow et al. (7) rode subject's at a supra-lactate exercise intensity, equal to ½ of the difference between LT and $\dot{V}O_2$ max, and each cadence condition lasted only 8 minutes. While within the same heavy exercise domain, Figure 11 demonstrates that the higher exercise intensity used by Barstow et al. elicits higher $\dot{V}O_2$ SC's across all cadence conditions, yet the cadence effect appears quite robust. The calculated minima from both studies, approximately 80 RPM, equals the estimated contraction velocity for peak efficiency of Type I fibers (22).

An unexpected finding in the present study was the seemingly close link between the cadence- $\dot{V}O_2$ SC relationshipship and the cadence- $\Delta$VE relationshipship. It has been previously shown that cost of increased ventilation only contributes approximately 23% to the overall $\dot{V}O_2$ SC (34), yet the calculated minimum for $\Delta$VE over cadence occurred at 78.1 RPM, very close to

80.8 RPM, the minimum calculated for the $\dot{V}O_2$ SC. Because the present study used a mixing chamber for measurement of $\dot{V}O_2$, it is not possible to compare the kinetic responses of these 2 variables. A progressive increase in VE during prolonged exercise has previously been directly related to an increase in rate of ventilation (26), and may also be influenced by arterial [$La^+$] (26), body temperature (26), circulating catecholamines (67), and most obviously systemic $O_2$ transport. However, none of these variables, except $\dot{V}O_2$, were measured in the present study. Irrespective of the underlying mechanisms, the present findings suggest there is a close relationshipship between $\Delta$VE and the $\dot{V}O_2$ SC over varying cadence during heavy exercise.

Unlike previously published data (20), which reported a consistently lower HR at 80 RPM for exercise at 85% $\dot{V}O_2$ max lasting 20 minutes in duration at 85% $\dot{V}O_2$ max, the present study showed no significant HR-cadence relationship at the end of the exercise period. While, the 4$^{th}$ minute average HR displayed a significant quadratic relationshipship, no $\Delta$HR-cadence relationshipship was observed. Regulation of HR during exercise is governed by at least 3 physiological mechanisms: the Frank-Starling law of the heart, the autonomic nervous system, and catecholamines (90).While the initial rise in HR is primarily due to a withdrawal of vagal tone, catecholamines, such as epinephrine and norepinephrine, are largely responsible for maintaining tachycardia during steady-state exercise. Plasma epinephrine and norepinephrine levels have no significant relationshipship to cadence during heavy exercise (20). Cardiovascular drift may also account for a slow rise in HR during continuous exercise where significant dehydration occurs, due to a gradual decrease in stroke volume (42). However, in the present study, it is unlikely that cardiovascular drift played an important role in HR regulation since exercise duration was only 15 minutes. In light of the more "central command" (42) regulating HR response during heavy exercise, it may not be surprising that $\Delta$HR did not display a relationshipship to cadence.

*v. synopsis*

It is not known why the present experimental conditions failed to elicit a neuromuscular fatigue response. While not fully understood, neuromuscular fatigue has been previously shown under conditions similar to the present study (87,88). It is possible that the exercise intensity in the present study was not high enough to elicit a neuromuscular fatigue response. While the end exercise intensity between subject group in the present study and in Takaishi et al. (88) was

similar, the initial exercise intensities differed. Regardless, it is clear that subjects in the present study did not incorporate information regarding the rate of neuromuscular fatigue into their selection of preferred cadence.

The present study did show a significant quadratic relationshipship between $\dot{V}O_2$ SC and cadence, with a minimization close to the estimated peak efficiency of Type I fibers. At present, there is not enough evidence to establish an underlying mechanism for either neuromuscular fatigue or the slow component of oxygen uptake. There exist fundamental limitations in the proposed etiologies of each. The present theorem of serial recruitment of Type II fibers owing to some loss in contractility of Type I fibers, possibly due to glycogen depletion or accumulation of $H^+$ (73), is questionable. The temporal characteristics of neuromuscular fatigue and $\dot{V}O_2$ SC are observable within the 1st 3 minutes of exercise. It is difficult to conceive that the Type I fiber population becomes glycogen depleted within the first few minutes of exercise. In fact, Vollestad et al. (94) have shown that there is no appreciable glycogen depletion or $H^+$ accumulation within the first few minutes of fatiguing exercise, and speculate that excitation/contraction coupling failure is primary cause of fatigue. Regardless, the present study failed to correlate neuromuscular fatigue and the $\dot{V}O_2$ SC over varying cadence conditions.

## 5. Conclusions

1. The rate of neuromuscular fatigue was not altered by speed of movement under the present experimental conditions. The present study failed to replicate the work of Takaishi et al. (88). No significant neuromuscular fatigue was observed for any of the muscle groups measured over any of the cadence conditions; therefore, no minimum rate of neuromuscular fatigue was found at a selected pedal rate.

2. The cadence eliciting the lowest metabolic cost, termed optimal cadence, equaled 60.3 RPM in the present study. Because no relationshipship between neuromuscular fatigue and cadence was established, no comparison between optimal cadence and neuromuscular fatigue can be made.

3. No significant difference between the $\dot{V}O_2$ SC over cadence conditions was found; however, a significant quadratic relationshipship was observed with a minimum occuring at 80.8 RPM.

4. The failure to elicit neuromuscular fatigue in the present study precludes any comparisons of the slow component of oxygen uptake and neuromuscular fatigue. No relationshipship between these two measures was observed.

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# Appendix I – Literature Review

*i. optimal and preferred cadence*

In recent years, much information has been published regarding the apparent discrepancy between the preferred and the most economical pedaling frequency chosen by cyclists. This apparent paradox is based on the grounds that cyclists prefer to ride at a significantly higher cadence than is metabolically most economical (57,58). These findings are at odds with research conducted on preferred stride length/ cadence during walking and running. For such activities, the preferred stride length/ cadence combination generally coincides with that which is most economical (17,21). Researchers thus feel that there is some determinant for cadence selection other than lowest metabolic cost. Numerous studies have addressed factors other than $\dot{V}O_2$ which may contribute to cadence selection including rate of perceived exertion (54,69), pedal forces (70), muscle blood flow (41), muscular or mechanical efficiency (31,80,98), force-velocity curves (82), fitness level (12,57), glycogen depletion rates and fibre type recruitment (1,38,86), muscular activity measured by EMG (40,59), and fatigue measured by EMG (87,88). Confounding this issue, it has been noted that cyclists employ a significantly higher pedaling frequency in both training and racing than recreational or non- cyclists (43,76). From these findings, researchers have attempted to explain the differences found as an adaptation acquired from cycling training (89).

Early research on the effect of pedaling speed on oxygen uptake was conducted by Garry et al. in 1931 (37). His work found an optimal gross efficiency at a cadence of 52 RPM, and that gross efficiency rose both above and below this pedaling rate. He also stated that the fundamental difference between trained and untrained subjects is the cost of the unloaded movement. Trained cyclists were able to better coordinate movement and stabilize the body with use of fewer accessory muscles. Seabury et al. were the first researchers to conduct a systematic study assessing the influence of both pedaling rate and power output (77). Their study was limited in subject size (n=3); however, each subject rode at cadences between 30 and 120 RPM at 4 power outputs, ranging between 0 and approximately 200 Watts. They determined optimal pedaling rates to be between 42 and 62 RPM for power outputs between 40.8 and 326.8 Watts. They observed that optimal pedaling rate was positively related to power output, higher power outputs yielded higher optimal pedaling rates. This relationshipship between power output and

optimal cadence resembles a logarithmic function, with a leveling off at higher power outputs. It was suggested that the existence of the most efficient pedaling rate was due to both a reduction of load on each fibre per contraction and a reduction in the total number of contracting fibres. If either the internal friction could be reduced or the force with which each fibre could contract could increase, then the most efficient pedaling rate would increase. Also examining the relationship between $\dot{V}O_2$, pedaling frequency and power output, Coast and Welch (1985) rode five trained cyclists at pedal rates of 40 through 120 RPM at power outputs between 100 and 300 Watts (19). They found optimal pedal rates between 60 and 80 RPM, with a linear relationshipship to power output. The differences between their results and those of Seabury et al. (1977) were explained in terms of acquired skill in their trained subjects which lead to greater efficiency at higher pedal rates. They felt that the skill of a rider was positively correlated with optimal cadence.

Addressing the apparent discrepancy between a metabolically optimal cadence and the preferred or self-selected cadence of trained cyclists, Hagberg et al. analyzed seven road-racing cyclists riding at 80% of their $\dot{V}O_{2\ max}$ at various cadences (43). He found preferred pedaling rates between 72 and 102 RPM with an average of 91 RPM. They examined various physiological variables such a $\dot{V}O_2$, heart rate, RER, rate of perceived exertion (RPE), minute ventilation ($V_E$), and blood lactate. Their general results indicated that "competitive cyclists when tested on their road-racing bicycles are most efficient at an average pedaling rate of 91 RPM...", pp.451. They hypothesized that the sharp increase of most physiological variables against various pedaling frequencies may be the result of increased recruitment of FT muscle fibres, the rise in external work, and the use of various muscles to stabilize the trunk and eliminate extraneous motion.

Most early studies of optimal and preferred cadence involved short bouts of exercise at each cadence, allowing only enough time for physiological value to settle before measurement. Coast et al., in 1986, were the first group to experiment using longer duration rides at $\dot{V}O_2$ levels equally those seen in race conditions. Trained bicycle racers rode at 85% $\dot{V}O_{2\ max}$ for each of 5 cadences, 40, 60, 80, 100, and 120 RPM. Each ride lasted between 20-30 minutes, approximately the time needed to complete a 16 km timetrial. They found minimal values for HR, RPE, and blood lactate all occurred at 80 RPM. In their concluding remarks they stated that

the discrepancy between metabolically optimal and preferred cadence may be as the result of the need for rapid acceleration in bicycle races. Such acceleration is very difficult at slow pedal rates in high gears, thus the need to spin at higher cadences may outweigh the disadvantage of using a less economical pedaling rate.

Rate of perceived exertion has been explored as a possible explanation for the discrepancy between optimal and self-selected cadence. The general hypothesis was that RPE would be minimized at the preferred cadence of cyclists, coincident with the minimization of some other variable. Pandolf and Noble (1973) found that RPE was inversely related to pedaling frequency up to 80 RPM. They felt that $\dot{V}O_2$ was not the major factor in the subjective rating of work stress. Instead, they attributed RPE responses to local factors associated with feelings of strain in the working muscles and joints. Lollgen et al. conducted a study assessing RPE along with muscle metabolites and pedal forces (54). They found RPE to be lowest at between 60 and 80 RPM for workloads ranging from 0 to 100% $\dot{V}O_2$. There was a general lack of relationshipship between central factors measured and RPE. The authors did feel that peripheral muscle receptors may play an important role and suggested that these mechanoreceptors may code for both magnitude of tension and rate of development of tension.

The application of force to the pedal may be an important indicator of preferred cadence. Patterson and Moreno, 1990, found that as pedaling frequency is increased the resultant force applied to the pedal decreases non-proportionally, with a leveling off between 90 and 100 RPM (70). As well, the orientation of force application is also important. As cadence increases, the index of effectiveness, a measure of the proportion of the resultant force which is applied perpendicular to the crank thus providing positive impulse, decreases linearly. Patterson and Moreno felt that cyclists may increase cadence until there is no further reduction in the resultant force applied to the pedals. This pedal rate would minimize fatigue by operating at a lower percentage of the cyclist's maximum strength with only a relatively small increase in oxygen uptake. Lollgen et al.'s RPE study also found that resultant pedal force decreased to a level near 100 RPM (54). They also found that pedal forces were always less than 30% of maximum voluntary contraction, even cycling at 40 RPM at 100% $\dot{V}O_2$.

When addressing the question of why there exists a discrepancy between a cyclist's preferred and metabolically optimal cadence, some researchers have questioned the recruitment patterns of muscle fibers in working muscles of the lower limb. Early work by Gollnick et al., 1974, found that the total force exerted in each pedal thrust had no effect on the pattern of glycogen depletion (38). For all workloads below 100% $\dot{V}O_{2\,max}$ slow twitch (ST) fibres were preferentially recruited, independent of contraction velocity. In a study by Y. Sukuki, subjects were selected on the basis of having either a very high or very low percentage of ST fibres (86). He found that the delta efficiency was lower for the ST fibre group at higher cadences. This was attributed to the low velocities at which ST fibres shorten most economically. More recently, Ahlquist et al., 1992, have also looked at the effect of pedaling frequency on the glycogen depletion rates of ST and FT fibres in the quadriceps muscle during cycling (1). In contrast to Gollnick et al., Ahlquist et al. found greater FT fibre depletion at a lower cadence. They suggested that tension development, and not contraction velocity per se, was the determinant in FT fibre recruitment when metabolic cost is held constant.

In an attempt to gain insight into contraction patterns of lower limb musculature, both in strength and timing, electromyography (EMG) has been implemented in several studies of cycling. It was hoped that muscle activity patterns, such as onset/offset of contraction, amplitude of contraction, and timing of peak contraction, may yield information as to why cyclists choose a particular cadence. Goto et al., 1976, were the first to study the integrated EMG (iEMG) of leg muscles during pedaling at various frequencies (40). This group was expanding on previous work by Bigland-Ritchie and Woods, 1974, in which a linear relationshipship was found between workload and iEMG (11). Goto et al. found curvilinear relationships for the gluteus maximus and tibialis anterior muscles and positive linear relationships for vastus lateralis and gastrocnemius muscles with varying pedaling frequencies. In general, they stated that iEMG was more affected by rate of pedaling than by work load. More recently, Marsh and Martin, 1995, examined the relationshipship between cadence and lower extremity EMG (59). They investigated such variables as average EMG amplitude, peak EMG amplitude and timing of peak EMG amplitude. Of the muscles measured, only EMG from the gastrocnemius muscle was found to have a substantial and systematic change over varying pedaling frequencies. This relationshipship was linear. Rectus femoris and vastus lateralis EMG both displayed small but significant relationshipships with cadence, quadratically and linearly respectively. Their conclusions were that EMG of lower extremity muscles is not minimized at the preferred

cadence of subjects, although the total contribution of muscle activity, or global EMG response, did follow the same shape of the aerobic response of the subjects over cadence.

An investigative technique used to quantify neuromuscular fatigue has recently been used to assess pedaling at different frequencies for prolonged periods (87,88). Neuromuscular fatigue is a gradual increase in the measurable electrical activity within a muscle, speculated to be due to a progressive recruitment of additional motor units and an increase in the firing rates of those motor units already recruited (87). The increase in integrated EMG (iEMG) as a function of time is used as a criterion to assess neuromuscular fatigue. Takaishi et al. have published two papers assessing neuromuscular fatigue over varying pedaling frequencies in both cyclists and non-cyclists. Takaishi et al. found that for both cyclists and non-cyclists, the pedaling rate which resulted in minimized neuromuscular fatigue was coincident with the subjects' preferred cadence, at 70 RPM and between 80 and 90 RPM respectively. The authors also speculated that there may be a direct relationshipship between neuromuscular fatigue and the slow component of $\dot{V}O_2$ during prolonged exercise (88). The major limitation with both of their studies is that EMG was only measured in the vastus lateralis muscle while generalizations were made to all working muscles of the lower limb.

In addressing the issue of preferred cadence, it has been widely speculated that cyclists undergo some adaptation following high intensity training which in turn allows them to pedal more effectively or efficiently at higher cadences (12,19,23,37,43,77,89). Most of the literature, however, fails to address any reasons for adaptation or consequences thereof for higher pedaling rates. Papers which do provide some explanation of adaptations, cite theories ranging from increases in ST fiber composition to increased index of effectiveness and lowered muscle activation at higher cadences. Investigating the application of effective force to the pedal, Coyle et al. found that the peak effective force for elite-national level cyclists was higher than that of good-state class cyclists (23). As a consequence of lower peak effective force application, the good-state class cyclists had a higher index of effectiveness throughout the pedal revolution. In terms of performance, the elite-national class cyclists were capable of maintaining a higher absolute power output; however, index of effectiveness was not positively correlated with cycling performance. Takaishi et al. found that the peak normal pedal force for cyclists was lower than for non-cyclists (89). They felt that cyclists acquire skilled performance related to efficient exertion of leg force by the knee extensors from prolonged higher cadence pedaling.

They also base arguments of pedaling skill acquisition on differences in normalized iEMG between cyclists and non-cyclists. Non-cyclists required greater activity in the vastus lateralis and vastus medialis at cadences higher than 90 RPM while cyclists had increased biceps femoris activity. From their EMG findings, they felt that the muscle activity patterns of cyclists indicated that positive pedal lifting was being performed to alleviate the load needed in pushing down on the opposite pedal. In contrast to Takaishi et al.'s findings, Marsh and Martin, 1995, found that there were no significant differences in muscle activity patterns between cyclists and non-cyclists (59). It has also been observed that cyclists do not have a positive index of effectiveness during the recovery phase of the pedal cycle; even national level pursuit riders do not "pull up" during the recovery phase (16,53).

Examining changes in muscle composition, Coyle et al. has published two papers dealing with the percentage of ST fibers with lower limb musculature (23,22). His first paper was descriptive in nature, and stated that elite-national level cyclists possessed a greater percentage of ST fibers in their vastus lateralis than good-state class cyclists (23). Also, percentage of ST fibers in vastus lateralis was highly correlated with the number of years performing endurance training with the legs; however, it was not possible to determine whether percent ST fibers actually increased in these cyclists as training duration progressed or whether those cyclists with a higher percentage of ST fibers continued to train and race for more years. His second paper dealt with muscular efficiency in cycling and the relationship to percentage of ST fibers (22). He found that the percentage of ST fibres was positively correlated with muscular efficiency for both cycling tasks and novel leg extension tasks. He based his arguments examining velocity of contraction, stating that for each of the tasks, cycling and leg extension, the muscles in the vastus lateralis were contracting closer to the velocity of peak efficiency for ST fibres compared to FT fibres. Their cycling tasks took place at a cadence of 80 RPM while their leg extension task took place at 200°/sec. These papers contradict earlier findings by Burke et al., 1977, in which no differences in percentage of ST or FT fibers were found between national/international level cyclists and less competitive cyclists (13).

Recently, Marsh and Martin have argued that there are no specific adaptations resultant specifically from cycling training which may account for differences in self-selected cadence between cyclists and non-cyclists (58,57). They argue that the determinant for higher preferred cadences is aerobic fitness and not cycling training. There was no significant difference in

preferred cadence between cyclists and non-cyclists of similar aerobic fitness in their studies. They speculated that for less trained non-cyclists who cycled at a higher percentage of $\dot{V}O_{2\,max}$, preferred cadence may be lower to reduce aerobic demand. They also found that preferred cadence is not variable over power output or time.

*ii. fatigue*

Muscular fatigue is a complex and widely debated topic; there is still little consensus regarding the underlying mechanisms and cause. Fatigue can be defined as any exercise induced reduction in the ability to exert muscle force or power, regardless of whether or not the task can be sustained (10). Generally, fatigue can be divided into two broad categories, central and peripheral. Central fatigue is a progressive exercise induced reduction in the voluntary activation of a muscle (36). Central fatigue is generally measured using maximal voluntary contractions with twitch interpolation (36,95). Generally, during prolonged isometric contractions a steady decline in measurable force is observed. This decline can be attributed to a diminution in voluntary drive, which can be restored by an additional extra effort (36). Peripheral fatigue broadly encompasses all sites of possible fatigue excluding failure of excitation of motorneurons. Included mechanisms are a failure of action potential propagation along the axon, inadequate presynaptic release of acetylcholine, insufficient depolarization of the postsynaptic membrane, failure of action potential propagation along the sarcolemma, failure of excitation-contraction coupling, and failure at the myofibular level (81).

It seems probable that all of the possible mechanisms of fatigue are intimately associated. It would be biologically unsound if force production always failed because of an impairment at one step in the chain (36). In stating this, however, varying exercise protocols are designed for the assessment of fatigue at different sites in the activation chain (95). For prolonged exercise protocols under aerobic conditions, a longer duration fatigue has been observed, also called low-frequency fatigue (61). Baker et al., 1993, found that for aerobic exercise lasting 20-25 minutes, the primary mechanism for long-lasting fatigue may be excitation-contraction (E-C) coupling (3). This type of fatigue is characterized by a faster decline and marked slower recovery in twitch force compared to tetanic force (3,95). E-C coupling refers to the sequence of events which occur in the muscle fiber between the generation of an action potential and the activation of the contractile elements (85). This sequence of events comprises the initiation and propagation of an

action potential along the sarcolemma and into the transverse tubular system (t-system), signal transmission from the t-system to the sarcoplasmic reticulum (SR) where $Ca^{2+}$ is stored, $Ca^{2+}$ release from the SR into the myoplasm and the rise in myoplasmic $[Ca^{2+}]$, and binding of $Ca^{2+}$ to the regulatory system and activation of the contractile apparatus. There are numerous events in E-C coupling which are altered by intense muscle fiber activity. These alterations may either individually or collectively lead to a potentiation of the contractile response (85).

### *iii.* $\dot{V}O_2$ *kinetics*

At the onset of exercise, there is an increase in pulmonary oxygen uptake in response to ATP demand in working muscles. This increase can generally be characterized into 3 phases (6). Phase 1 is due primarily to increases in cardiac output and thus pulmonary blood flow as well as changes in mixed venous $O_2$ content and lung gas stores. This phase represents the initial 15-25 seconds of exercise. Phase 2 is initiated by the arrival at the lung of venous blood from exercising muscles, representing augmented $O_2$ extraction and continued increase in pulmonary blood flow (34). It is probable that Phase 2 pulmonary $\dot{V}O_2$ changes are in direct relationship to changes in oxygen uptake at the exercising muscle. There is a temporal correspondence between changes in pulmonary $\dot{V}O_2$ and changes in phosocreatine in the working muscle. In addition, muscle $\dot{V}O_2$ kinetics, measured by leg blood flow, are similar to pulmonary $\dot{V}O_2$ kinetics (74). The time constant for Phase 2 may be dependent on the fitness level of subjects but invariant to work rate. At most intensities, Phase 3 reflects the achievement of steady-state $\dot{V}O_2$. At this phase, oxygen uptake increases as a linear function of work rate.

The linear relationshipship between $\dot{V}O_2$ and work is only defined for work rates at which there is no significant increase in lactate concentration in blood or muscle. Thus exercise domains are defined on this basis (34,102). Moderate exercise includes all exercise intensities which do not induce significant increases in blood lactate. The heavy exercise domain includes exercise intensities which elicit a significant increases in blood lactate but where there is an eventual plateauing of lactate values at some new, elevated level. Severe exercise includes all remaining sub-maximal and supra-maximal work rates in which $\dot{V}O_2$ rises inexorably until

attainment of $\dot{V}O_2$ max and/or cessation of exercise due to volitional fatigue. In this domain, no stabilization of blood lactate is achieved.

There are several proposed mechanisms accounting for the slow component of $\dot{V}O_2$ observed during heavy and severe exercise. These include the effects of lactate, epinephrine, cardiac and ventilatory work, temperature, and fiber type recruitment changes. In consideration of the postulated factors, it is necessary to acknowledge that approximately 86% of the pulmonary increases in $\dot{V}O_2$ can be accounted for by increases in leg $\dot{V}O_2$ (74). While this does not preclude any of the above proposed mechanisms completely, it does indicate that the majority of the slow component originates from the working muscles. The finding of peripheral origin of the slow component of $\dot{V}O_2$ does limit the extent to which to which central mechanism may contribute to the slow component. Such limited mechanisms include glycogen synthesis from lactate in the liver or non-working muscle, cardiovascular drift, cost of ventilation and core body temperature changes.

Lactate has received the largest amount of attention of all hypothesized mechanisms of the slow component of $\dot{V}O_2$. It has been reported that the temporal characteristics of the slow component appear to be related to the magnitude and time course of the increase in blood lactate (97). Both the onset and stabilization of the slow component are coincident with the onset of blood lactate as well as its stabilization. This relationship is also apparent when examining training effects. Training induces a lowering of both blood lactate values and in the slow component of $\dot{V}O_2$ (35). Wasserman et al. contend that it is not lactate, but the resultant lowering of pH, which initiates the slow component of $\dot{V}O_2$ (34,101). They suggest that a reduction in pH causes a shift of the oxyhemoglobin dissociation curve to the right, via the Bohr effect, thus causing greater unloading of $O_2$ at the muscle capillary. This would cause $\dot{V}O_2$ to rise. There is a fundamental flaw in this reasoning. Such an effect of pH may cause time constants to steady-state to be altered, thus decreasing the time it takes to reach steady-state $\dot{V}O_2$ but it cannot explain why $\dot{V}O_2$ rises above the projected level from the sub-$T_{lac}$ $\dot{V}O_2$-WR relationshipship. If the assumption is made that this $\dot{V}O_2$-WR relationship is valid, then this postulation is invalid. In addition, there are several lines of evidence indicating that lactate may not be the cause of the slow component of $\dot{V}O_2$. While generally, the correlationship between lactate and the slow component of $\dot{V}O_2$ is good, elevated blood lactate without a concomitant

increase in $\dot{V}O_2$ has been reported (34). As well, epinephrine infusion can elevate blood lactate significantly during exercise without any changes in $\dot{V}O_2$ (34). Perhaps the most significant finding is the lactate infusions into the arterial blood supply of electrically stimulated dog gastrocnemius muscle did not increase $\dot{V}O_2$ despite a significant increase in both muscle and blood lactate (75). It is probable that while the relationshipship between lactate and the slow component has a strong correlationship, it is not a casual effect. It is plausible that some common underlying mechanism gives rise to both processes.

