Muscle recruitment patterns regulate physiological responses during exercise of the same intensity

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NUMEROUS STUDIES HAVE BEEN conducted to determine the impact of different pedaling rates on physiological responses to cycle ergometry. In general, the purpose of these investigations has been either to assess the metabolic efficiency of cycling at different cadences (5, 8, 10, 11, 20), or to identify which physiological variable is most instrumental in the selection of preferred pedaling rates (9, 15, 16). Interestingly, it has been demonstrated that neuromuscular efficiency, rather than metabolic economy, is maximized at the high pedaling cadences (80–90 rpm) favored both by trained cyclists (25) and untrained individuals (24).

To date, the investigation of the effects of different pedaling rates has typically employed constant mechanical power output, i.e., watts, eliciting exercise intensities of 70–85% of peak oxygen uptake (VO2) with exercise durations of <10 min. Perhaps due to the use of such high exercise intensities and short exercise durations, little is currently known about the consequences of various pedaling cadences on the rate of recovery after prolonged cycle ergometry. Coast et al. (4) examined recovery patterns subsequent to cycling exercise at a constant mechanical workload at disparate pedaling rates but only for 5 min after the cessation of exercise. The present study is unique in that its purpose was to determine the physiological effects of different pedaling rates during prolonged cycling of constant exercise intensity (50–55% of peak VO2) rather than mechanical power output and to examine the rate of recovery for up to 15 min postexercise. Our hypothesis was that although VO2 rates did not vary, pedaling at different rates would evoke specific patterns of quadriceps muscle activation that, in turn, would be associated with specific physiological responses to cycling exercise.

METHODS

Subjects. Ten healthy men (20.6 ± 0.4 yr, 174.9 ± 1.3 cm, 72.9 ± 3.6 kg; means ± SE) participated in the research project. None were formally trained, but all were recreationally active. After receiving a verbal description of the study, experimental procedures to be used, and potential risks involved, the subjects provided written informed consent. A physician reviewed each subject’s medical records before approving his inclusion in the investigation. All experimental procedures were approved by the Committee for the Protection of Human Subjects at The College of William & Mary.

Experimental design. Subjects initially performed a graded exercise test to volitional exhaustion on an electrically braked cycle ergometer (Excalibur Unit, Lode, Groningen, The Netherlands). The test protocol included a 3-min warm-up at 80 W followed by 2-min work intervals beginning at 140 W and increasing by 30 W at each successive stage. During testing, metabolic data were collected with an open circuit, online system (model 2900, SensorMedics, Anaheim, CA) to establish peak VO2. During this test session, subjects were allowed to self-select pedaling rate because cycling...
cadence does not affect the determination of peak \( \dot{V}_O_2 \) (22). Each subject's preferred seat height was recorded during maximal testing so that it could be replicated in subsequent submaximal test sessions. Toe clips were not used in the maximal or submaximal exercise tests. This was done to maximize reliance on the quadriceps, whose muscle activation patterns would be measured with electromyography (EMG) during the submaximal exercise bouts featuring different pedaling rates.

After the determination of peak \( \dot{V}_O_2 \), subjects returned to the laboratory to complete two submaximal exercise tests, one with a pedaling rate of 40 rpm and one at 80 rpm, at the same time of day (±1 h) in a balanced, randomized design. Submaximal tests were separated by at least 48 h but no more than 1 wk. Before each of these exercise tests, subjects were instructed to fast for 6–10 h and refrain from heavy exertion for 24 h.

On arrival at the laboratory, subjects first inserted a rectal temperature probe ~150 mm beyond the external anal sphincter (19). Then, a 20-gauge Teflon catheter fitted with a male adapter was placed in an antecubital vein and kept patent with heparin-treated isotonic saline solution. After this, the vastus medialis (VM) and vastus lateralis (VL) of the right thigh were prepared for EMG recordings. A one-square-inch area of skin over the VM and VL was shaved, abraded with fine sandpaper, and cleansed with an alcohol wipe. Along the longitudinal contour of the muscle, 2-mm-diameter surface electrodes filled with electrolyte gel were secured on the skin with adhesive collars at an interelectrode distance of 2 cm and were traced with ink. These tracings enabled electrode placement at the same sites during the second submaximal test.