The $\dot{V}O_2$ cost of respiratory muscle work undoubtedly influences pulmonary oxygen uptake during heavy exercise. To what extent cost of ventilation contributes is speculative. A recent review article quotes various published values for the cost of ventilation (34). Poole et al. estimated that 23% of the slow component of $\dot{V}O_2$ was attributable to an increase in ventilation. This combined with Poole's finding that increases in leg $\dot{V}O_2$ accounted for 86% of the slow component, reflects that ventilation probably accounts for only a small percentage of the total slow component. Another factor regarding cost of ventilation which has not been breached in the literature is an explanation of why a cost of ventilation would have a significant time constant and why increases are only seen during exercise in the heavy and severe domain. If the linear sub-$T_{lac}$ $\dot{V}O_2$-WR relationship can be extrapolated to the respiratory muscles then why is there a delay in the onset of this additional cost of ventilation. The increase in cost of ventilation should be, and most likely is, immediately seen in the pulmonary $\dot{V}O_2$. In addition, the cost should be a linear function of WR, thereby having a graded effect which could be accounted for at all WR's from moderate to severe. Unless there is some recruitment of new or unused respiratory muscle coincident with heavy exercise, then a separate and distinguishable increase in pulmonary $\dot{V}O_2$ due to increased cost of ventilation should not be seen.

Core and muscle temperature have also been implicated to play a role in the slow component of $\dot{V}O_2$. Both core and muscle temperature increase during exercise and thus may contribute to the progressive rise of $\dot{V}O_2$ via the $Q_{10}$ effect. Increased temperature elevates $O_2$ consumption of isolated mitochondria and decreases the phosphorylation potential (ADP/O ratio) (52). Elevated muscle temperature may speed $\dot{V}O_2$ kinetics by speeding the rate limiting reactions associated with oxidative phosphorylation and by shifting the oxyhemoglobin dissociation curve to the right via the Bohr effect. This, plus any muscle vasodilation associated

with increased temperature, may facilitate $O_2$ delivery during transition to exercise. As pointed out above in rebut to Wasserman et al.'s (34) argument for the role of the Bohr effect in the slow component of $\dot{V}O_2$, such a shift would only alter $\dot{V}O_2$ time constants and not the magnitude of measured pulmonary $\dot{V}O_2$. As well, increased muscle temperatures have been observed without a concomitant increase in pulmonary $\dot{V}O_2$ (52,74). Increases in muscle temperature have no appreciable effect on the fast component (time constants) or the slow component of $\dot{V}O_2$ during heavy exercise.

Epinenphrine has been elucidated to be a possible mediator of the slow component of $\dot{V}O_2$. Plasma catecholamine levels increase appreciably during exercise at work rates that elicit a pronounced slow component (35). The mechanism by which epinephrine has an effect on $\dot{V}O_2$ may be action via $beta_2$ receptors (50). Epinephrine infusion has been reported to increase resting $\dot{V}O_2$ in humans (84), as well as increase the maximal $\dot{V}O_2$ in isolated canine skeletal muscle (83). Epinephrine infusion has been shown to have no effect on $\dot{V}O_2$ during moderate exercise (48) or heavy exercise (33). Due to exogenous plasma catecholamines' effect on $\dot{V}O_2$, infusion of epinephrine may not elicit any additional changes past what is naturally present. Kalis et al. reported that propanol minimized the slow increase in $\dot{V}O_2$ seen during prolonged moderate exercise (50). For exercise above $T_{lac}$, in the heavy domain, Gaesser beta-adrenergic blockade with propanol has no influence on the slow component but did alter exercise onset $\dot{V}O_2$ kinetics (time constants) (35).

### *iv. Delimitations of electromyography*

The use of surface electromyography in the assessment of fatigue has been widely debated in recent years. There are a number of true limitations in using EMG to measure muscular fatigue which are worthwhile in addressing. It is imperative to have a good understanding on the underlying principles of EMG to gain an understanding of its uses and limitations. When an action potential is propagated along a motorneuron, it innervates all muscle fibers in a particular motor unit. Transmission of the signal takes place across the motor end plate, depolarizing the postsynaptic membrane. Depolarization propagates in both directions along the muscle fiber, accompanied by a movement of ions which generates an electromagnetic

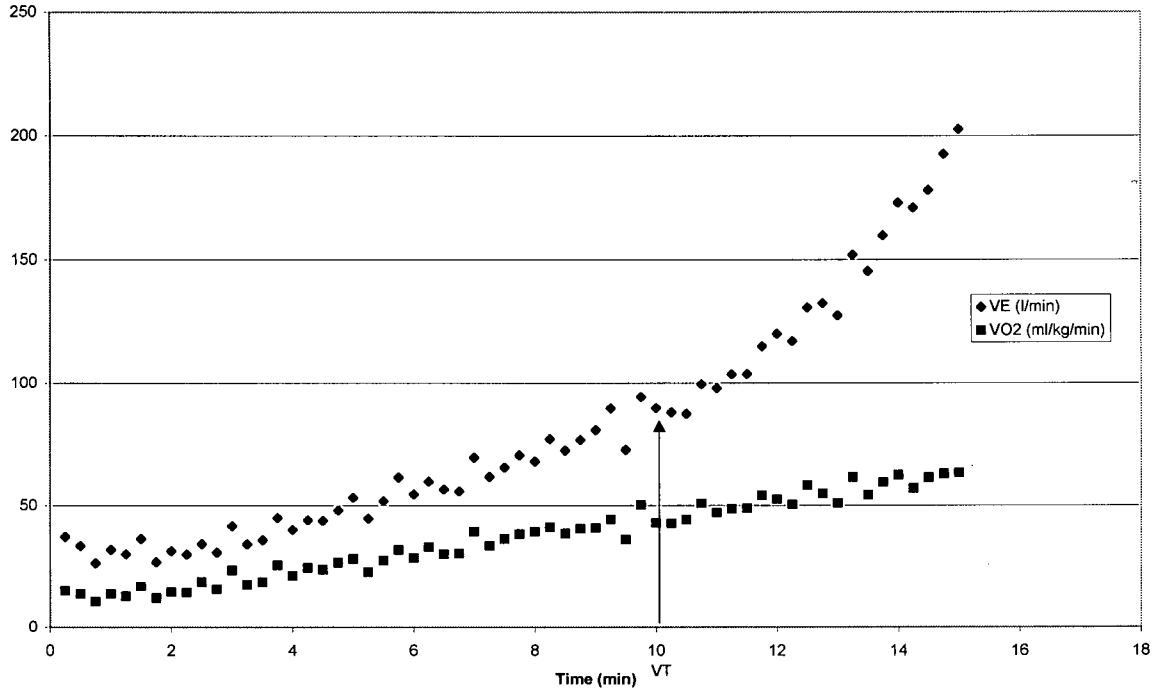
field in the vicinity of the muscle fibers. A recording electrode, placed on the surface of the skin, may detect this potential.

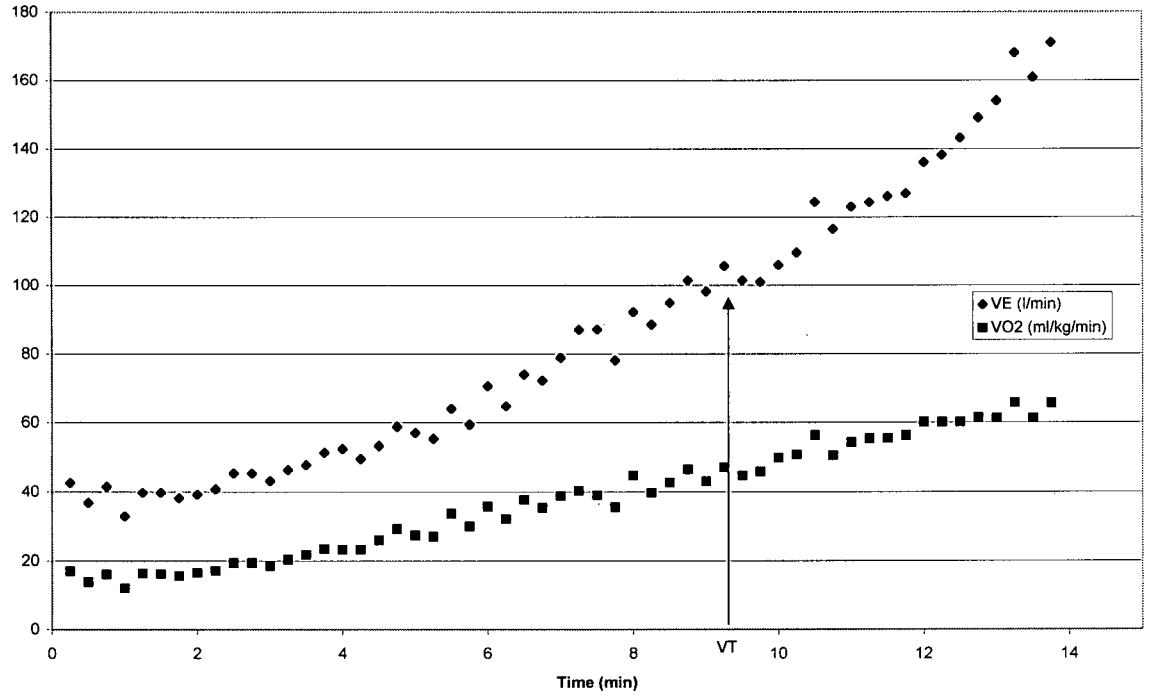
The relative magnitude of the recorded potential is dependent on a number of factors, including electrode placement, tissue impedance and filtering properties, distance of electrode to active fibers, conduction velocity of muscle fiber, muscle temperature, and muscle fiber type distribution. Placement of electrodes for surface EMG will greatly affect the magnitude of the recorded potential (102). Generally, electrodes should be placed over the center of the muscle belly as an electrode position near the middle of the muscle is at a smaller average distance from all points of potential sources. Deviation from this site will result in a reduction of recorded potential. In human muscle tissue, the amplitude of recorded potential is inversely proportional to the distance between the active fiber and the recording site (25). As well, tissue acts as a low-pass filtering system in which the signal bandwidth decreases as the distance from active fiber to recording site increases (25). The temperature of a muscle directly affects the transmission of potential and subsequent recording via surface electrodes. An increase in muscle temperature acts to decrease the amplitude of iEMG while increasing the center frequency of the mean power spectra (71). Another concern when using surface EMG is that the recording electrode is only sampling a small area of the muscle in close proximity to its placement. The distribution pattern of muscle fibers in a given muscle will affect the amplitude of EMG signal measured. Surface EMG can only reflect a true sample of whole muscular activity if motor units of all sizes are randomly distributed throughout the muscle.

During the course of fatigue, there are a number of changes in the electromyographic signal. Some of these changes are the result of increases in recruitment of motor units and increases in the firing rates of already active motor units, while other changes reflect inherent properties of both muscle and EMG recording devices. It is believed that additional recruitment and increases in firing rates are due to some failure in the muscle contractility, so that contraction of the individual active fibers exert progressively less force (91). Evidence for this is seen as increases in both amplitude and timing of averaged motor unit potentials (60,91). In addition, during the course of fatigue in a muscle the conduction velocity of the motor unit action potentials (MUAP) decreases (25,65). This decrease in conduction velocity is reflected in a concomitant shift to lower frequencies in the power density spectrum. As MUAP duration increases, the duration of time each potential change is detected from a stationary recording electrode will increase, thus

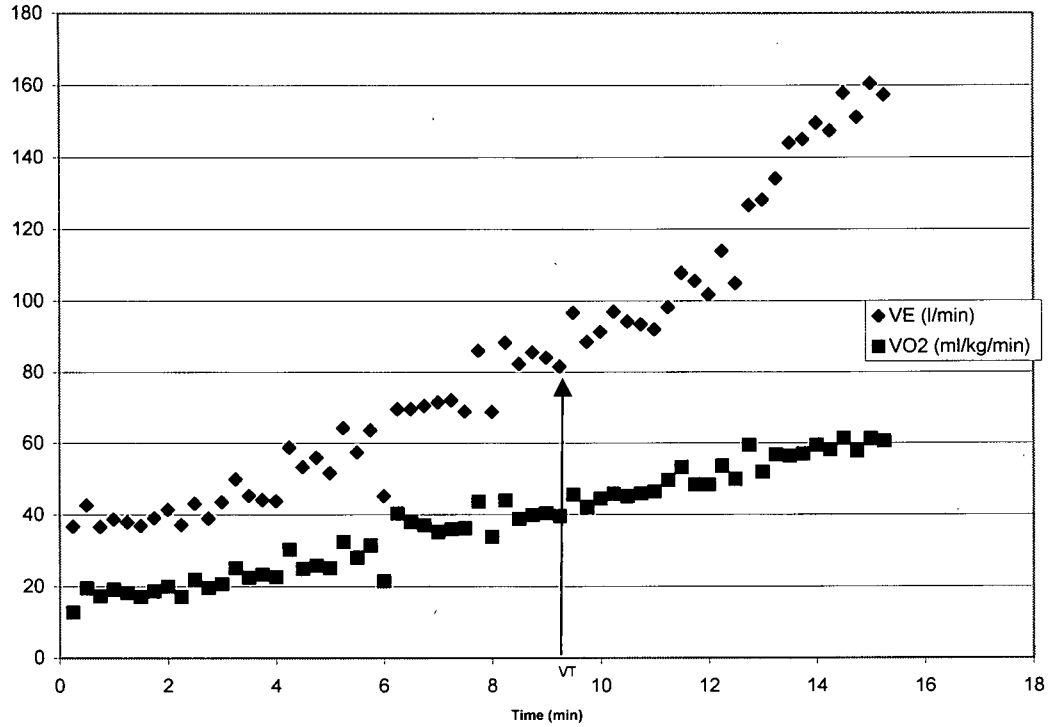
causing a decrease in the center frequency. Because both muscle tissue and differential electrodes act as low-pass filters, a shift to a lower frequency spectrum would result in an increase in the measurable electrical activity within the muscle (25). In a recent review of methods of muscular fatigue measurement, it was noted that because electrical activation of muscle fibers precedes force generation, EMG changes may in principle occur independent of force or power output (95). While there are significant limitations in the use of electromyography for the assessment of muscular fatigue, the basic underlying principles governing EMG signal characteristics are valid. The use of EMG to assess neuromuscular fatigue has validity and has been proven reliable (28).

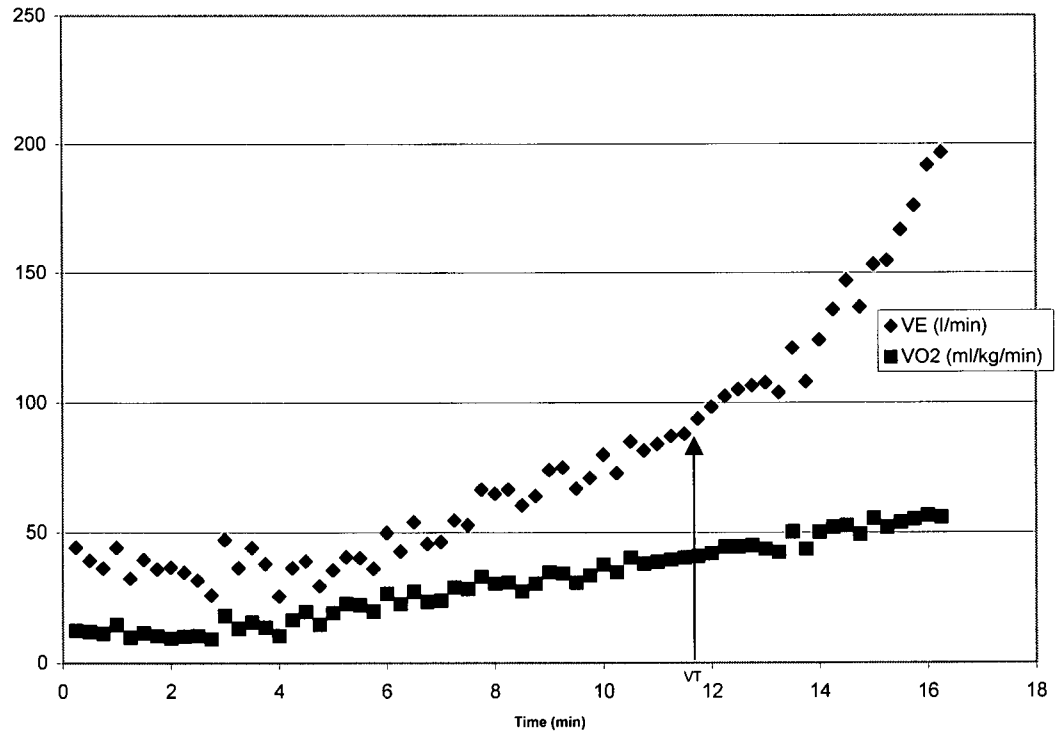
## Appendix II – Individual subject VT calculations



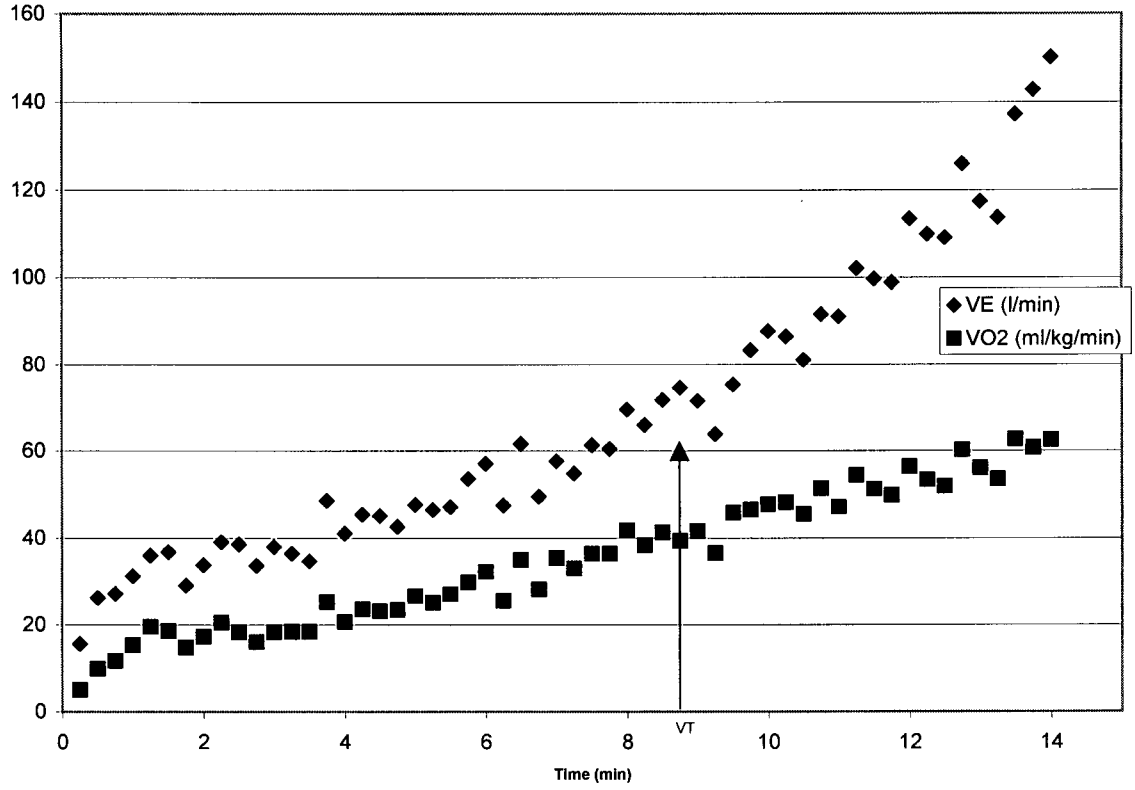



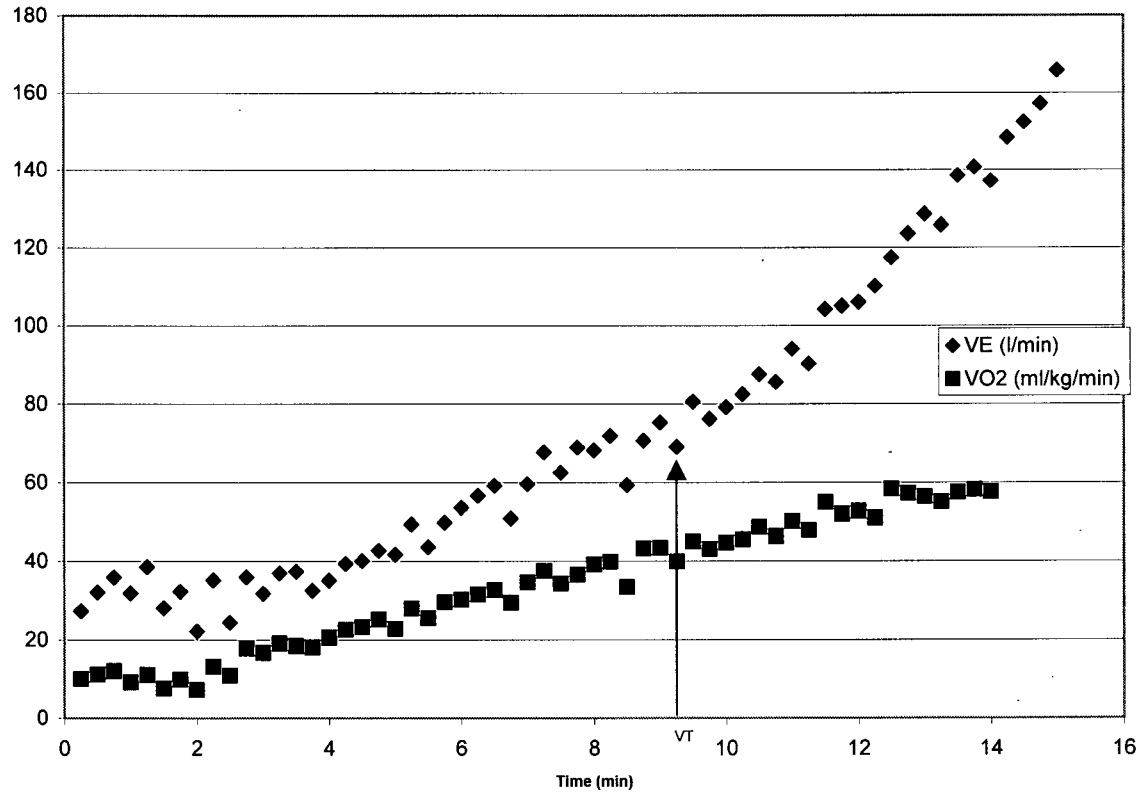

Subjects 1, 2 - Maximal oxygen uptake test, calculation of ventilatory threshold (VT). 68



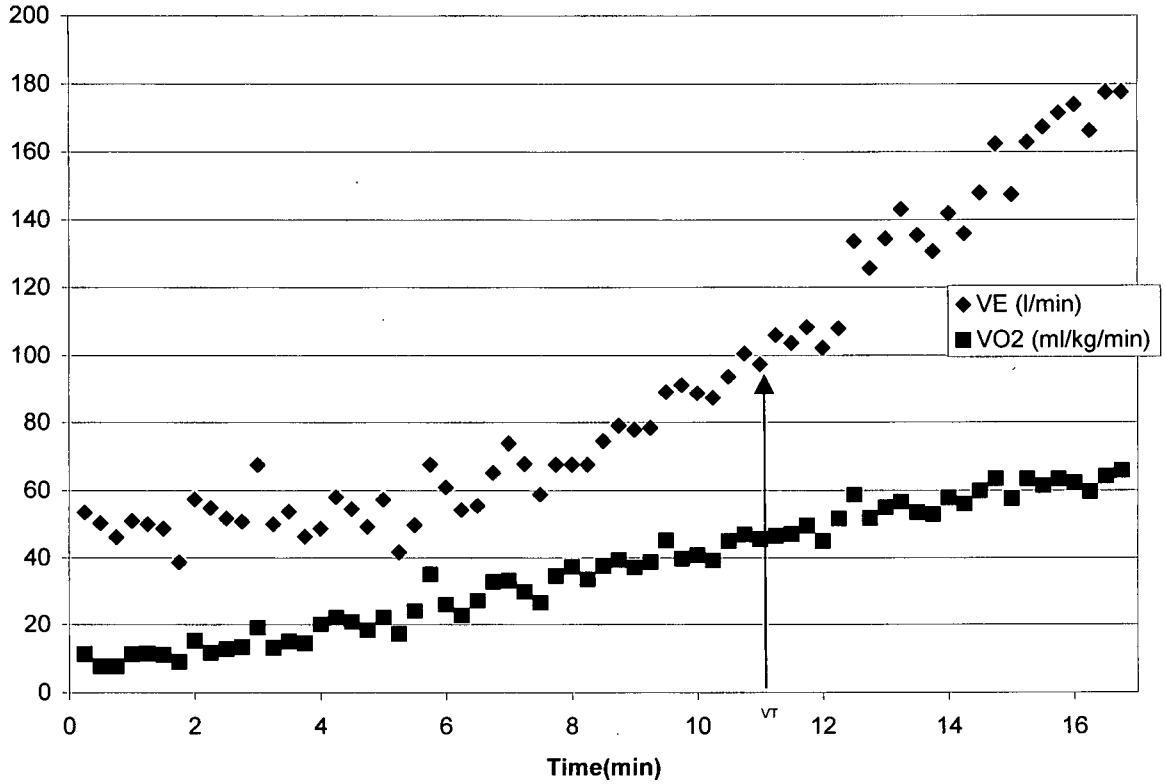



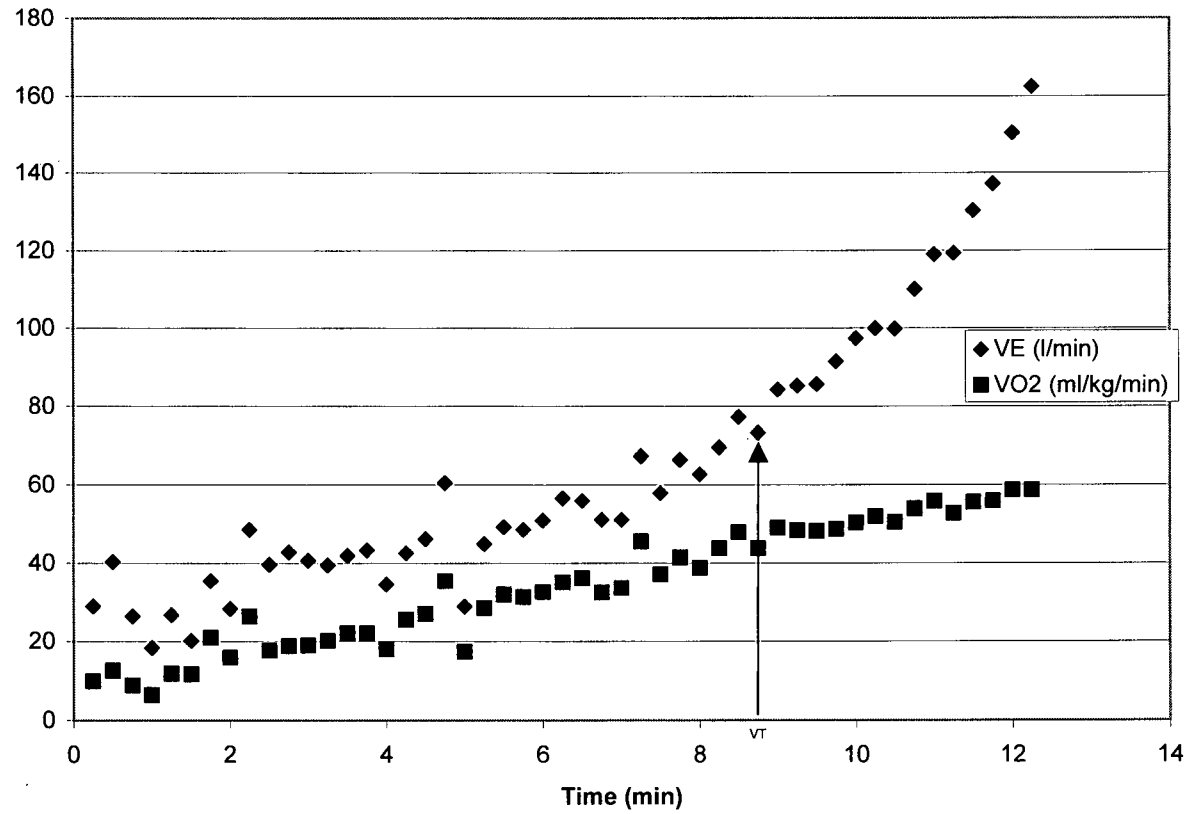

Subjects 3,4 - Maximal oxygen uptake test, calculation of ventilatory threshold (VT).



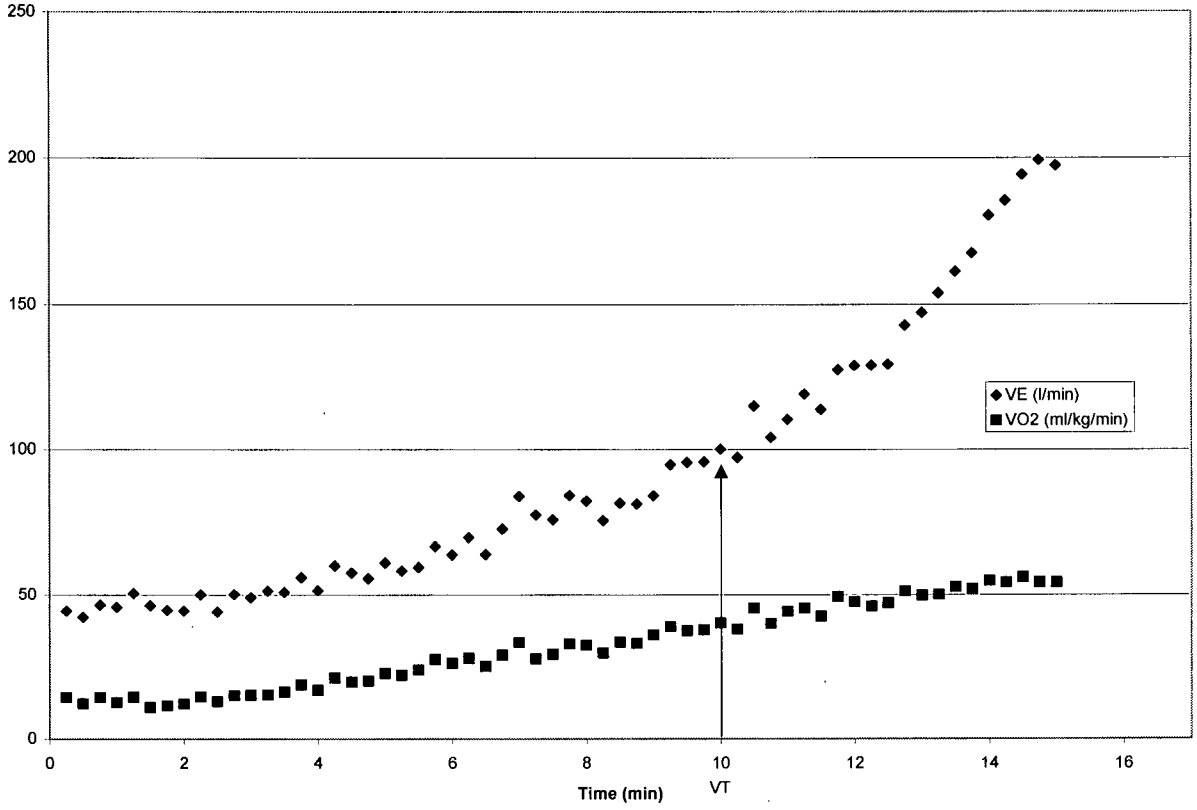



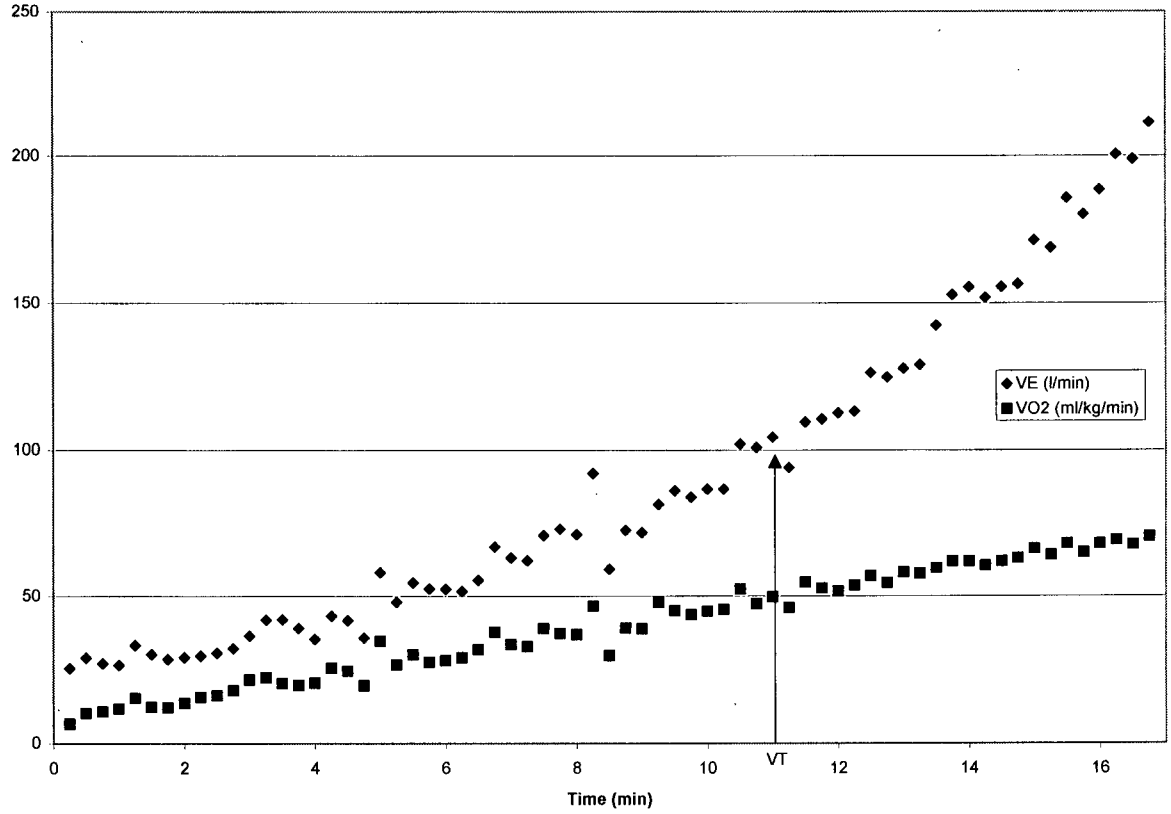

Subjects 5, 6 - Maximal oxygen uptake test, calculation of ventilatory threshold (VT).



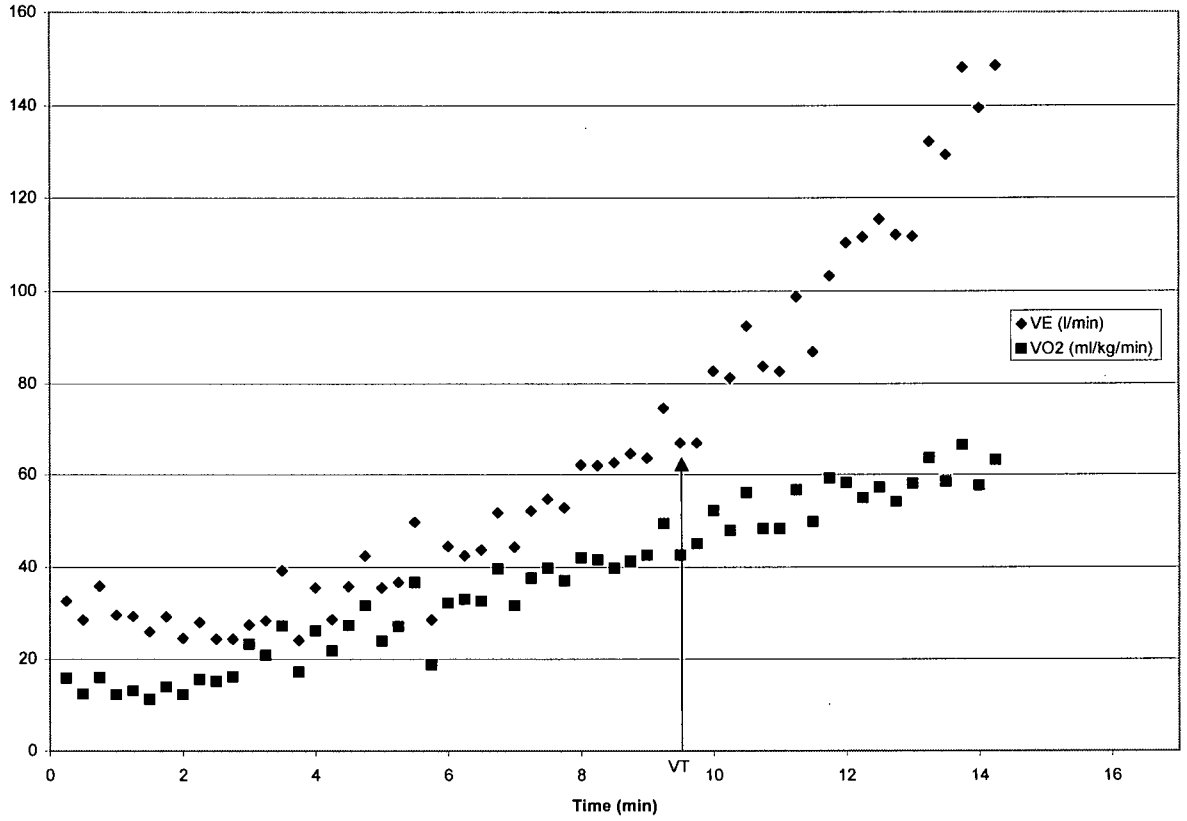



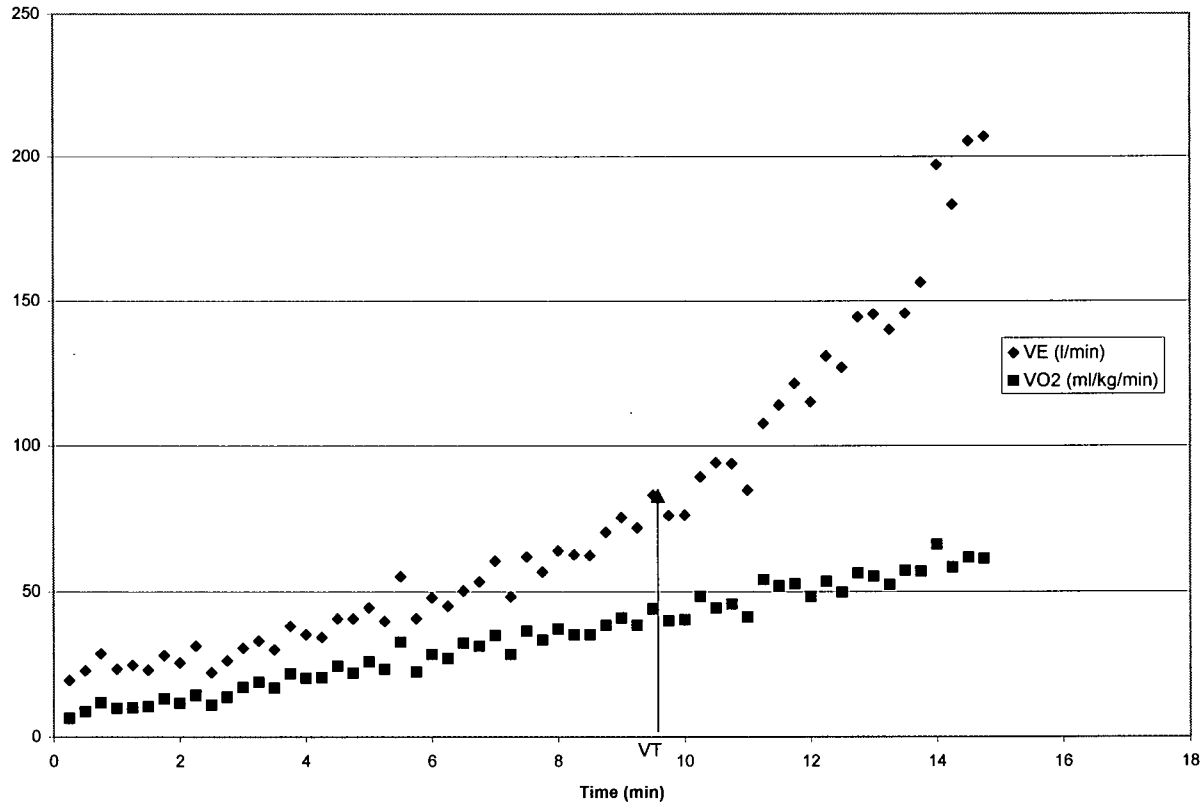

Subjects 7, 8 - Maximal oxygen uptake test, calculation of ventilatory threshold (VT).



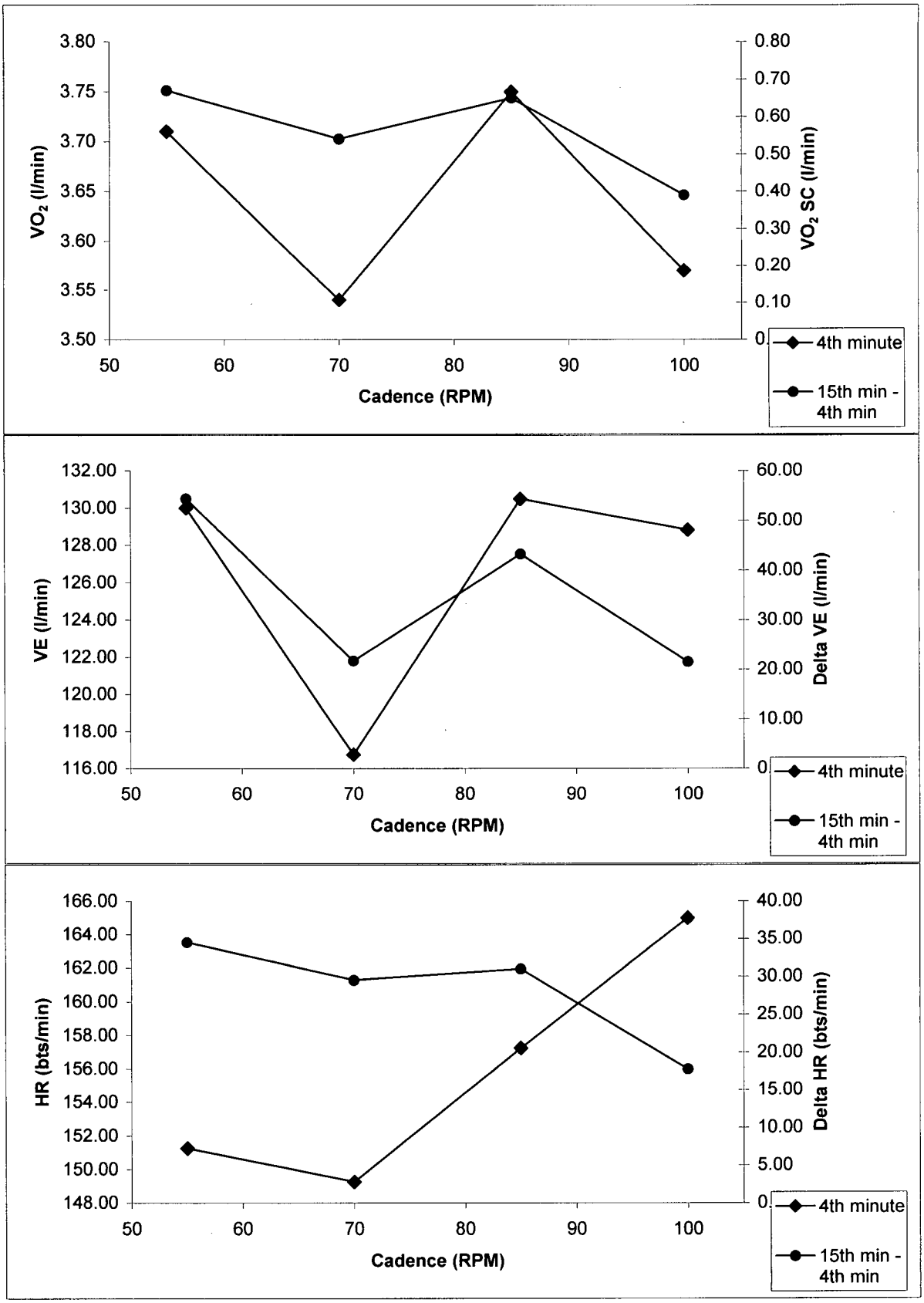

Subjects 9, 10 - Maximal oxygen uptake test, calculation of ventilatory threshold (VT). 72



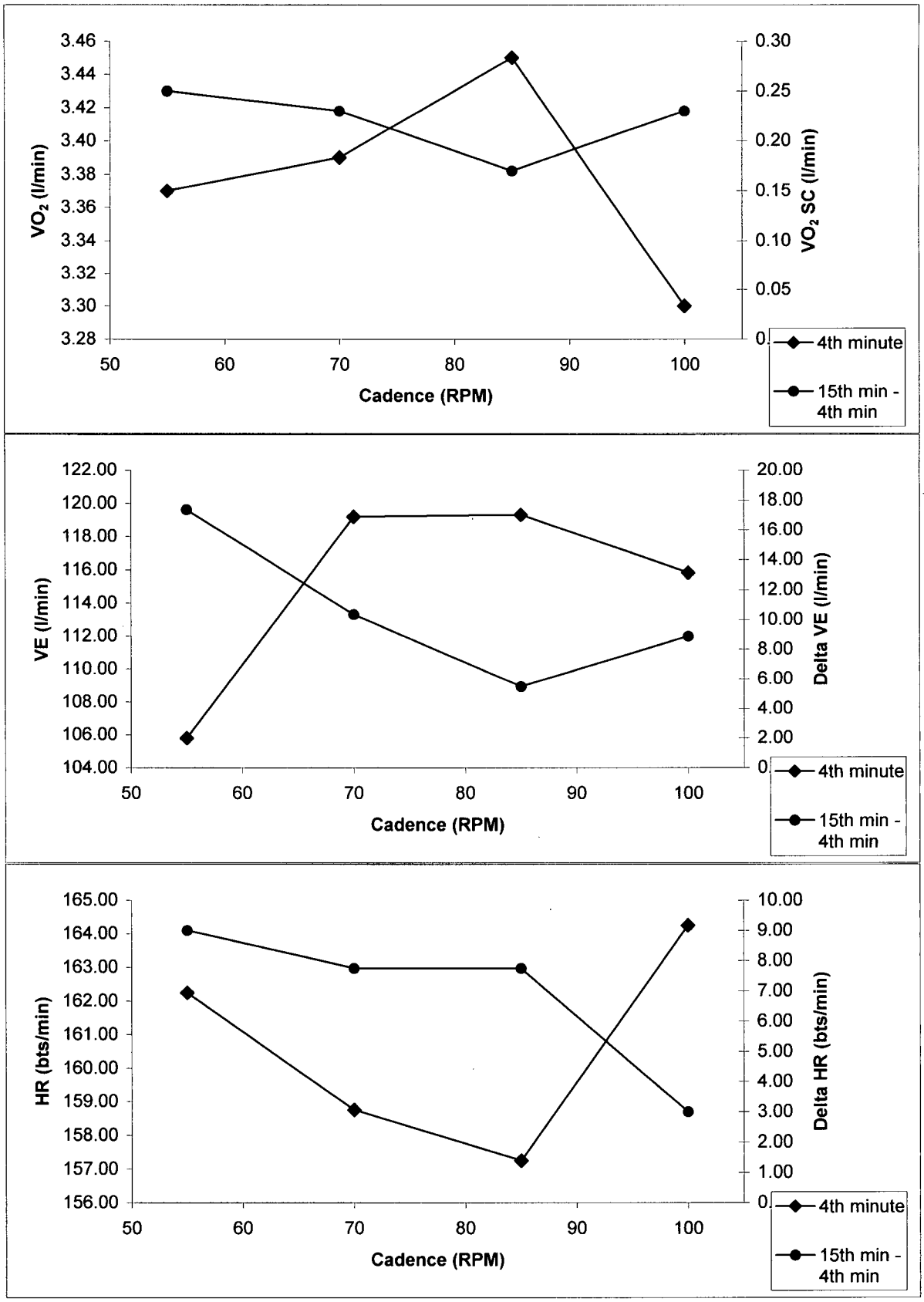




Subjects 11, 12 - Maximal oxygen uptake test, calculation of ventilatory threshold (VT). 73

## Appendix III – Individual subject $VO_2$, VE, and HR response to cadence




Subject 1 - VO2, VE, and HR over cadence conditions.