The subject then mounted the cycle ergometer, was prepared for expired gas analysis by the metabolic cart, and remained still for 5 min, after which preexercise data including heart rate (HR), blood pressure, and rectal temperature were recorded. Also, a 3-ml blood sample was obtained at this time. The subject then performed a light (40 W) warm-up for 3 min at the predetermined cadence of either 40 or 80 rpm. Visual and auditory (metronome) cues were provided to assist the subject in pedaling at the proper frequency. After the warm-up, the experimenter increased the workload so that the subject's exercise intensity, i.e., \( \dot{V}_O_2 \), would be brought to 50–55% of his peak \( \dot{V}_O_2 \). The desired exercise intensity was established within 5 min and was maintained for the rest of the exercise session by altering the workload (watts) as needed. At the completion of the 30-min exercise bout, the subject remained still on the cycle ergometer for a 15-min passive recovery period. The same parameters recorded pre-exercise were also collected during the 15th and 30th min of exercise as well as 5 and 15 min into recovery. Rating of perceived exertion (RPE) was assessed at the 15th and 30th min of exercise. At 5 and 25 min of exercise, EMG recordings for a minimum of five complete revolutions of the crank shaft were collected.

Quantitation. HRs were determined with a portable telemetry unit (Cardiochamp, Sensor Dynamics, Sacramento, CA) that was secured around the subject's chest. Blood pressure was measured with a sphygmomanometer (Welch Allyn Tycos, Tycos Instruments, Arden, NC) and a stethoscope (Littmann Select, 3M Health Care, St. Paul, MN). Mean arterial pressure (MAP) was calculated as the diastolic pressure plus 33% of the difference between the systolic and diastolic pressures. This value reflects the average pressure driving blood into the tissue over the entire cardiac cycle (26). Rectal temperature was monitored with a thermistor (model 400, VWR Scientific, Bridgeport, NJ). RPE was assessed using Borg's original 15-point scale (3). Blood samples were collected into heparin-treated tubes (Vacutainer, Becton Dickinson, Franklin Lakes, NJ). Aliquots of whole blood were immediately used for hemoglobin and hematocrit analyses. Hematocrit was assayed in triplicate using microcapillary tubes after centrifugation at 4,000 g for 5 min, and hemoglobin values were determined via the cyanmethemoglobin method. Exercise-induced plasma volume shifts were ascertained from hematocrit and hemoglobin values according to Dill and Costill (7). The remaining whole blood was centrifuged at 3,000 g for 10 min. The resultant plasma was stored at −75°C until blood-borne variables were analyzed.

Plasma lactate and glucose concentrations were determined in duplicate with an automated blood chemistry analyzer (Vitros DT 60 II, Johnson and Johnson Clinical Diagnostics, Rochester, NY). Cortisol and insulin concentrations were assayed in duplicate using solid-phase 125I radioimmunoassays (Diagnostic Systems Laboratories, Webster, TX) with sensitivities of 10 and 9 pmol/l, respectively. For each hormone, all plasma samples were quantified in a single run to avoid interassay variance; intra-assay variance was <10%.

During recordings, EMG signals were amplified by a factor of 1,000 and passed through a bandwidth filter set at 30 and 500 Hz along with a 60-Hz notch filter. Signals were digitized at a sampling frequency of 1,000 Hz and recorded by an online computer system. To collect accurate, reproducible EMG data during pedaling, the cycle ergometer was modified to include a magnetic switch that emitted a pulse when the pedal reached top dead center defining one complete pedal revolution. The EMG signal was then full-wave rectified and integrated (iEMG).

Statistical analysis. Standard descriptive procedures were employed in analyzing subject characteristics. Main effects of exercise on each physiological variable of interest under each rate of pedaling were determined with repeated measures of analysis. In the event of a significant F ratio, Fisher's protected least-significant differences post hoc analysis was used to identify pairwise differences. Dependent t-tests were conducted to make direct comparisons of variables at each time point of data collection under the two cadence conditions. An alpha level of 0.05 was used to determine statistical significance.

RESULTS

Metabolic variables. Peak \( \dot{V}_O_2 \) data (51.0 ± 2.6 ml·kg⁻¹·min⁻¹; mean ± SE) indicate that although the subjects were untrained, they were reasonably fit. During the submaximal exercise sessions, subjects maintained the desired exercise intensity under both cadence conditions. At 15 min of exercise, \( \dot{V}_O_2 \) was 55% and 54% of peak \( \dot{V}_O_2 \) at 40 and 80 rpm, respectively. During the 30th min of exercise, subjects were performing at 50% of peak \( \dot{V}_O_2 \) at both cadences. Thus subjects were exercising at the same relative intensities during the submaximal exercise sessions at 40 and 80 rpm. Similarly, no cadence effects were evident during passive recovery from exercise. By 5 min postexercise, \( \dot{V}_O_2 \) had returned to preexercise levels.

Differences in pedaling rate had no effect on minute ventilation (Ve) during or after exercise. As expected, Ve was significantly enhanced during exercise, but
within 5 min after that stimulus, it had been reduced to preexercise values.

Although respiratory exchange ratio (RER) was significantly elevated during exercise, this response was similar under both cadences. Peak RER values were detected 5 min postexercise, but again they were not different between the exercise conditions. And by 15 min after exercise, whether at 40 or 80 rpm, RER was no different than before cycling. All metabolic data are presented in Table 1.

Rectal temperature. Prolonged, moderate-intensity exercise significantly increased rectal temperature, and this response persisted throughout the 15-min passive recovery period. While exercising, rate of pedaling did not affect temperature response. However, a significant drop in temperature from 5 to 15 min postexercise was observed subsequent to the 40- but not the 80-rpm session. Although statistically significant, this difference was not considered physiologically meaningful. It amounted to a disparity of 0.03°C under the two cadence conditions. Rectal temperature data can be found in Fig. 1.

Cardiovascular variables. Significant elevations in MAP were displayed while cycling at both 40 and 80 rpm, yet these exercise-induced responses were similar for both cadences. Under both conditions, blood pressure remained steady throughout the duration of exercise; no gradual increase while exercising was observed. However, it is noteworthy that there was a trend (P = 0.08) for MAP to be higher during the final minute of cycling at 40 rpm compared with the same time point during the 80-rpm session.

Repeated-measures ANOVA revealed differential main effects of cadence on blood pressure after exercise, however. Recovery of MAP was determined to be significantly slower after cycling at 40 than at 80 rpm. For example, 5 min after the session at 40 rpm, blood pressure remained significantly higher compared with preexercise, whereas MAP returned to normal within 5 min of cessation of exercise at 80 rpm. Under both cadence conditions, blood pressure at 15 min postexercise was no different than before exercise. Blood pressure responses to, and recovery from, exercise are displayed in Fig. 2.

At both 40 and 80 rpm, significantly greater HRs were detected both during exercise and recovery than before exercise. However, although HRs under the two conditions were similar midway through the exercise sessions, during the final minute of exercise, HR at 40 rpm was significantly higher than at 80 rpm. This cadence effect was maintained at 5 min postexercise. Unlike at the faster cadence, pedaling at 40 rpm appeared to have a cumulative effect; there was a significant increase in HR from the 15th to the 30th min of exercise (Fig. 3).

Plasma glucose. When measured over time, cycling cadence differentially impacted glucose response. While cycling at 40 rpm, no significant variation in plasma glucose concentrations was noted during or after exercise. In contrast, the cadence of 80 rpm resulted in significant alterations in glucose levels. Specifically, plasma glucose concentrations during exercise were lower than those at 5 and 15 min of recovery. Among the time points measured, plasma glucose concentration was greatest at 15 min postexercise. These results can be found in Fig. 4.