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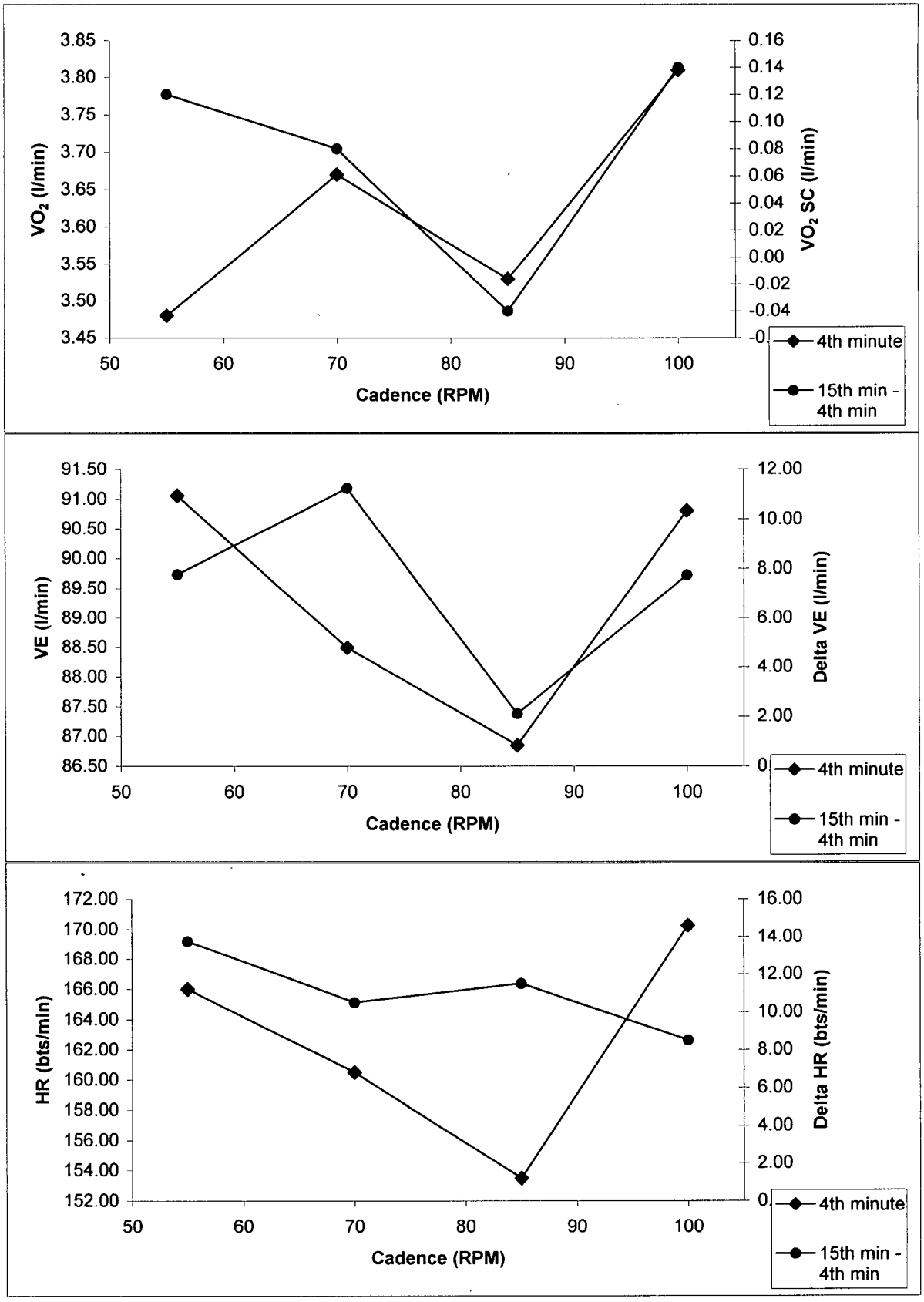
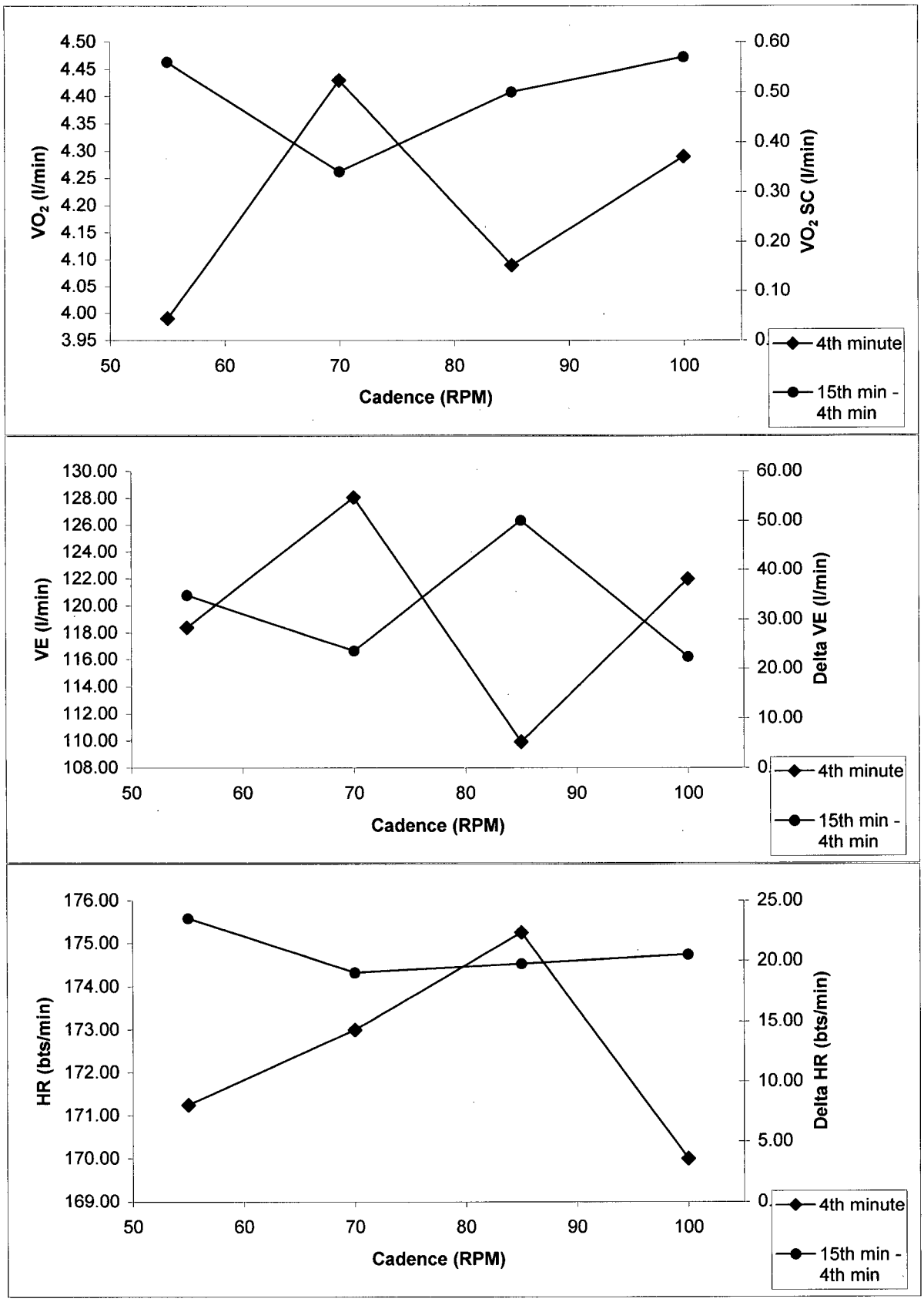



Subject 3 - VO2, VE, and HR over cadence conditions.

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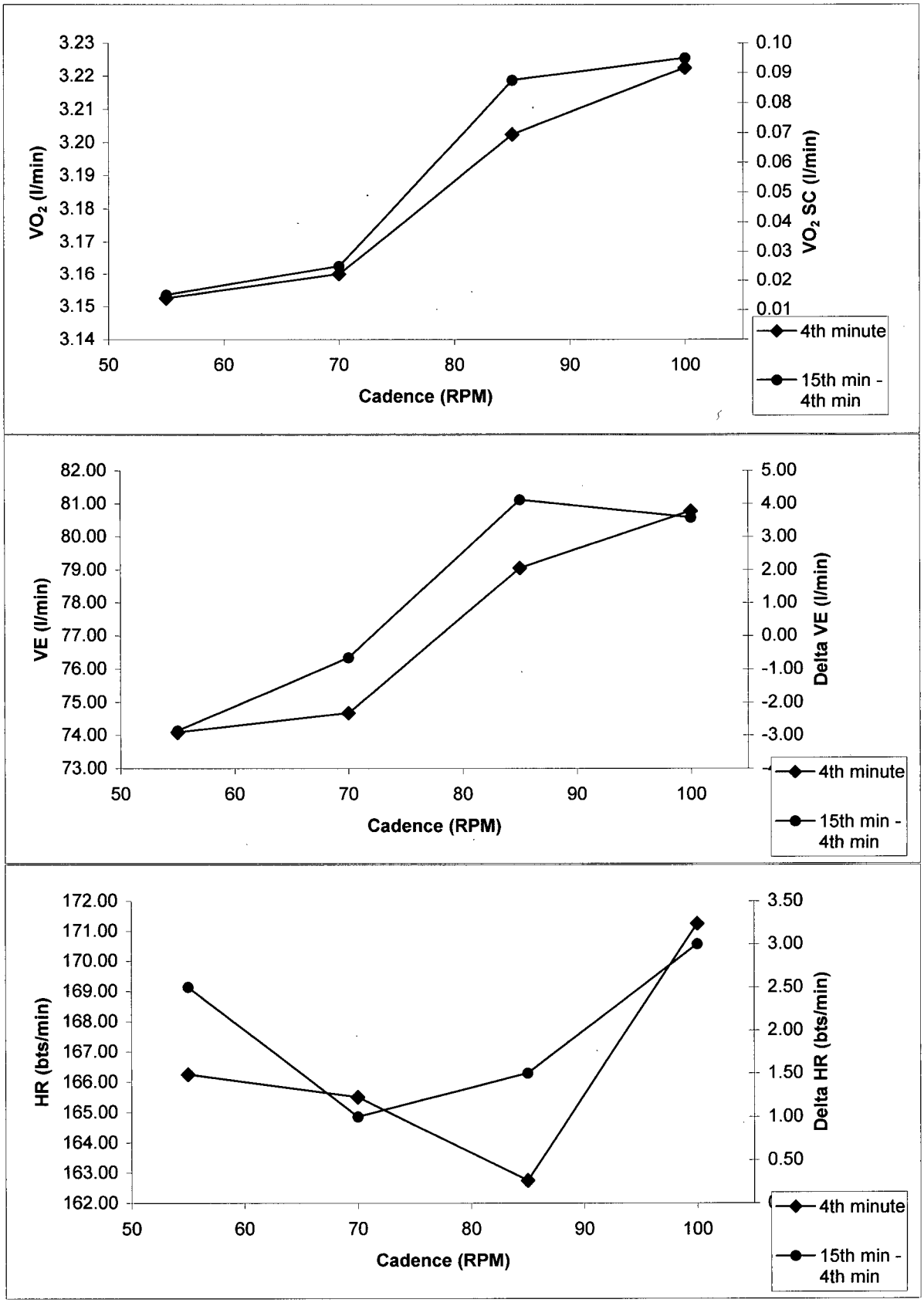


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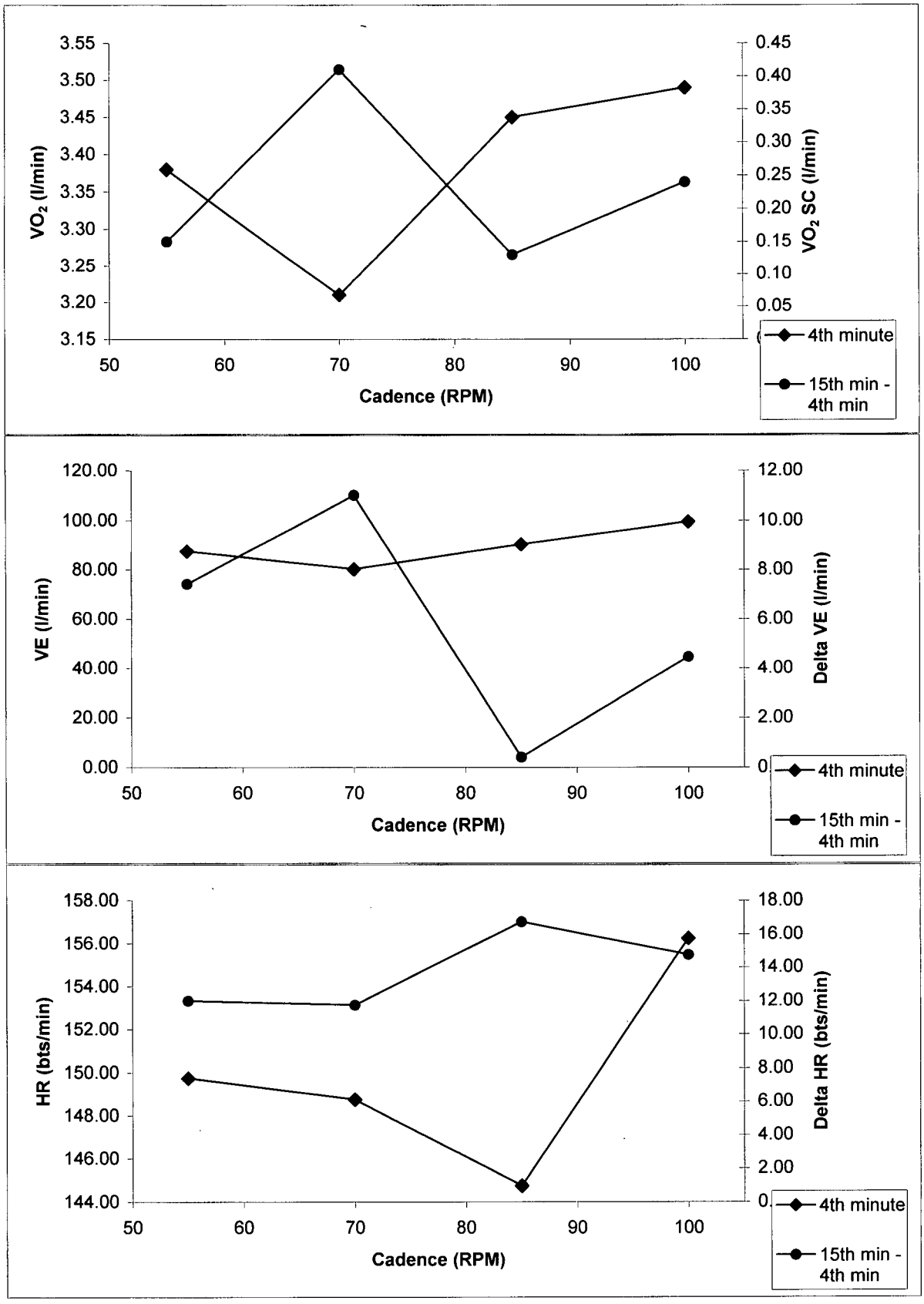


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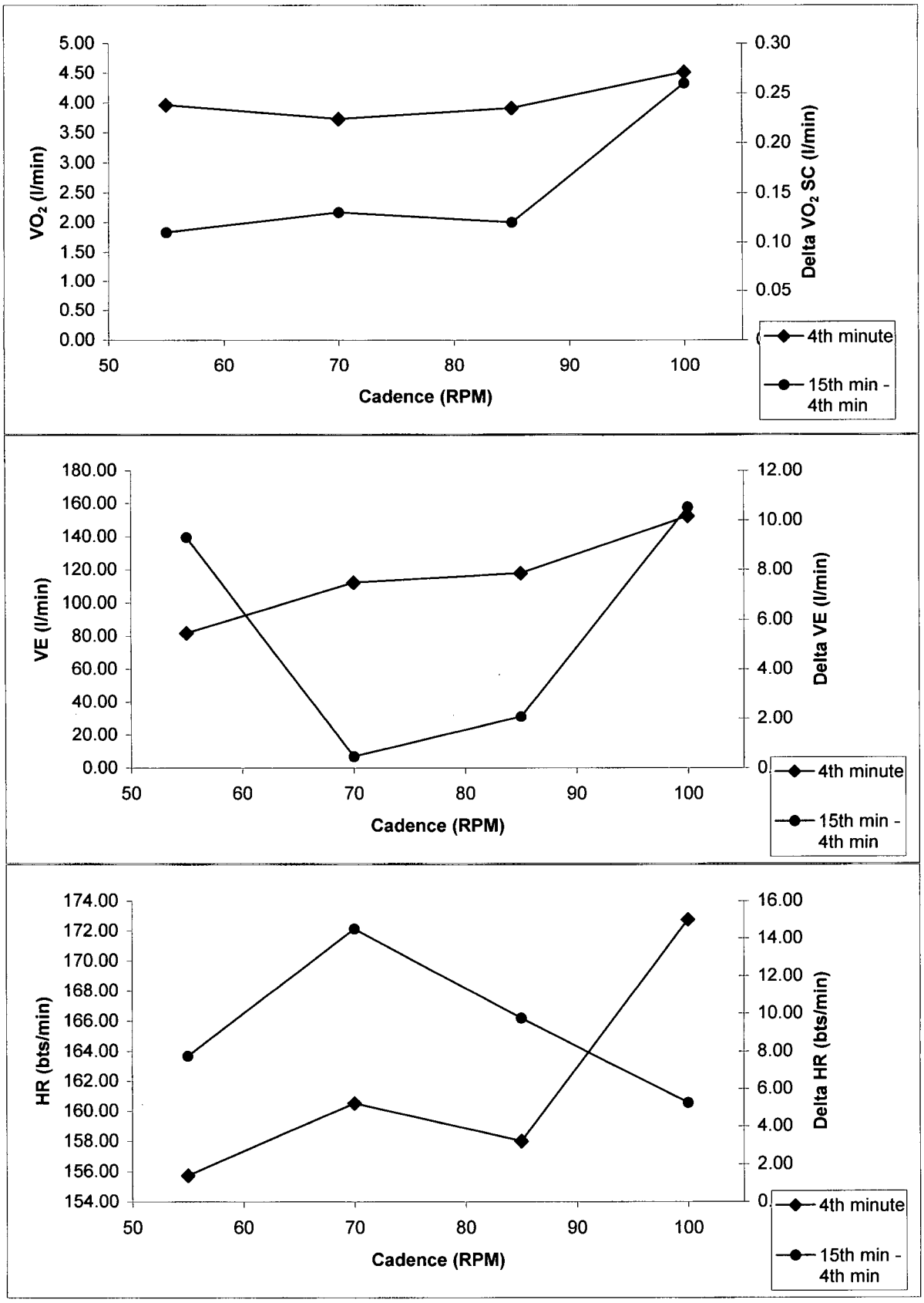


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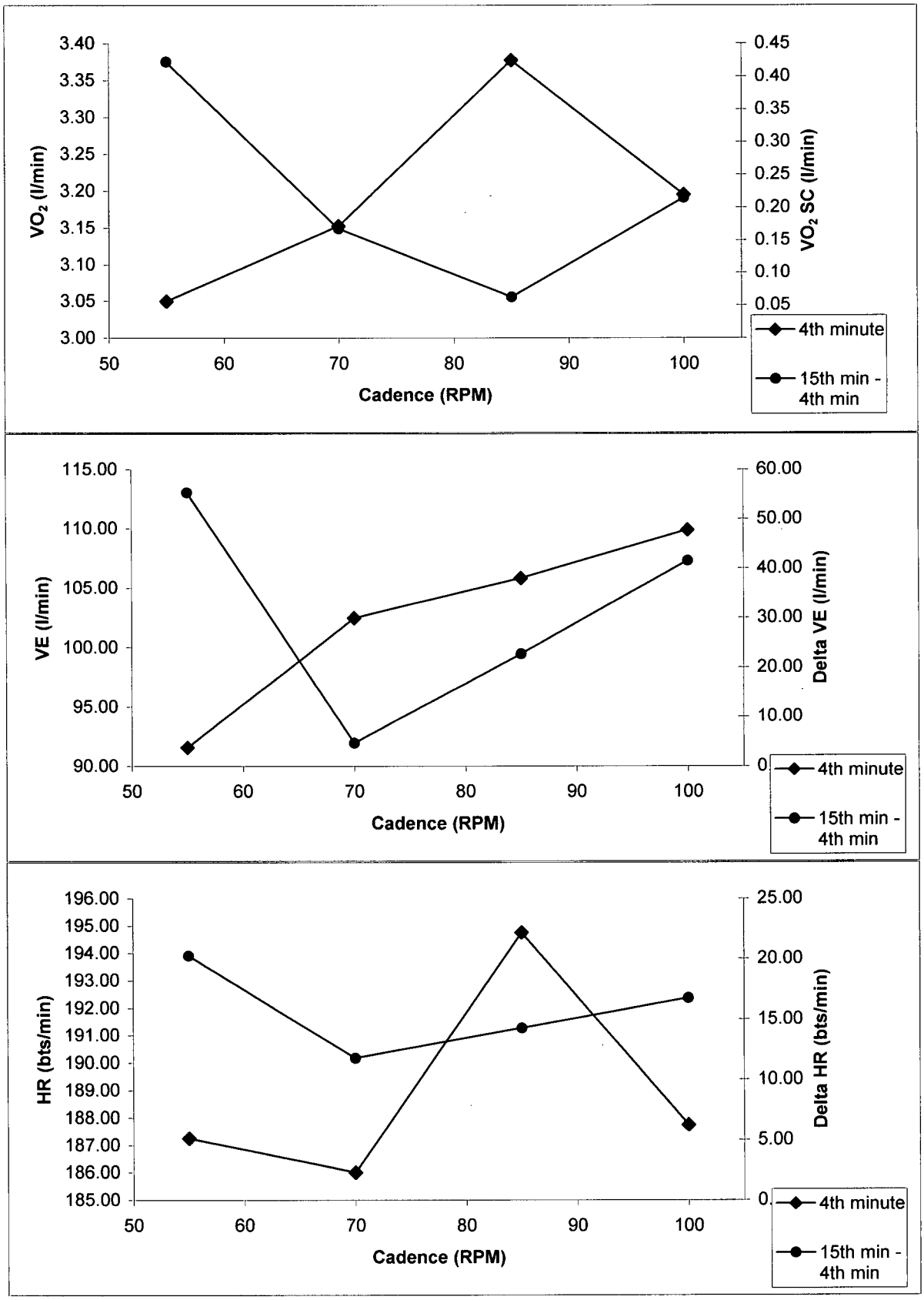


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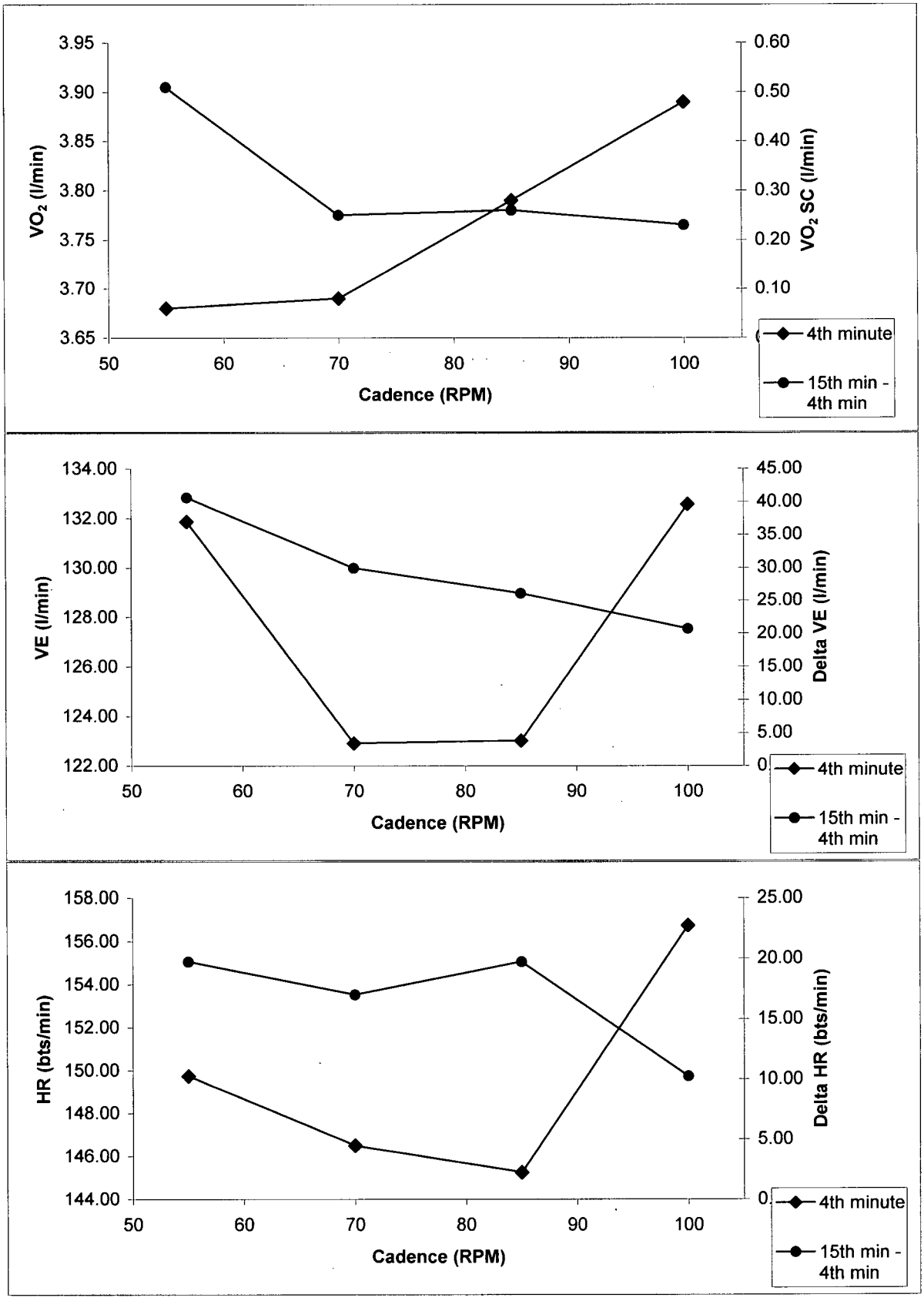
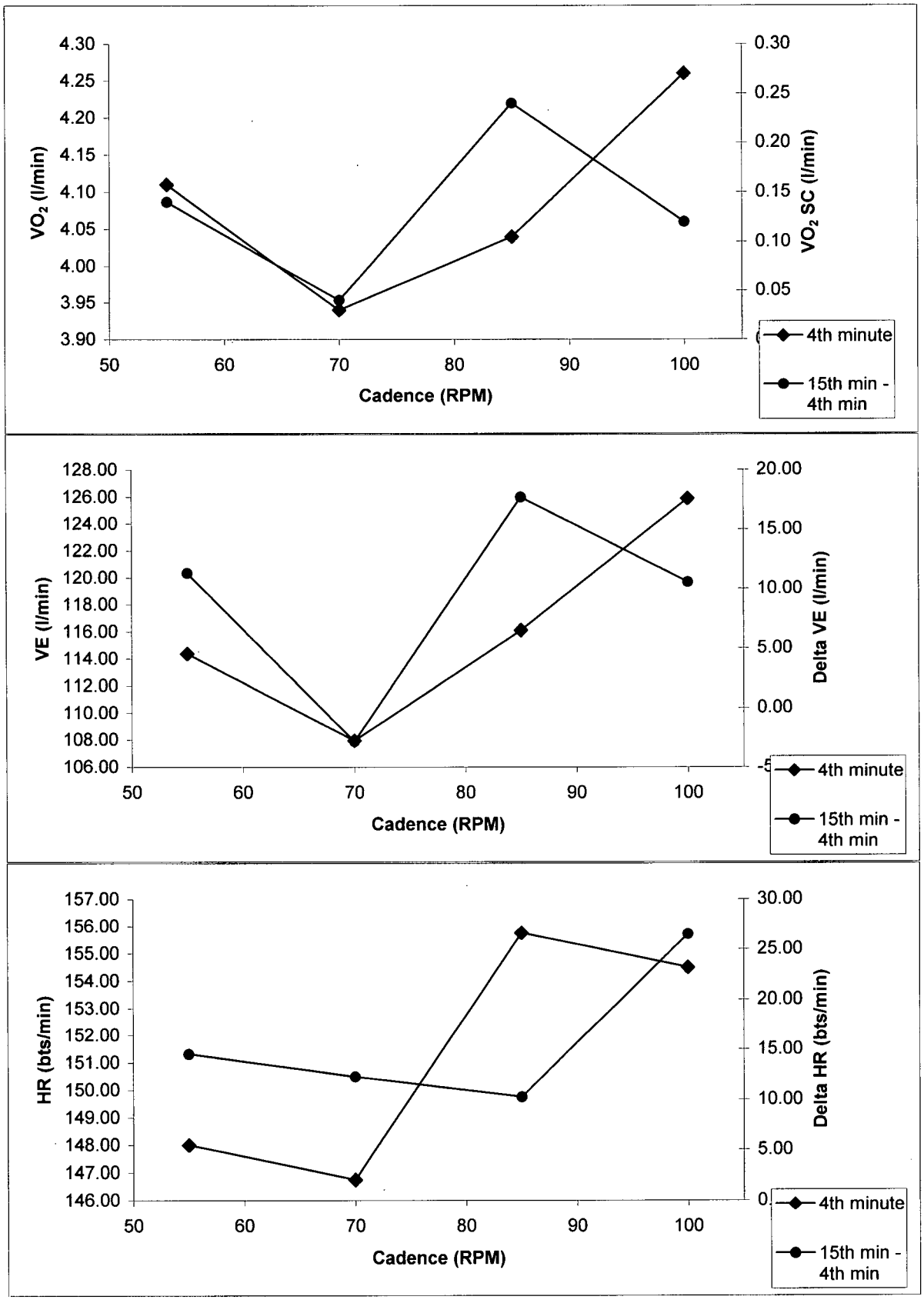
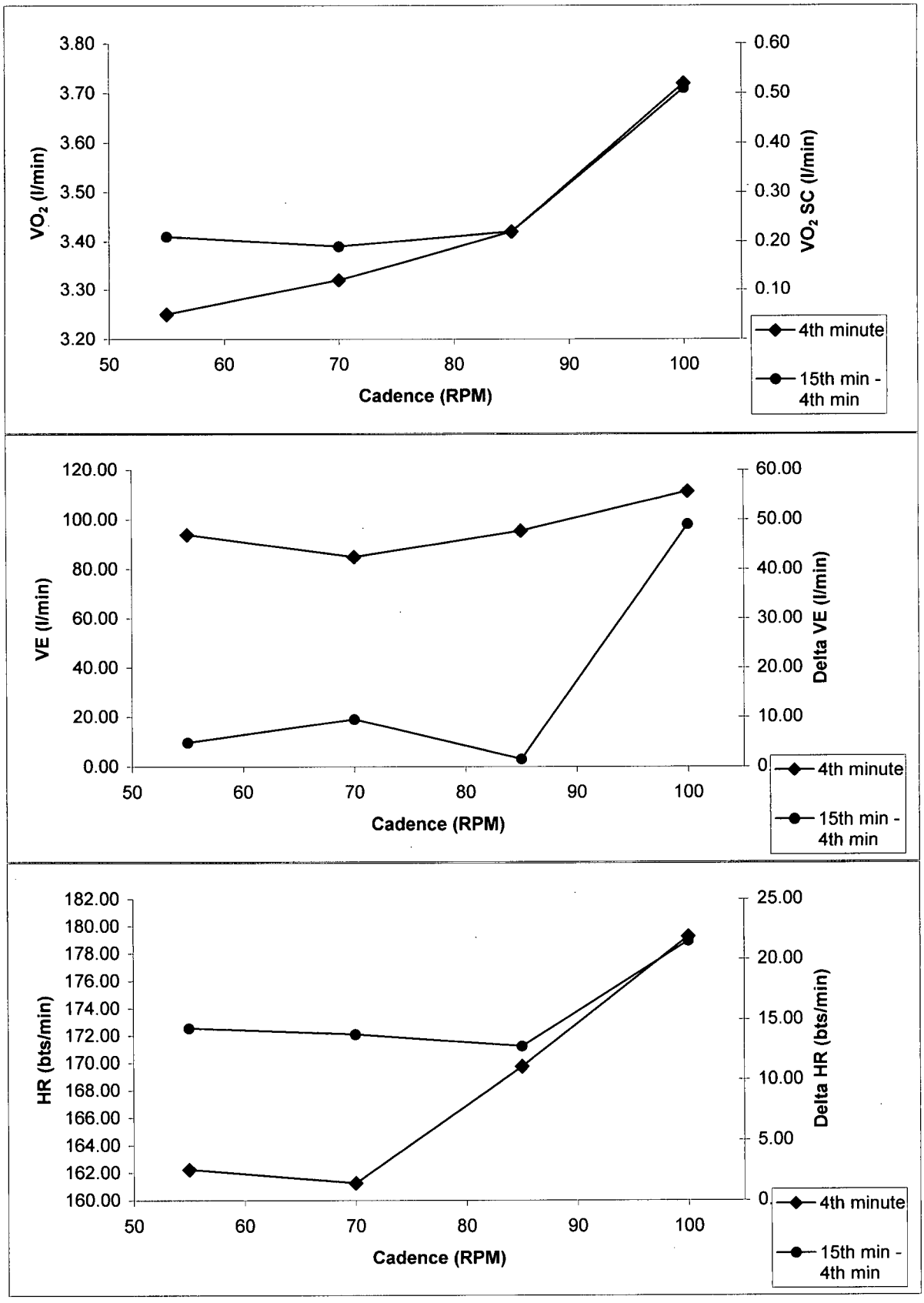