Table 1. Metabolic responses to and recovery from cycling exercise at different pedaling rates

<table>
<thead>
<tr>
<th></th>
<th>Preexercise</th>
<th>15 min Exercise</th>
<th>30 min Exercise</th>
<th>5 min Postexercise</th>
<th>15 min Postexercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂ (40 rpm)</td>
<td>4.9 ± 0.8</td>
<td>28.3 ± 1.2*</td>
<td>25.6 ± 1.7*</td>
<td>5.3 ± 0.6</td>
<td>4.5 ± 0.4</td>
</tr>
<tr>
<td>VO₂ (80 rpm)</td>
<td>4.7 ± 0.9</td>
<td>27.5 ± 1.3*</td>
<td>25.5 ± 1.9*</td>
<td>5.2 ± 0.3</td>
<td>4.4 ± 0.2</td>
</tr>
<tr>
<td>VE (40 rpm)</td>
<td>11.6 ± 1.6</td>
<td>53.3 ± 2.5*</td>
<td>54.6 ± 2.5*</td>
<td>16.0 ± 1.8</td>
<td>11.6 ± 1.0</td>
</tr>
<tr>
<td>VE (80 rpm)</td>
<td>11.1 ± 2.4</td>
<td>50.8 ± 2.8*</td>
<td>51.1 ± 3.4*</td>
<td>14.1 ± 1.2</td>
<td>11.1 ± 1.2</td>
</tr>
<tr>
<td>RER (40 rpm)</td>
<td>0.74 ± 0.01</td>
<td>0.85 ± 0.01†</td>
<td>0.86 ± 0.01†</td>
<td>0.91 ± 0.03†‡</td>
<td>0.77 ± 0.02</td>
</tr>
<tr>
<td>RER (80 rpm)</td>
<td>0.74 ± 0.01</td>
<td>0.83 ± 0.01†</td>
<td>0.86 ± 0.01†</td>
<td>0.89 ± 0.03†‡</td>
<td>0.76 ± 0.02</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 10. Units for oxygen uptake (VO₂) are ml·kg⁻¹·min⁻¹; units for VE are l/min. *Significant (P ≤ 0.05) difference from pre- and postexercise values. †Significant (P ≤ 0.05) difference from preexercise and 15-min postexercise values. ‡Significant (P ≤ 0.05) difference from 15-min exercise value.
Plasma lactate. In contrast to glucose, cycling both at 40 and 80 rpm elicited significant changes in plasma lactate concentrations. Under each cadence condition, plasma lactate levels were elevated at 15 and 30 min of exercise as well as 5 min postexercise. Relative to preexercise, plasma lactate remained elevated 15 min into recovery from cycling at 40 but not 80 rpm. In addition, after 30 min of exercise at 80 but not 40 rpm, there was a significant decrement in lactate within 5 min. These findings indicate that, with respect to plasma lactate, recovery is quicker after pedaling at a faster rate even though exercise-induced responses

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Fig. 2. Mean arterial pressure (MAP) responses to cycling exercise at 40 and 80 rpm. Values are means ± SE, n = 10. #Significant (P ≤ 0.05) difference from preexercise and 15-min postexercise values of same (40 rpm) trial. *Significant (P ≤ 0.05) difference from preexercise and 5- and 15-min postexercise values of same (80 rpm) trial.

Fig. 4. Plasma glucose responses to cycling exercise at 40 and 80 rpm. Values are means ± SE, n = 10. *Significant (P ≤ 0.05) difference from 15- and 30-min exercise values of same (80 rpm) trial.

Fig. 5. Plasma lactate responses to cycling exercise at 40 and 80 rpm. Values are means ± SE, n = 10. #Significant (P ≤ 0.05) difference from preexercise value of same (40 rpm) trial. *Significant (P ≤ 0.05) difference from preexercise value of same (80 rpm) trial. +Significantly (P ≤ 0.05) different value from 30 min of exercise during the 80- but not 40-rpm trial.
were similar between the two cadence conditions. Plasma lactate data are presented in Fig. 5.

**Plasma insulin.** Neither the 40- nor the 80-rpm pedaling cadence elicited significant modifications in insulin levels during exercise. Yet, in both cases, insulin levels were higher throughout recovery than they were at 15 and 30 min of exercise. At 15 min of recovery subsequent to the 80- but not the 40-rpm exercise bout, insulin was significantly greater than it was before exercise (Fig. 6).