Subject 9 - VO2, VE, and HR over cadence conditions.




Subject 10 - VO2, VE, and HR over cadence conditions.

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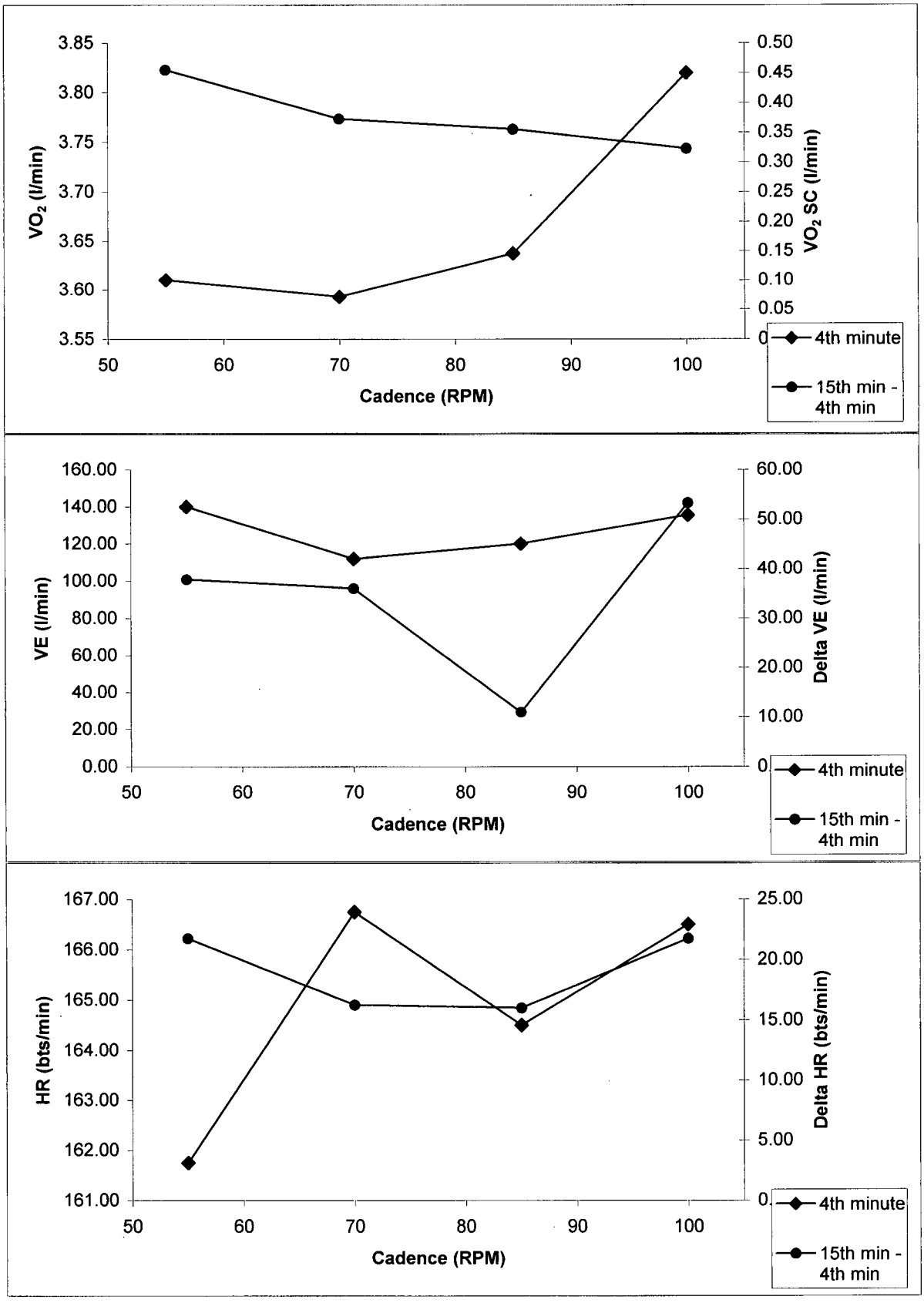
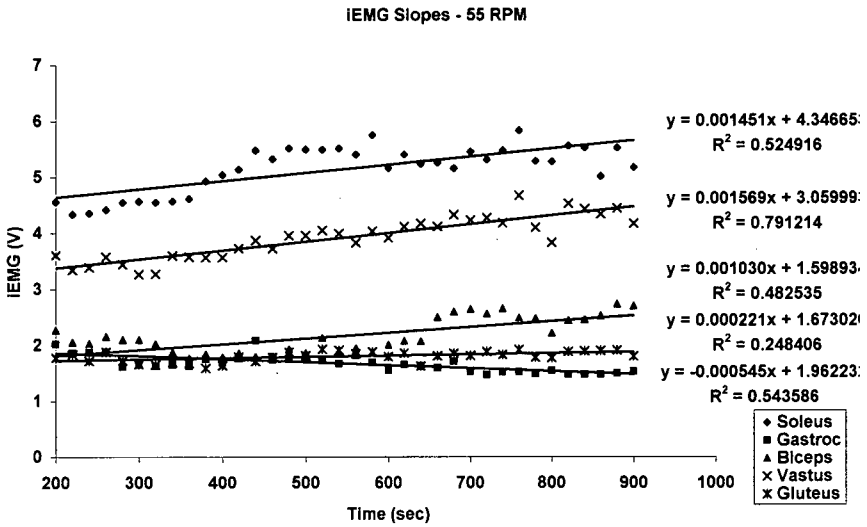
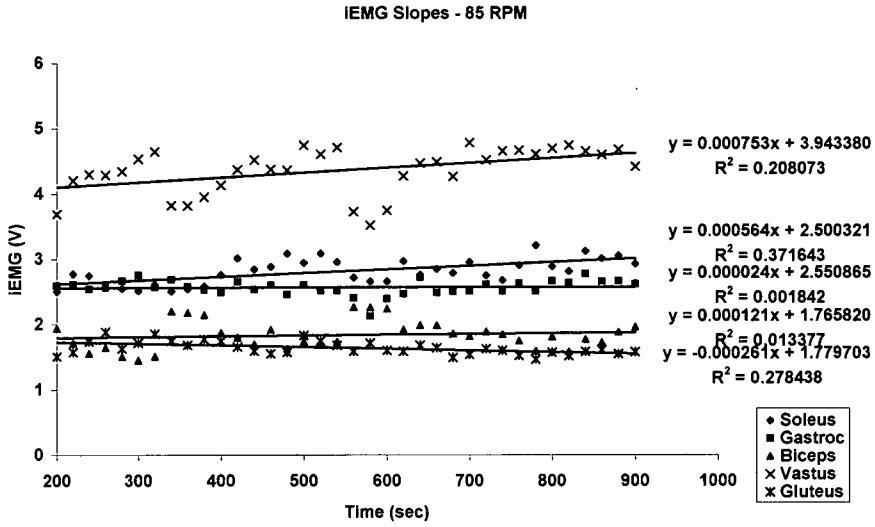
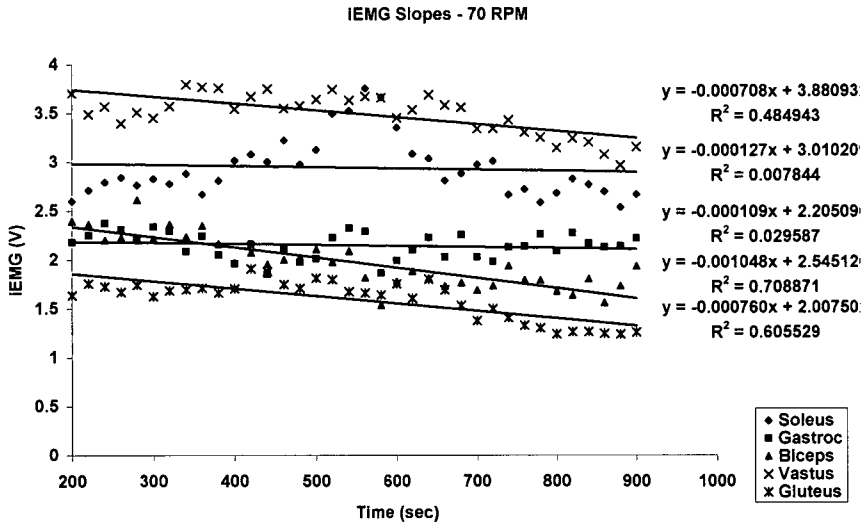


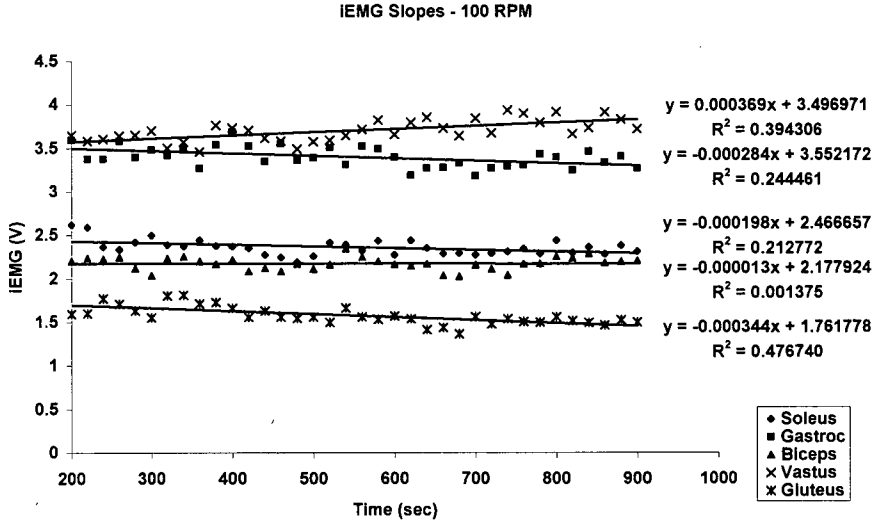

Subject 12 - VO2, VE, and HR over cadence conditions.

## Appendix IV – Individual subject iEMG slopes over cadence conditions



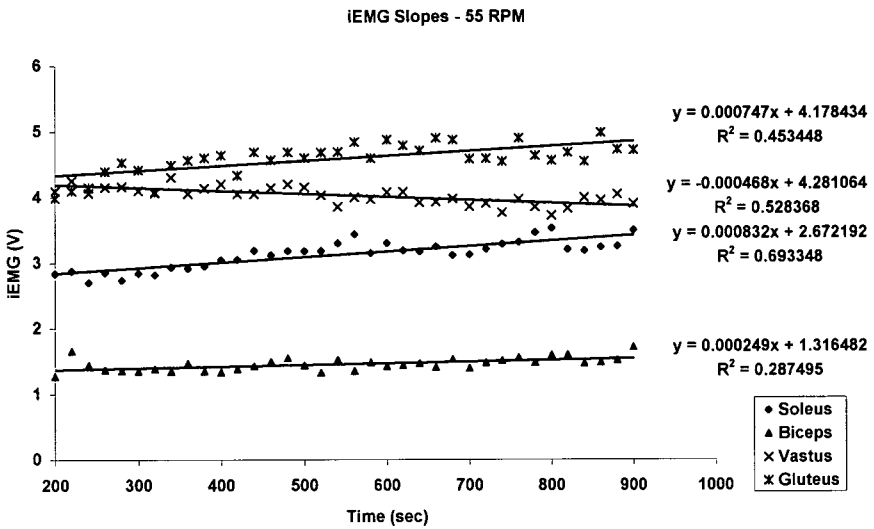



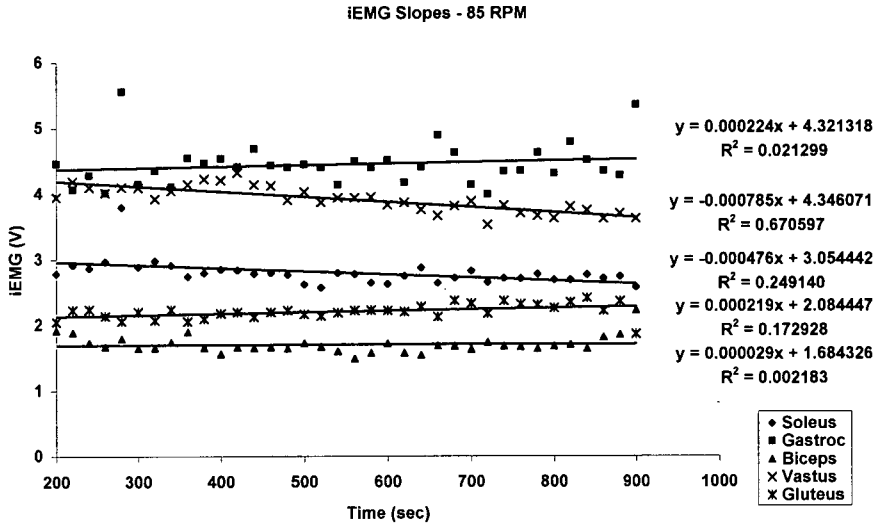



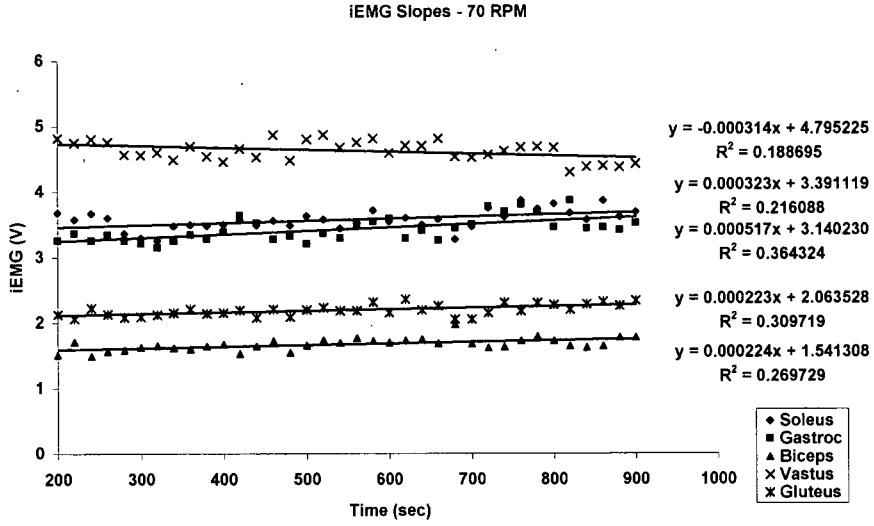



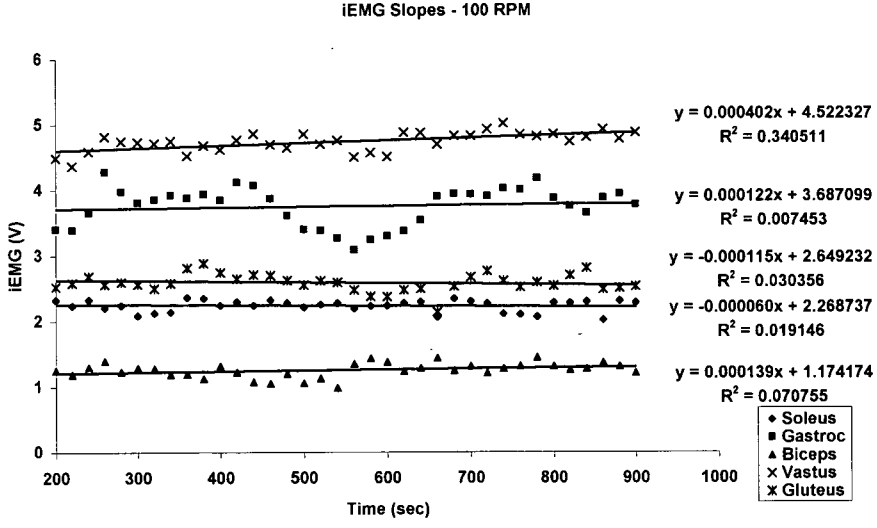



Subject 1 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions



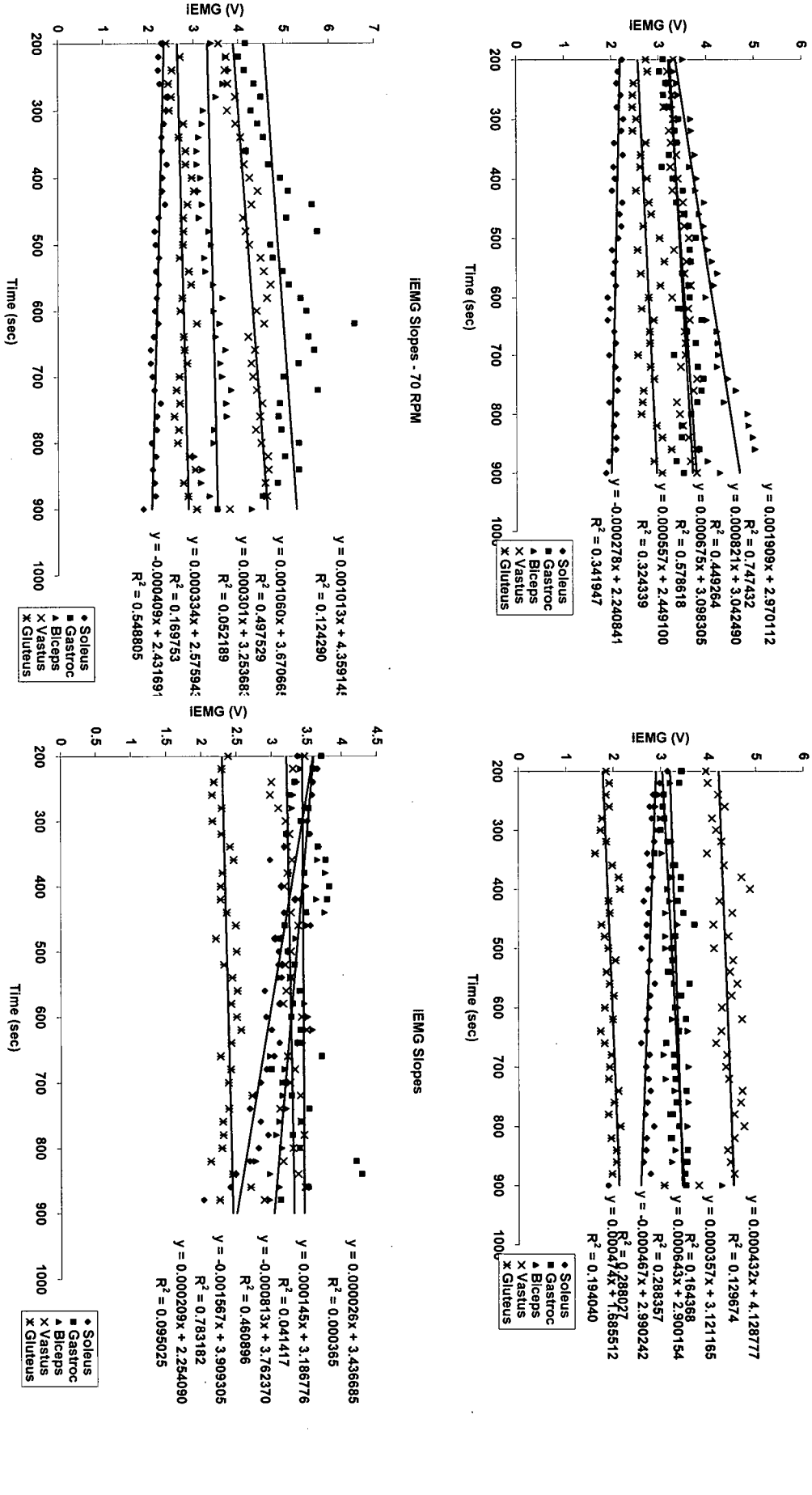






Subject 2 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions.