**Plasma cortisol.** Repeated-measures ANOVA analyses revealed that only during the exercise bout at 40 rpm was significant variation in cortisol detected. Compared with preexercise values, cortisol was elevated by the 30th min of cycling at 40 rpm and remained so throughout the 15-min recovery period. Direct between-condition differences in cortisol response to cycling were also identified. That is, plasma cortisol during the slower cadence was significantly higher at the last minute of exercise and at 5 and 15 min postexercise than it was during the 80-rpm session at those same time points (Fig. 7). These differences could not be attributed to cadence-specific shifts in plasma volume because those responses were similar \( P > 0.05 \) between the 40- and 80-rpm sessions, including recovery periods. In fact, no significant plasma volume shifts were found at any time point during either of the exercise trials.

**Perceived exertion.** In general, the subjects rated their level of exertion between “fairly light” and “somewhat hard” during the moderate-intensity exercise sessions. It was revealed that pedaling rate significantly influenced RPE. At 15 min of exercise, similar RPE scores were reported for each pedaling rate. However, by the 30th min of exercise, subjects sensed their efforts to be more strenuous when pedaling at 40 compared with 80 rpm. Differences in perceived exertion are illustrated in Table 2.

**EMG recordings.** As expected, iEMG data revealed different patterns of quadriceps muscle activation under the two pedaling rates. When quantified as iEMG activity per second during the force-production phase of pedaling, muscle recruitment was significantly greater while cycling at 40 than at 80 rpm, indicating more intense muscle contraction at the slower cadence. This was true of both the VM and VL muscles and at both the 5th and 25th min of exercise. It was also determined from iEMG data that muscle recruitment patterns did not vary from the 5th to the 25th min of cycling during either pedaling rate. Data related to iEMG activity can be found in Table 3.

### DISCUSSION

The objective of this investigation was to determine whether physiological responses to cycling exercise would vary with different pedaling rates even if rela-
The intensity of the exercise stimulus are fixed. A significant increase in the RPE score was detected from the 15th to the 30th min of exercise when cycling at 40 rpm; no such increment was reported while cycling at 80 rpm. In addition, during the last minute of cycling, the RPE indicated by subjects was greater when pedaling at 40 than at 80 rpm. These distinct RPE scores may be explained by the cadence-specific differences in cardiovascular responses addressed above; recall that HR was greater during the 30th minute of the 40-rpm bout relative to the 80-rpm session.

It has been postulated that both central, e.g., HR, and local factors, including muscle force, regulate the perception of exertion during exercise (17). However, Pandolf and Noble (18) compared RPE when subjects cycled at 40 and 80 rpm while mechanical power output was held constant and concluded that local factors primarily accounted for differences in perceived exertion. Yet, in our study, although muscle activation patterns were specific to cadence throughout the 30-min exercise tests, differences in RPE were identified during the 30th but not the 15th min of exercise. Likewise, HR was similar at minute 15 but dissimilar during the last minute of exercise, whereas local muscle recruitment did not vary within either of the trials. These data suggest that, at least with prolonged exercise, HR is the variable most tightly coupled with the sensation of exercise difficulty.

The plasma lactate findings of our study confirm that, although moderate, the intensity of exercise was sufficient to result in levels of ~4.0 mM, the concentration generally used to mark the onset of blood lactate accumulation (21). It appears that relative exercise intensity, rather than intensity of muscle contraction, regulates plasma lactate response during cycling. Pedaling rates of 40 and 80 rpm resulted in similar and significant increases in circulating lactate levels during exercise. However, recovery rates of lactate to normal concentrations after exercise were specific to muscle activation patterns employed during cycling. For example, a significant decrement in lactate from the last minute of exercise to 5 min postexercise occurred during the 80- but not the 40-rpm bout. In addition, plasma lactate remained elevated longer after the slower cadence, i.e., 15 vs. 5 min.

Interestingly, cycling exercise at 40 rpm induced no significant alterations in blood glucose during or after exercise, whereas cycling at the faster rate brought about significant responses. After the 80-rpm session, blood glucose concentrations at 5 and 15 min of recovery exceeded those observed while cycling. These results may be coupled with those of postexercise lactate described above. That is, after 80-rpm exercise, a significant decrement in lactate was noted that was not apparent after cycling at 40 rpm. Previously, it has been established that lactate can serve as a substrate for the process of hepatic gluconeogenesis that occurs after exercise (1, 2). Thus the rapid reduction of lactate we detected after 80-rpm cycling exercise may account for the similarly rapid increase in plasma glucose levels that occurred after that exercise session.