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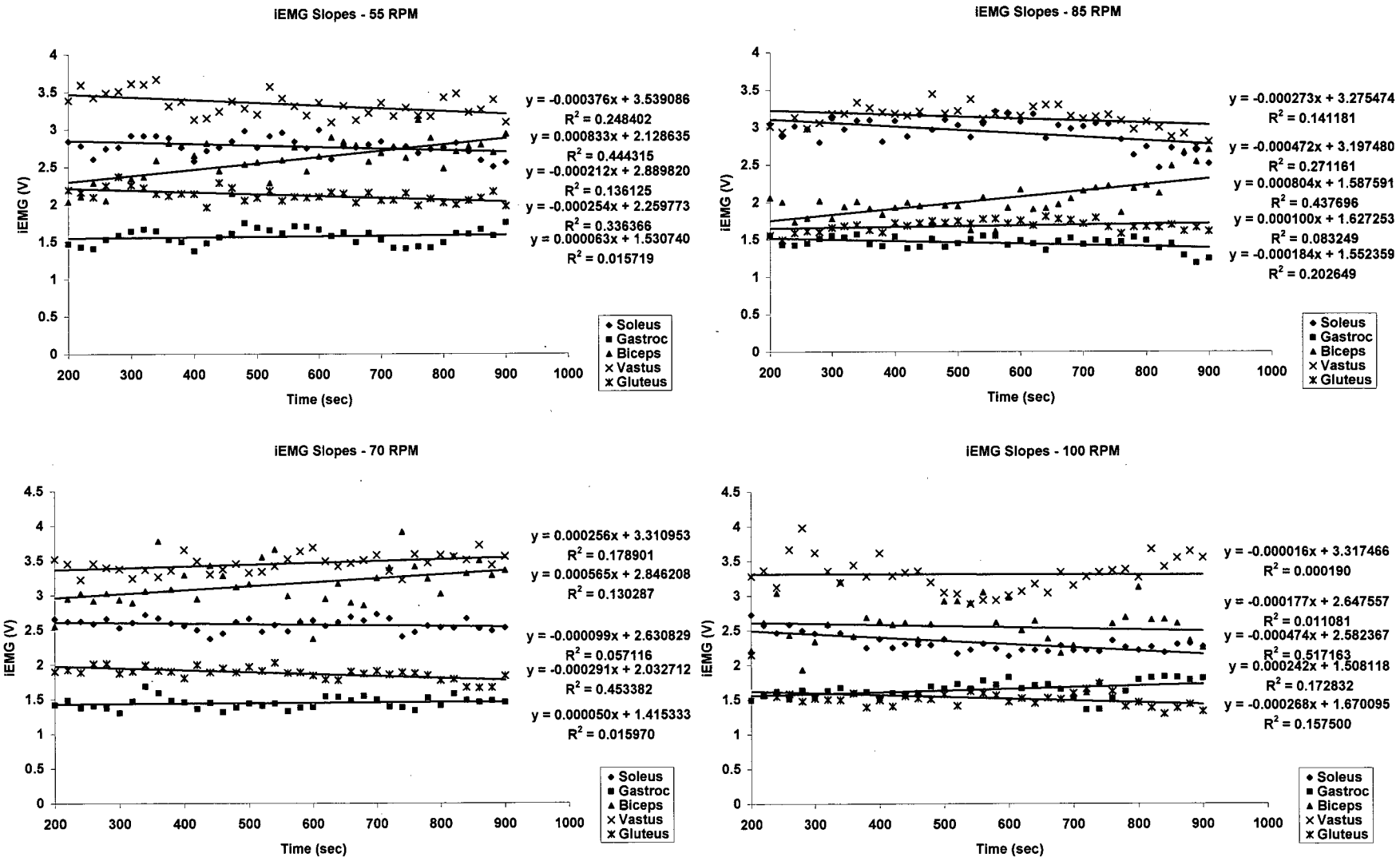
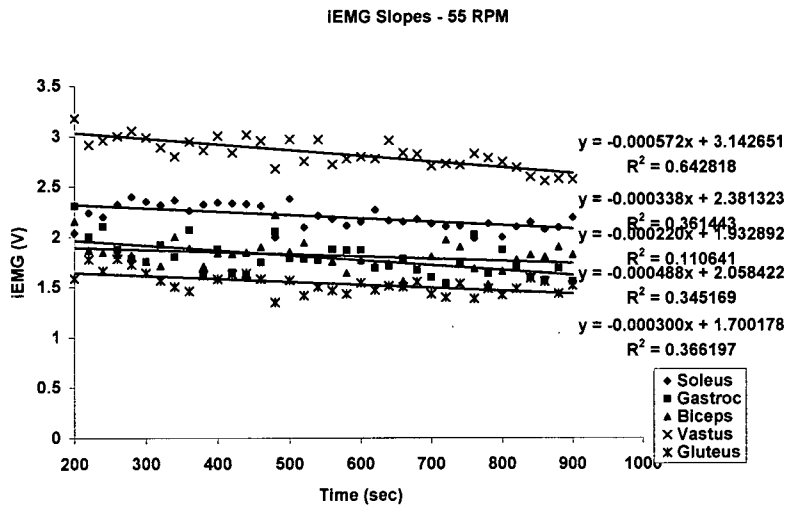
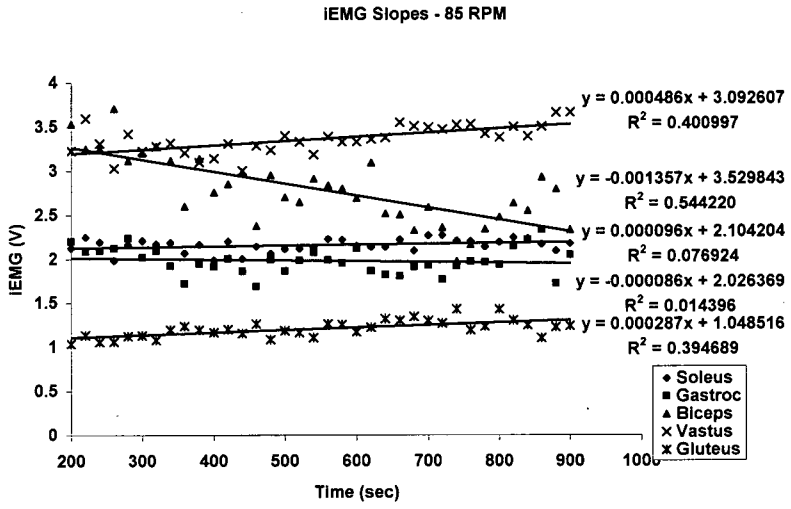
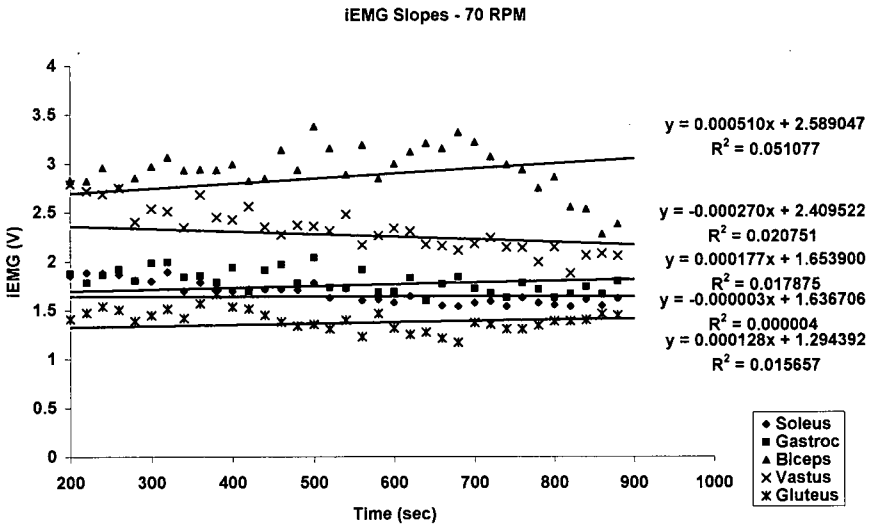


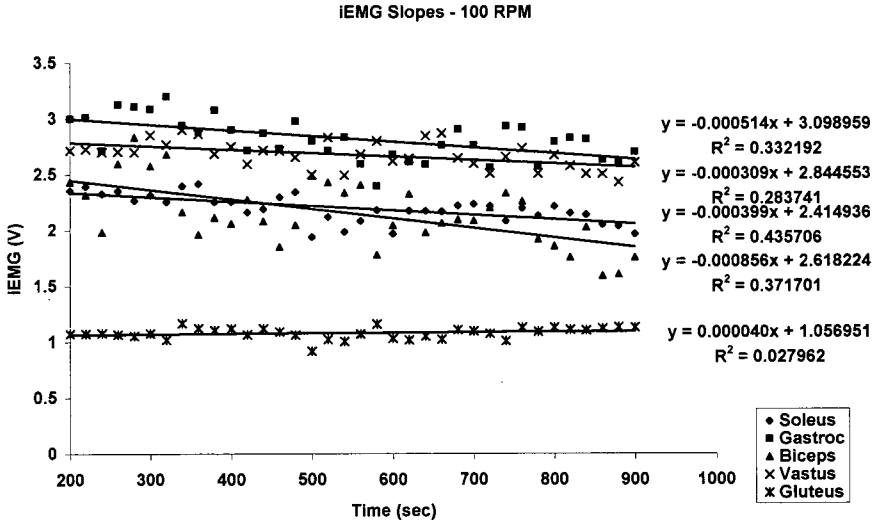



Subject 3 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions.



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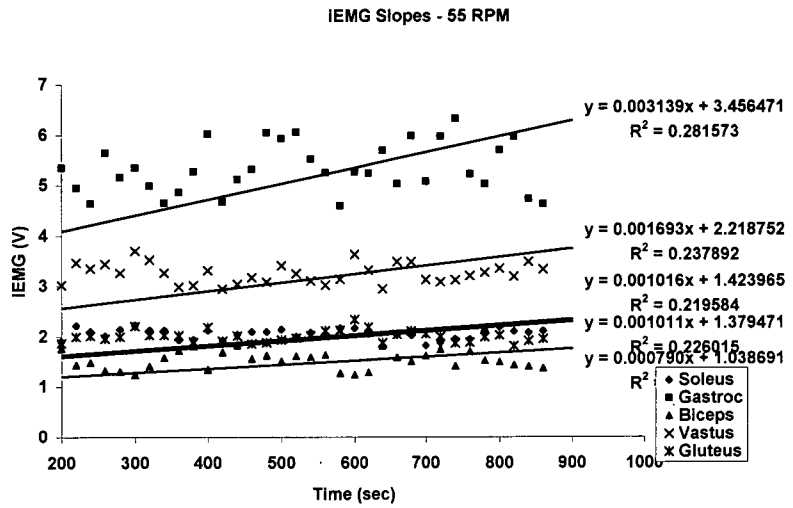
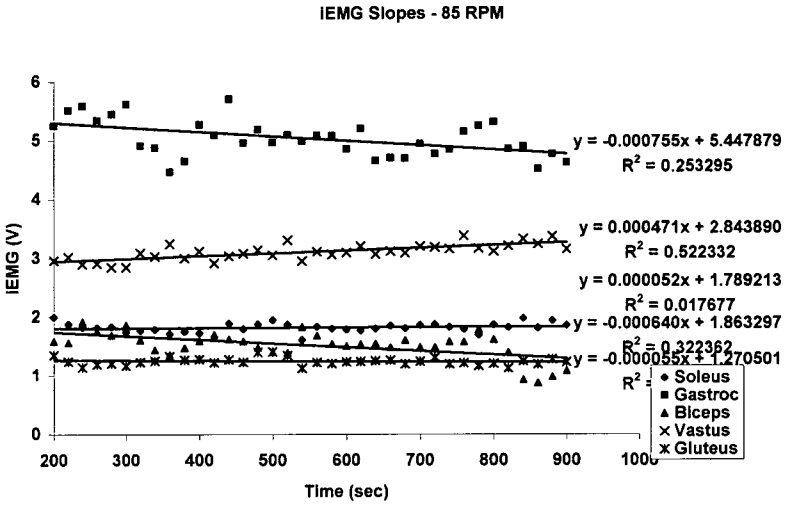
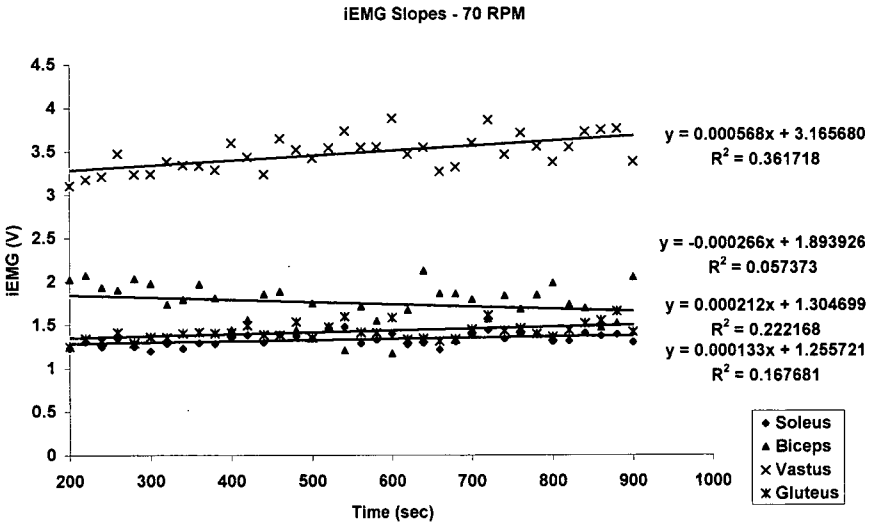
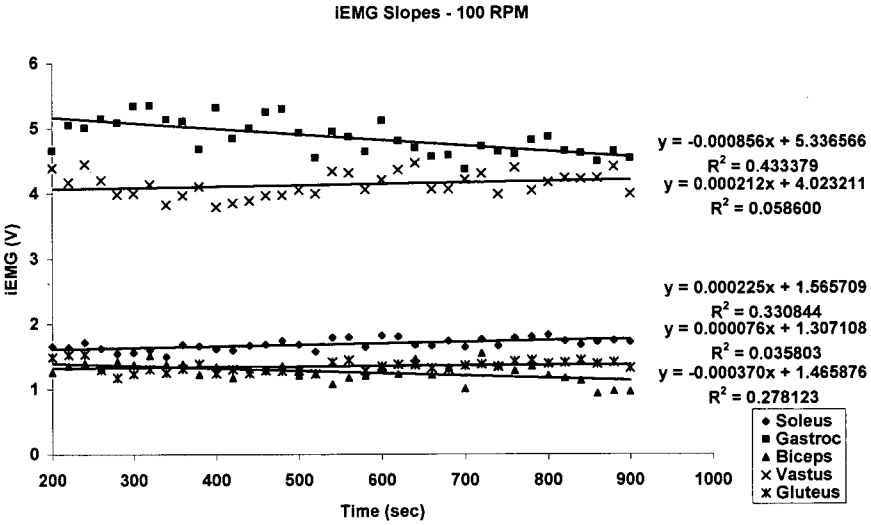


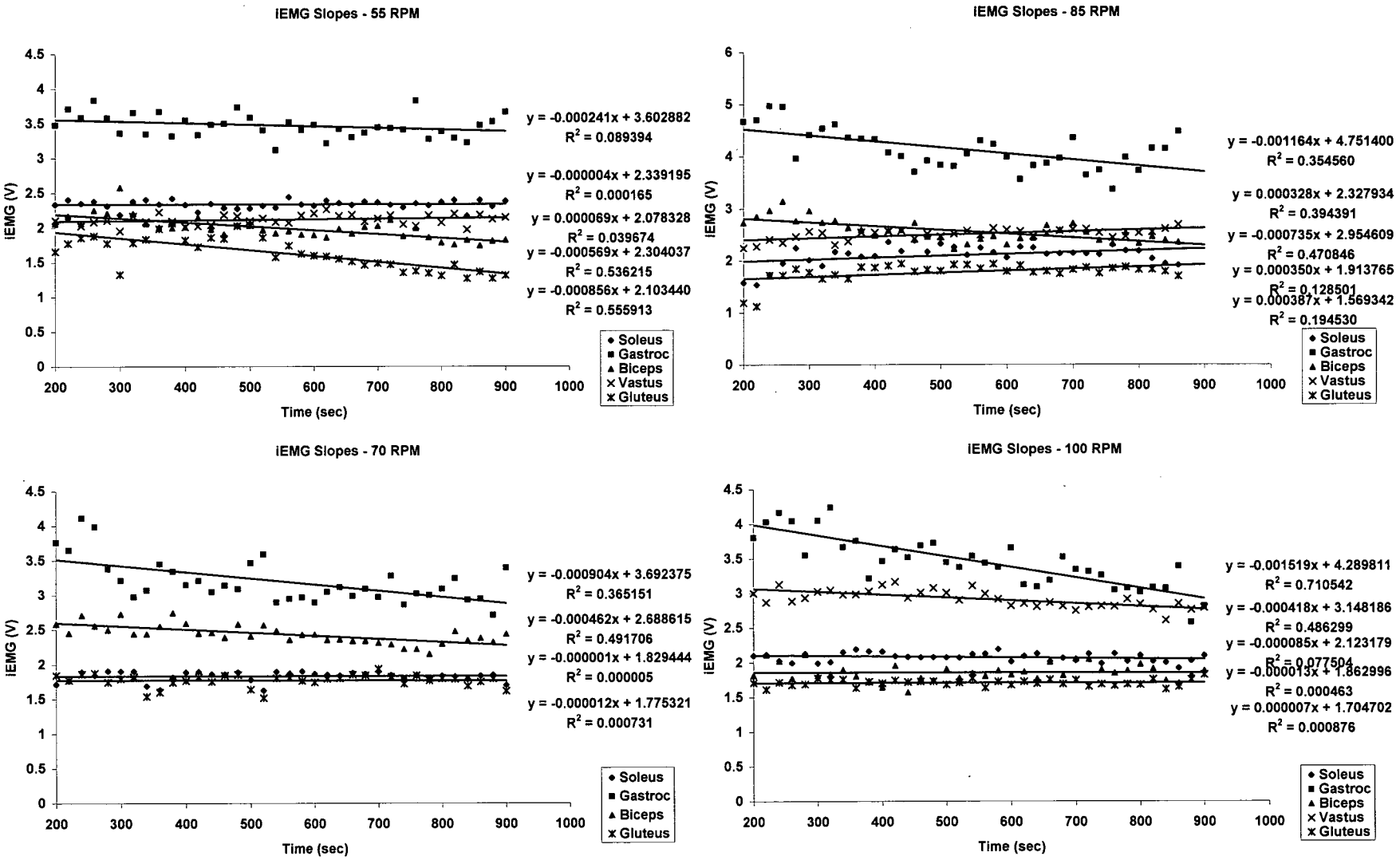



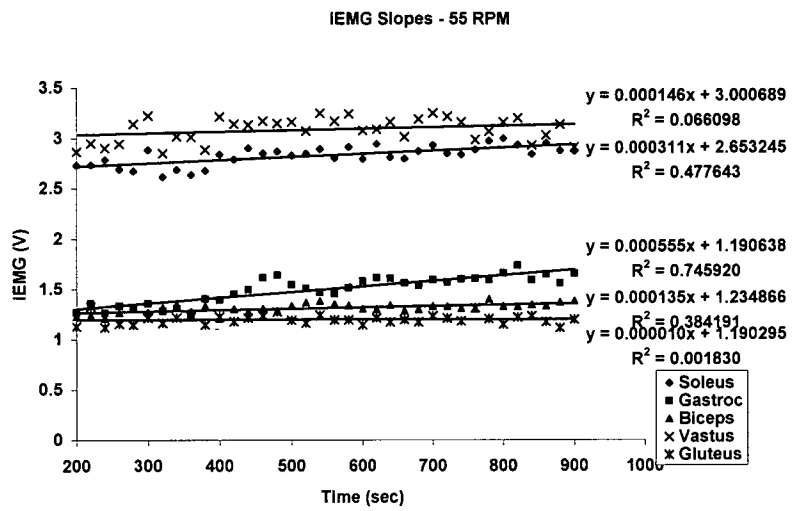



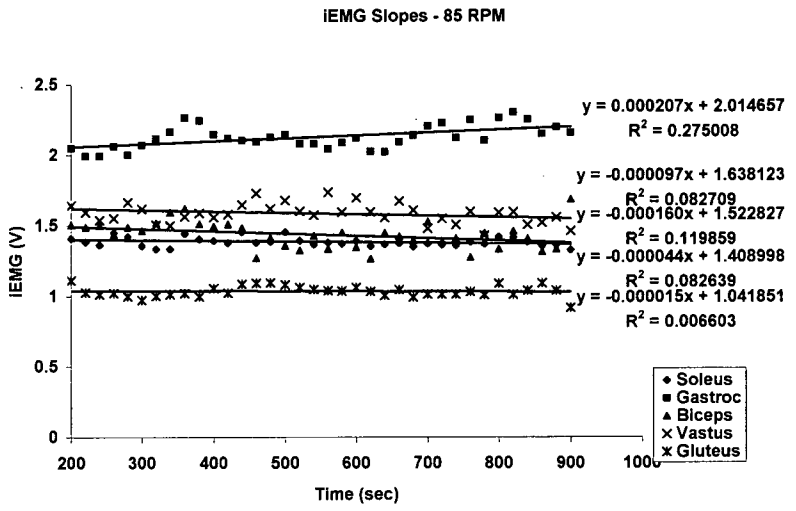


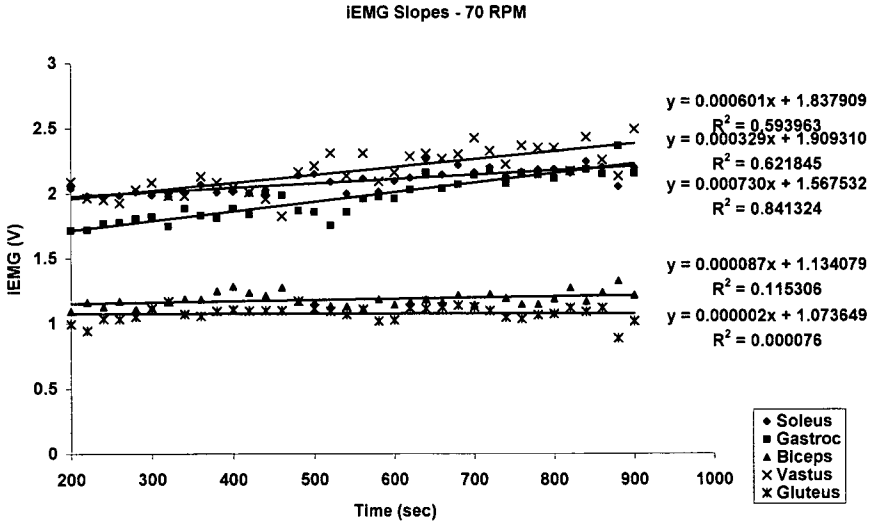

Subject 5 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions.

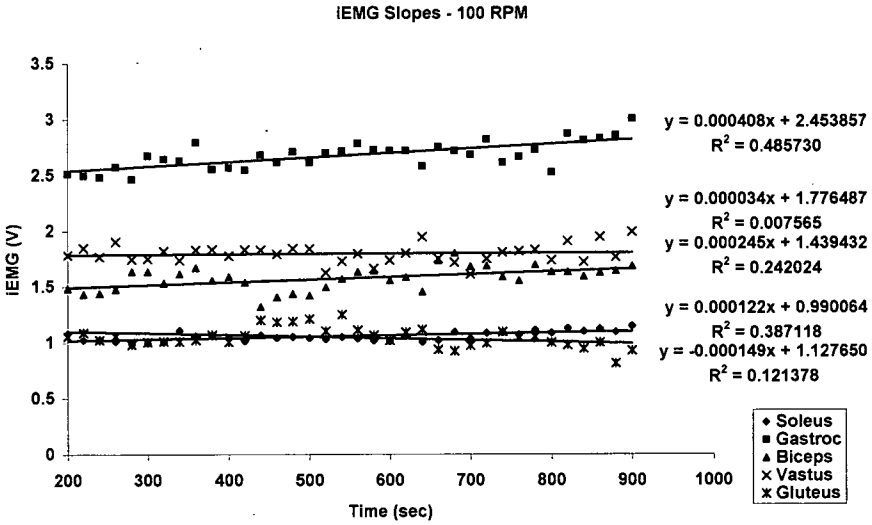



Subject 7 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions.

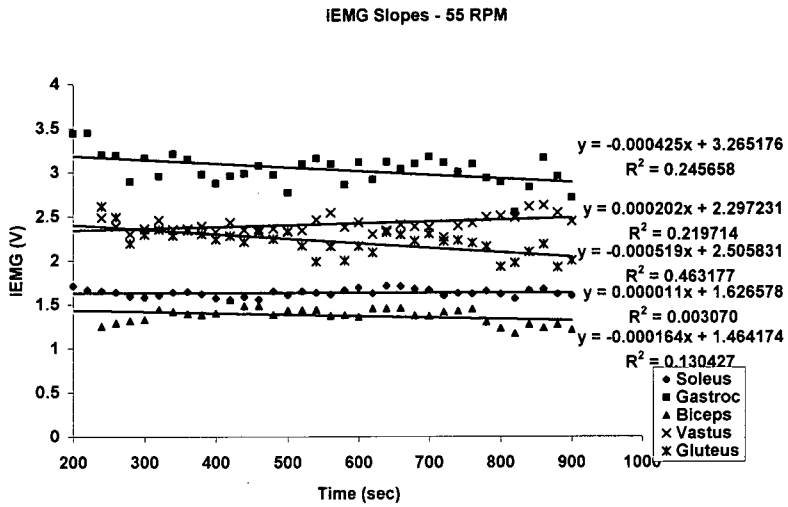


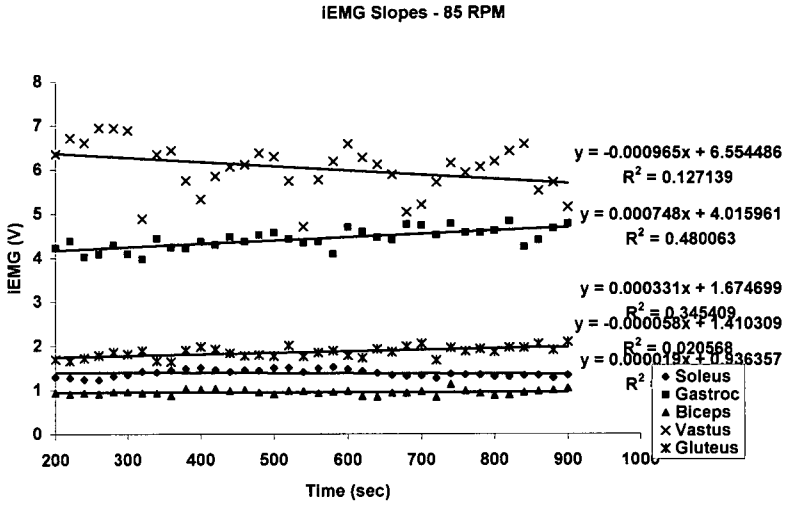



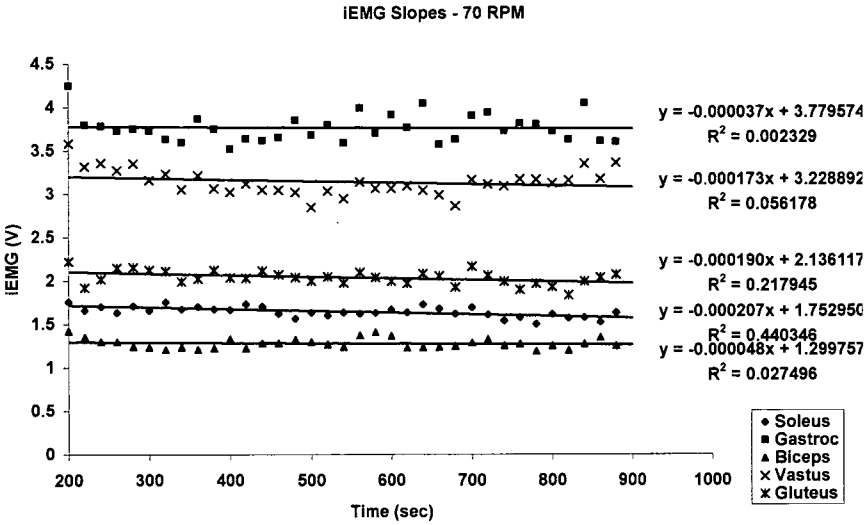



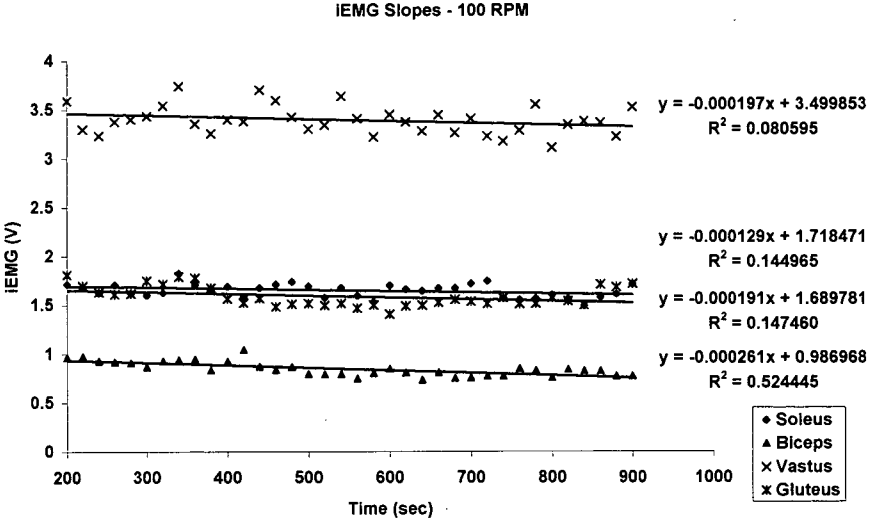




Subject 8 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions.



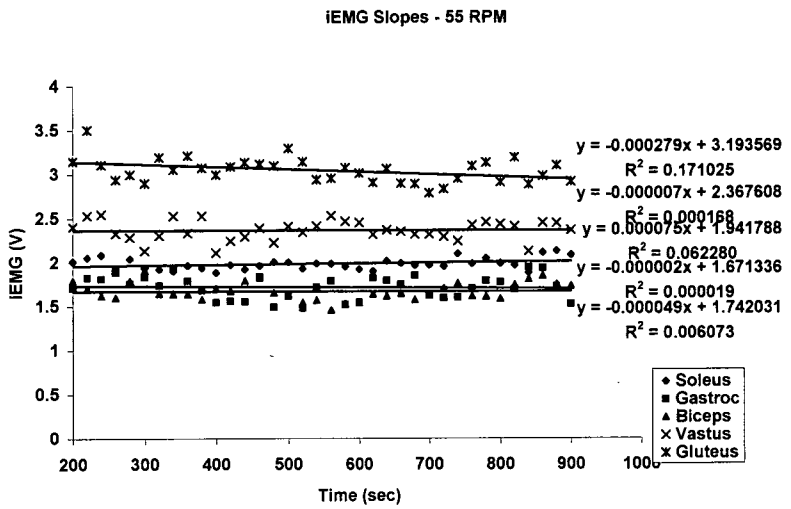



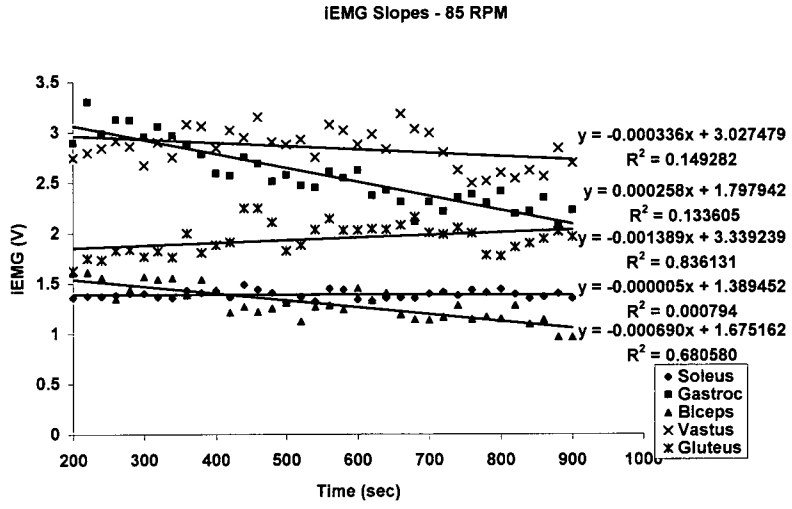



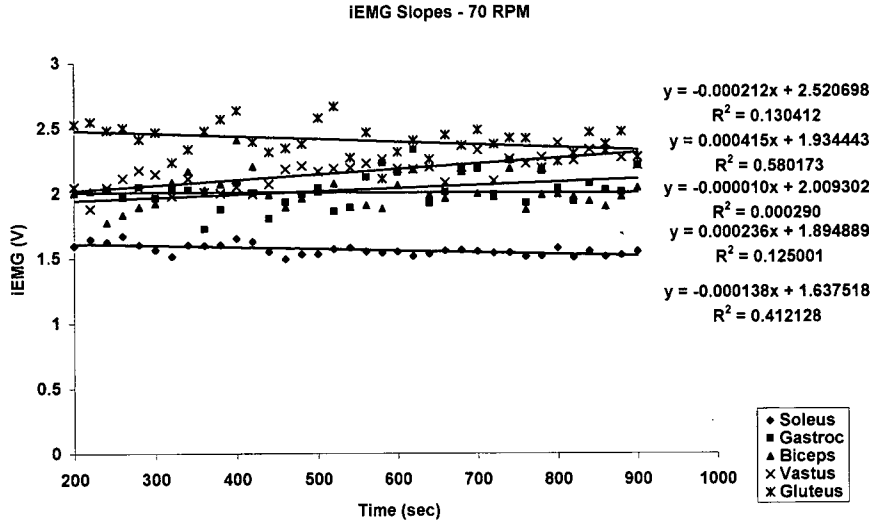



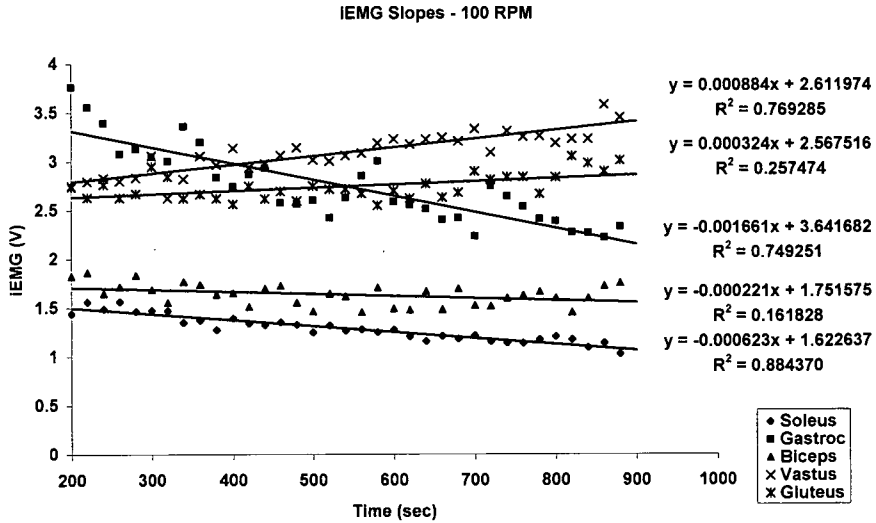



Subject 9 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions.



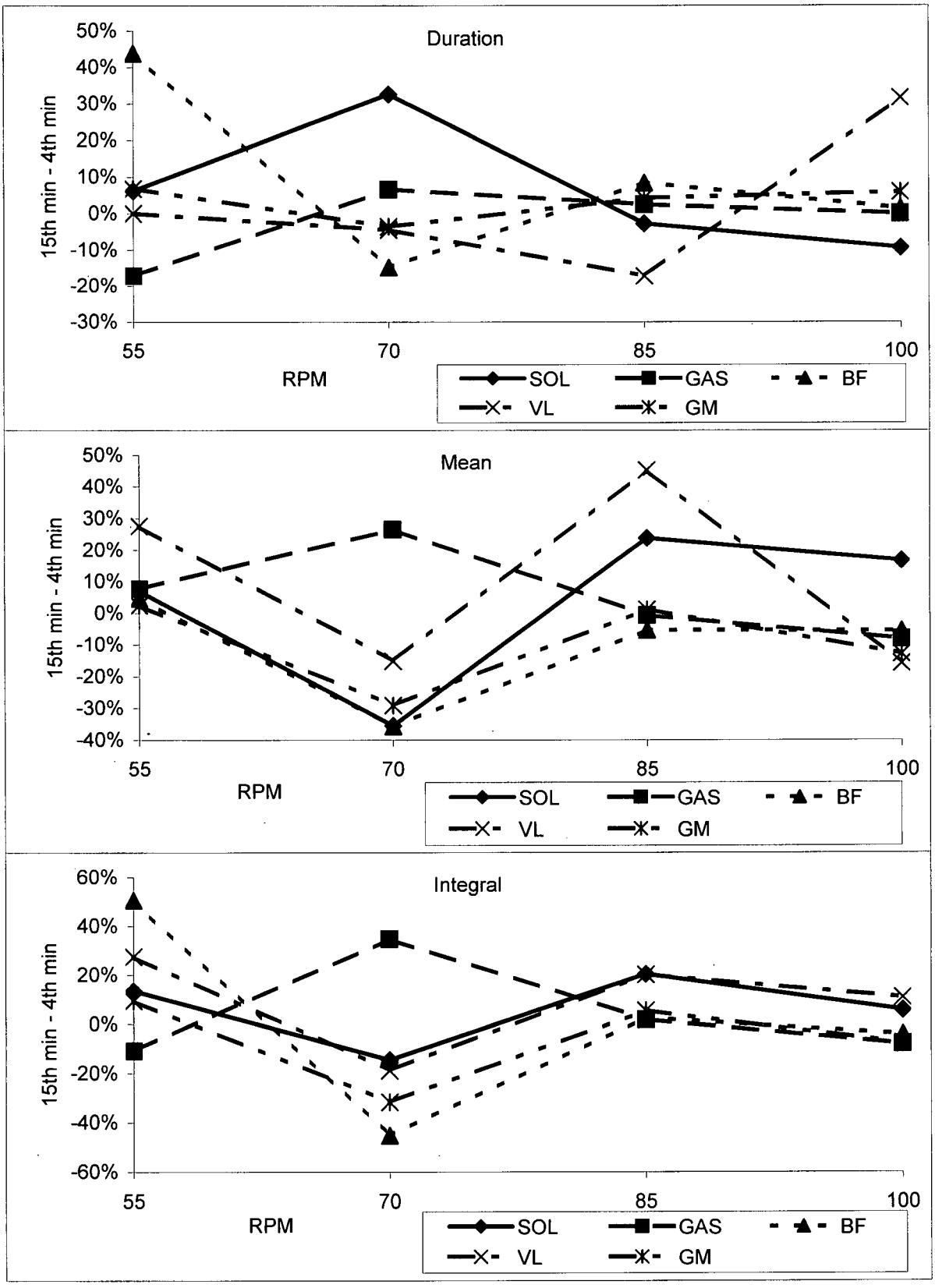



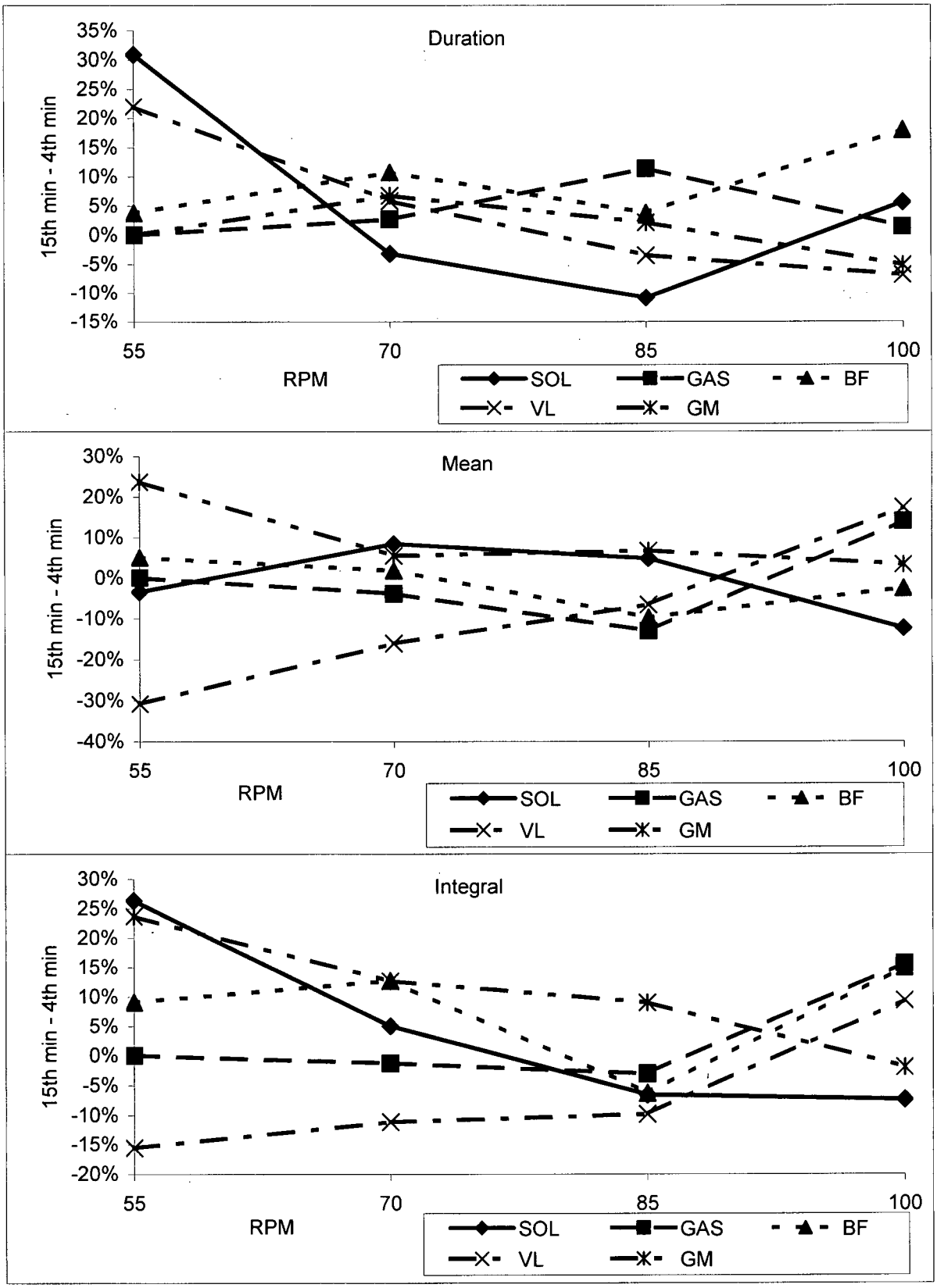



Subject 10 - iEMG slopes for sol, gas, BF, VL, and GM for 55, 70, 85, and 100 RPM cadence conditions.

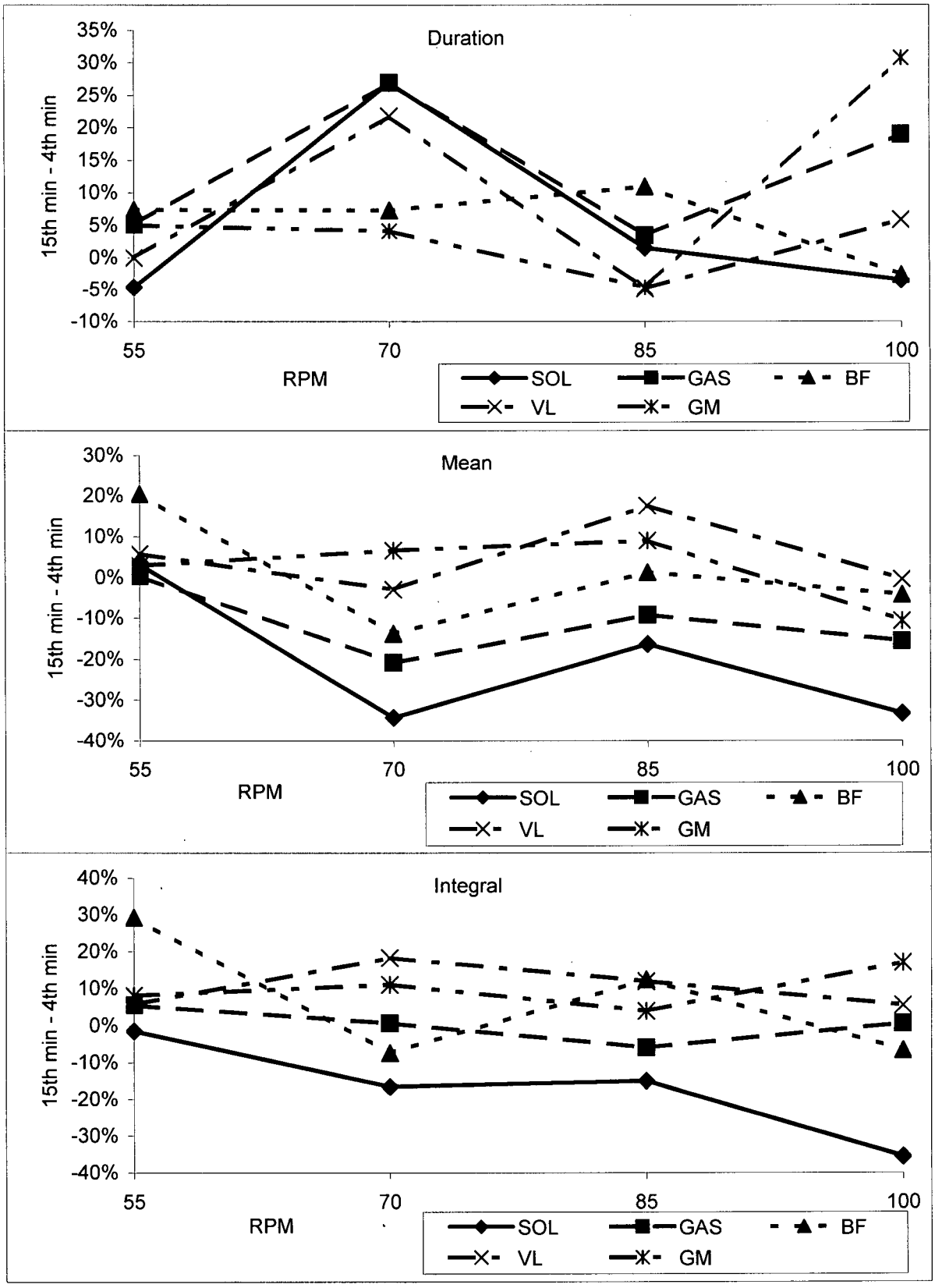
**Appendix V – Individual subject ($15^{th}$ min – $4^{th}$ min)% EMG burst durations, mean amplitudes, and integrals over cadence conditions**



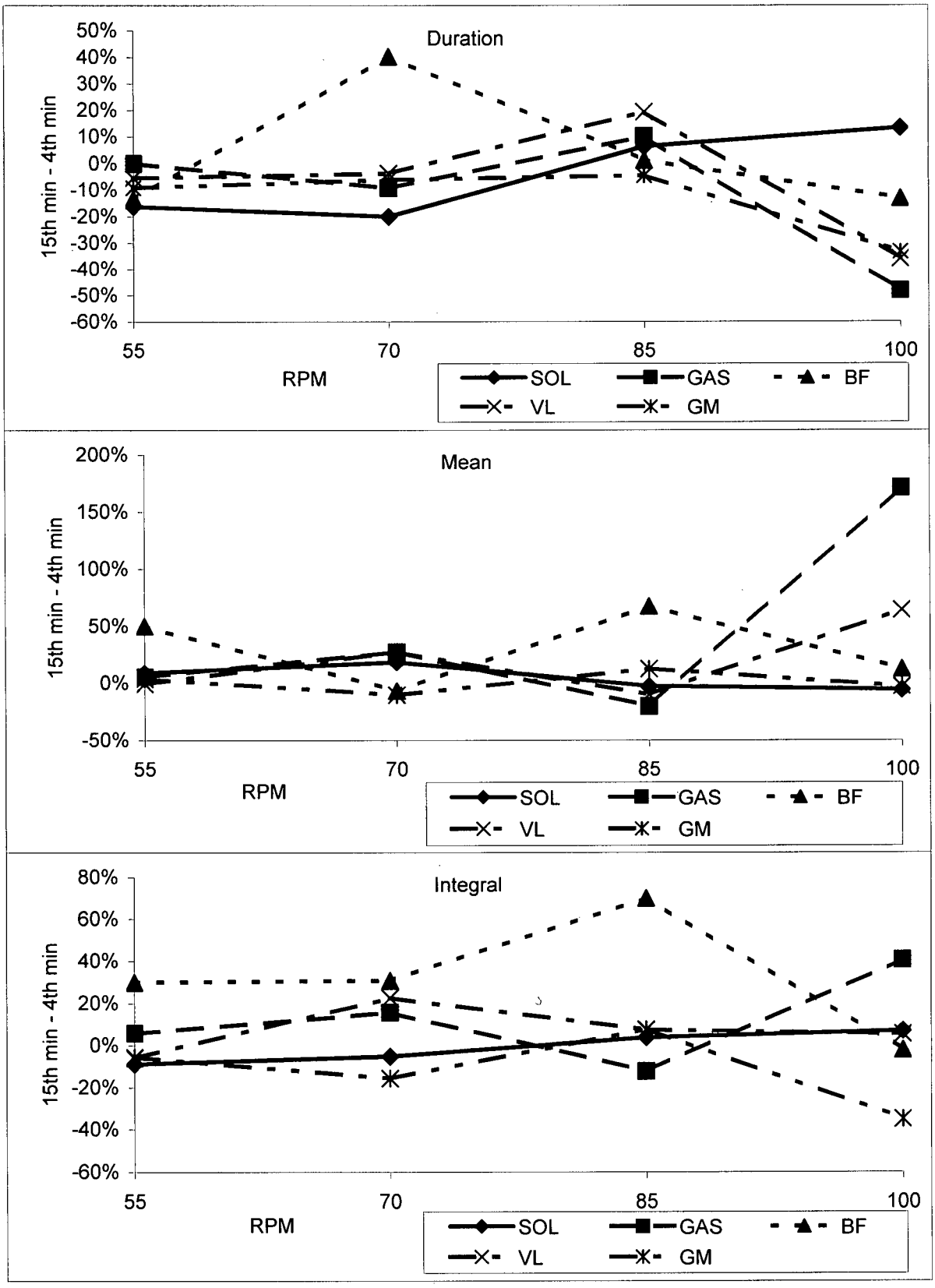

Subject 1 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.



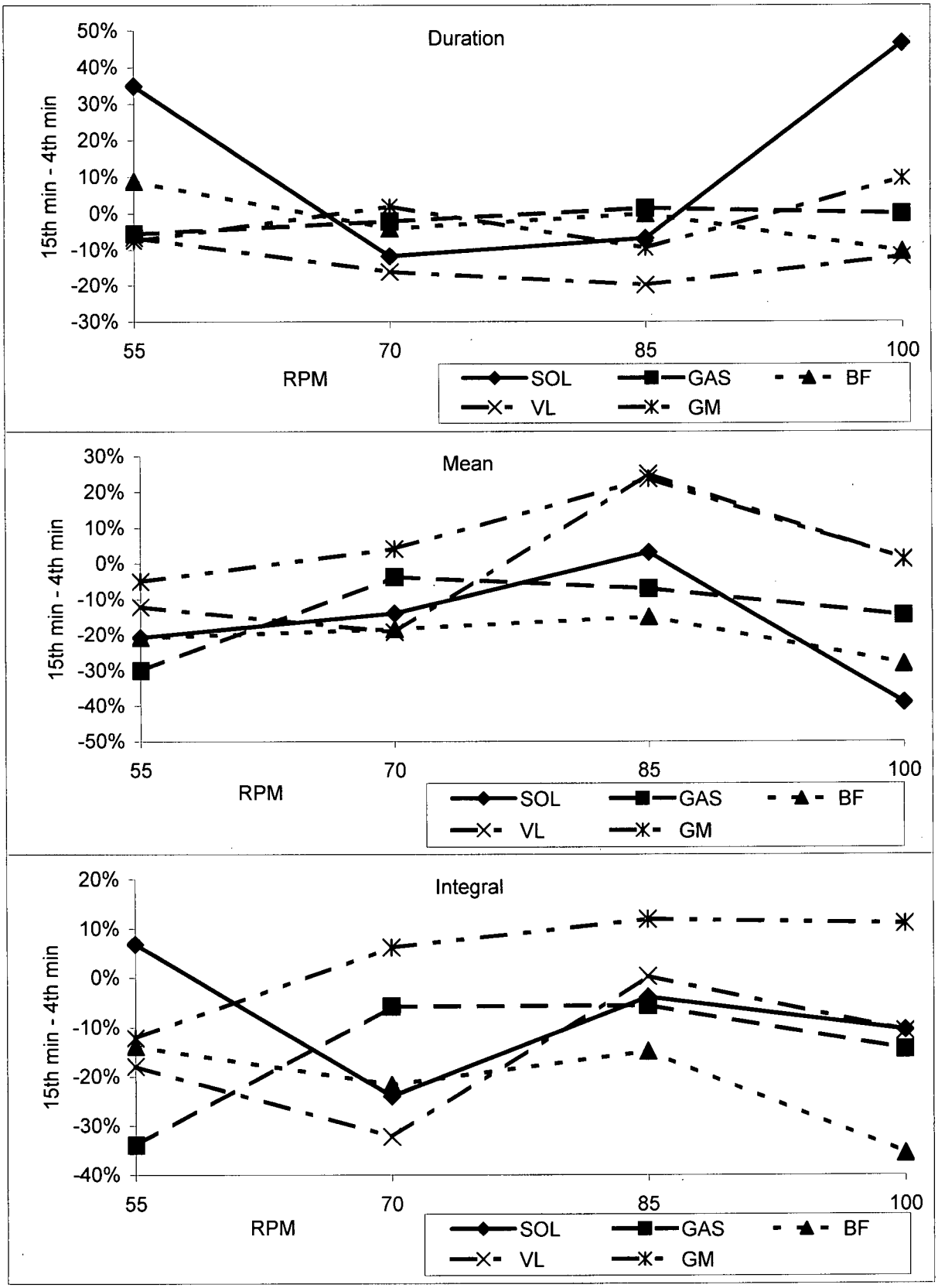

Subject 2 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.



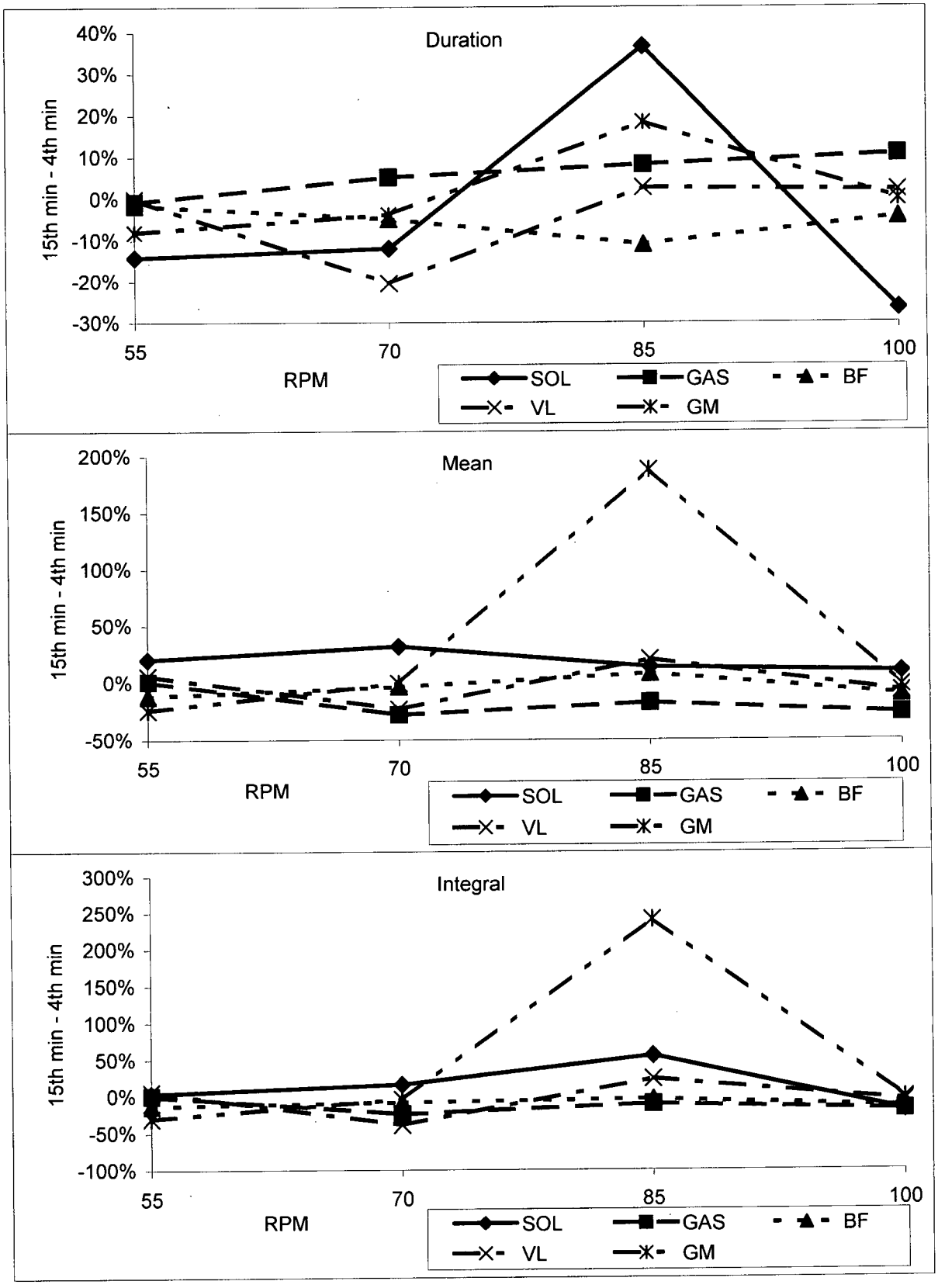

Subject 3 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.



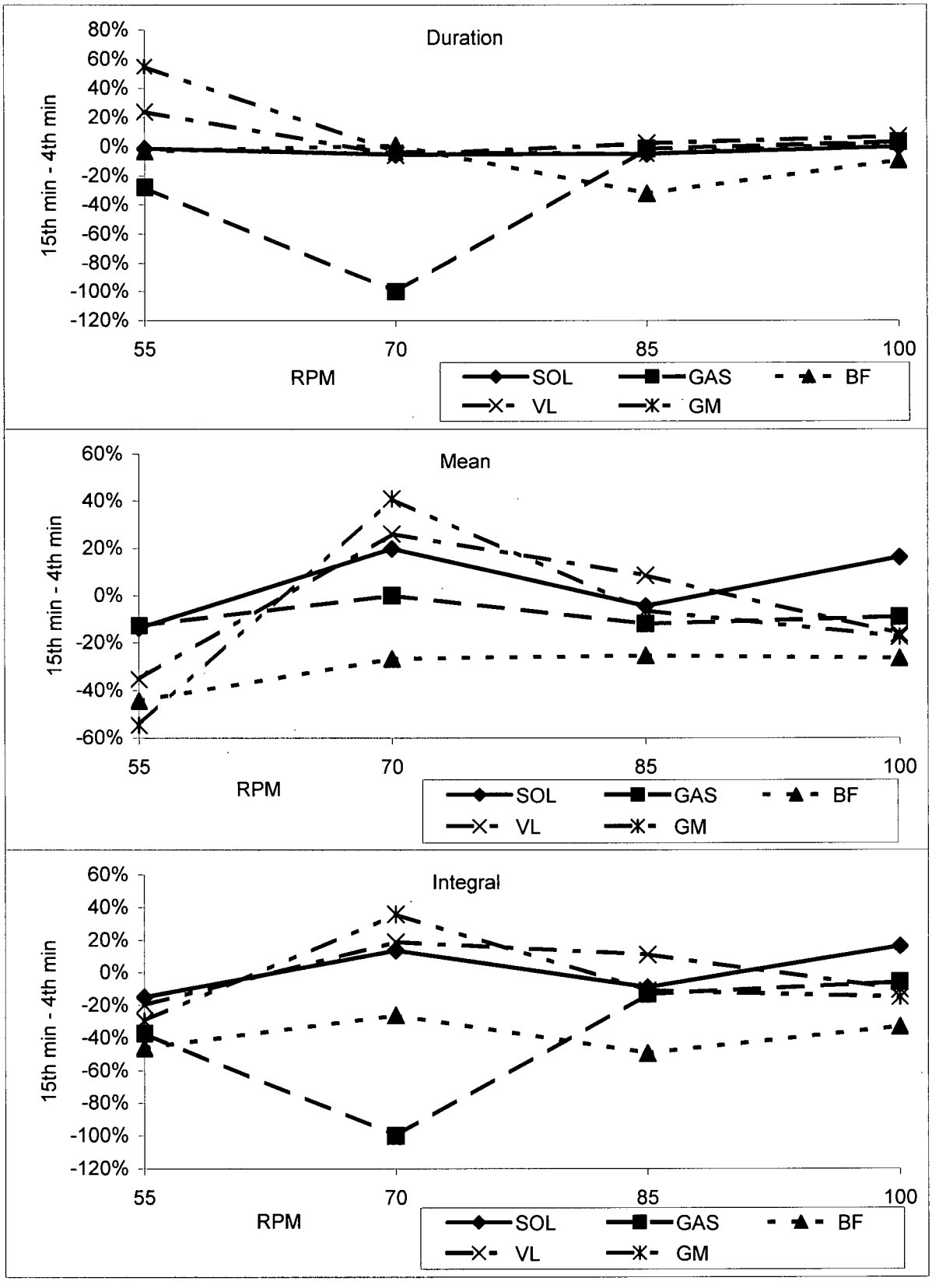

Subject 4 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.



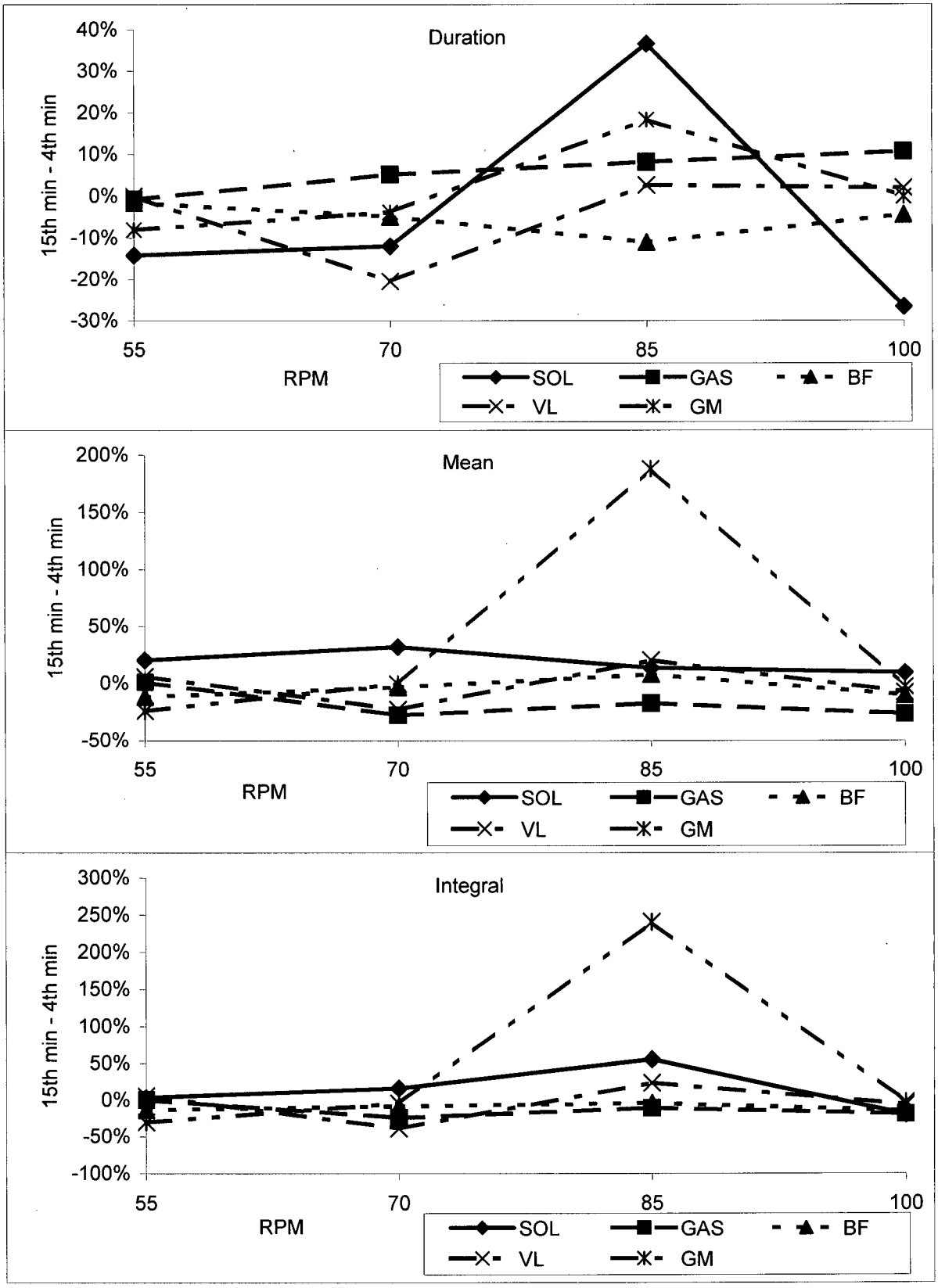

Subject 5 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.



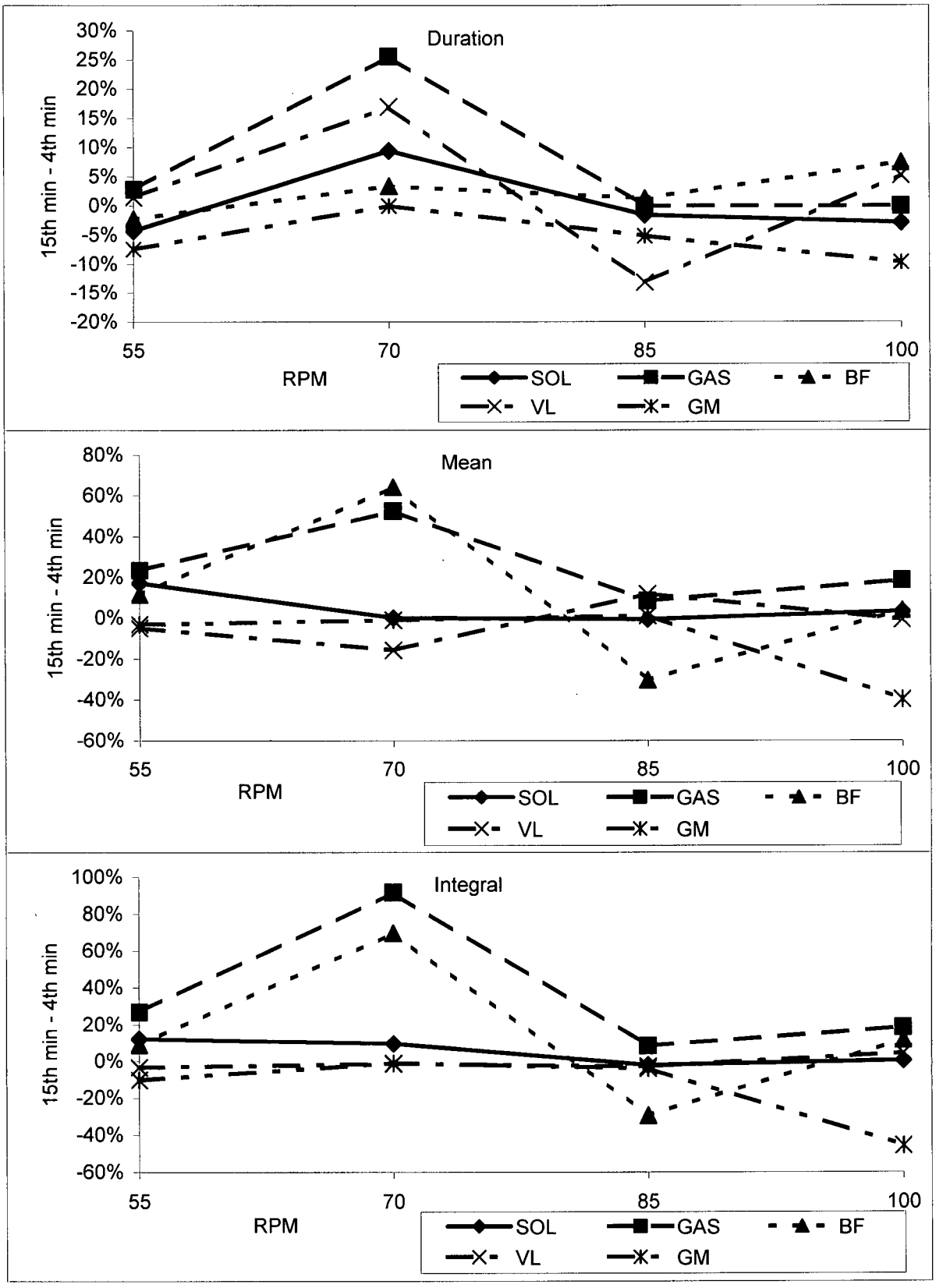

Subject 6 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.



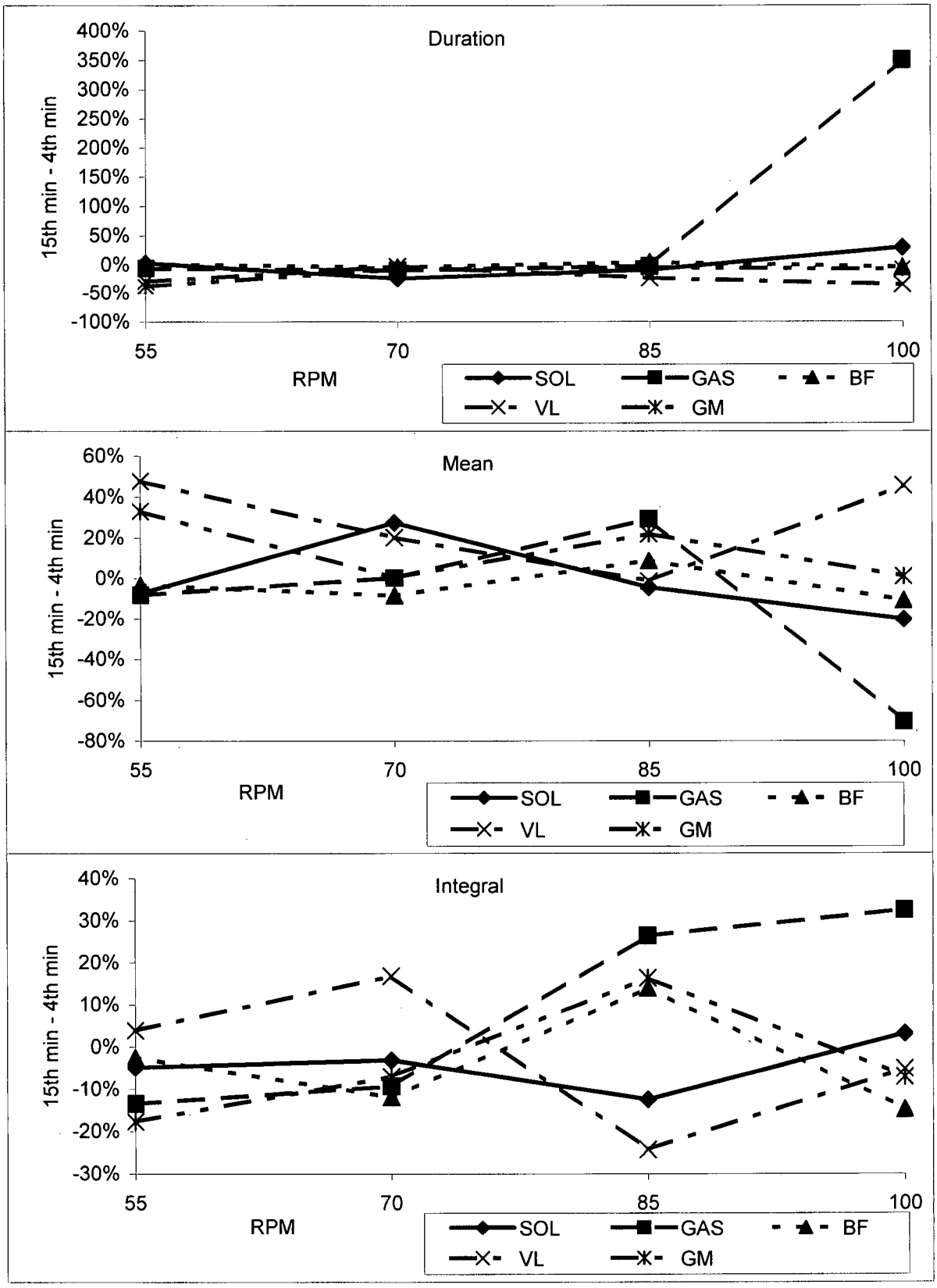

Subject 7 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.



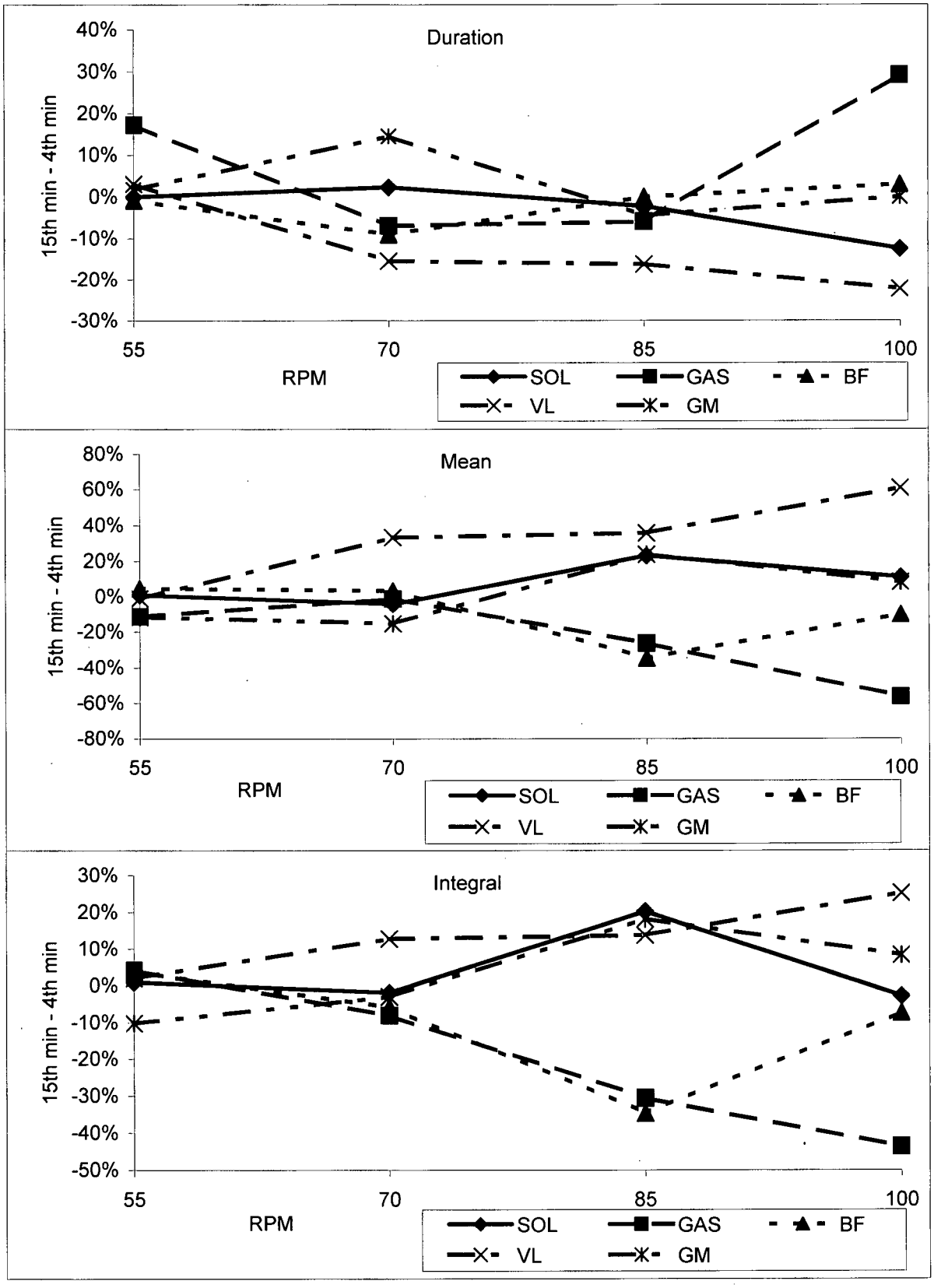

Subject 7 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.




Subject 8 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.




Subject 9 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.




Subject 10 - Average EMG burst duration, mean amplitude, and integral over cadence conditions.