<table>
<thead>
<tr>
<th>Vastus lateralis</th>
<th>Vastus medialis</th>
<th>Vastus lateralis</th>
<th>Vastus medialis</th>
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</thead>
<tbody>
<tr>
<td>40 rpm 48.1 ± 4.6*</td>
<td>49.2 ± 7.7*</td>
<td>45.7 ± 4.9*</td>
<td>45.2 ± 6.7*</td>
</tr>
<tr>
<td>80 rpm 34.7 ± 4.8</td>
<td>39.7 ± 7.3</td>
<td>33.3 ± 5.3</td>
<td>36.4 ± 6.5</td>
</tr>
</tbody>
</table>

Values are means ± SE, n = 10. Units are volts × seconds during muscle contraction. *Significant (P ≤ 0.05) difference from value of the same muscle during 80-rpm trial. iEMG, integrated electromyography.
During the 80-rpm exercise session, plasma insulin responses mirrored those of plasma glucose. Like glucose, insulin was elevated at both recovery time points compared with both time points assessed during exercise. This was not unexpected given the regulatory influence of blood glucose on insulin release from the β-cells of the pancreas (12). At 40 rpm, insulin was elevated in a pattern similar to that seen at 80 rpm (higher postexercise than during exercise), yet unlike the effects of 80-rpm cycling, the slower pedaling rate did not evoke changes in blood glucose either during or after exercise. In general, during exercise at either pedaling rate, insulin responses mirrored those of glucose. However, whereas parallel response patterns of these variables persisted during recovery from cycling at 80 rpm, such coupling was not evident after the 40-rpm trial.

Unlike insulin, different pedaling cadences resulted in markedly different responses of circulating cortisol concentrations. The greater muscle activation associated with the slower cadence elicited significant increments in cortisol both during and for up to 15 min after cycling exercise. In contrast, pedaling at 80 rpm failed to increase plasma cortisol levels. Previously, it has been reported that cortisol responses to prolonged endurance exercise are determined by intensity, i.e., cortisol increases reflect those of \( \dot{V}O_2 \) (14, 23). In the present investigation, however, exercise intensity was held constant between the two exercise trials. Our data suggest then that intensity alone, at least not as assessed by \( \dot{V}O_2 \), does not determine exercise-induced alterations in blood-borne cortisol concentration.

The fact that plasma cortisol is higher during and after the 40-rpm session than the 80-rpm bout is consistent with cadence-specific cardiovascular responses, plasma lactate responses, and perception of physical exertion. Recall that HR and blood pressure displayed more pronounced responses to 40- than to 80-rpm cycling during and/or after exercise. Also, exercise-induced increments of plasma lactate demonstrated delayed recovery after the 40-rpm trial. Subjects also reported that they experienced a greater degree of exertion while pedaling at 40 rpm. Together, these data suggest that greater stress attended the muscle recruitment pattern of the slower pedaling frequency. Stress is a primary stimulus for cortisol secretion, and elevations in blood-borne cortisol indicate the degree of stress experienced (12). Along with our cardiovascular, lactate, and RPE findings, the cortisol increases observed during and after the 40-rpm bout suggest that the muscle recruitment pattern observed with the slower cadence was associated with greater physiological stress, despite the fact that \( \dot{V}O_2 \) did not differ between the two cadence conditions.

The findings presented here suggest that relative exercise intensity (rate of \( \dot{V}O_2 \)) alone does not determine physiological responses to the stimulus of exercise. Specific patterns of muscle activation or contraction intensity also influence cardiovascular, plasma metabolite, and endocrine responses both during and after exercise. And probably due to these unique physiological responses, perceived exertion during exercise is also modulated by muscle contraction intensity, even when metabolic demands are held constant.

**Perspectives**

The rate of \( \dot{V}O_2 \) is commonly regarded as the best indicator of the intensity and physiological demand of exercise. Indeed, a strong linear relationship between HR and \( \dot{V}O_2 \) exists during prolonged physical activity. Given this relationship, many exercise adherents monitor HR to assess the intensity of the stress associated with exercise. The data presented here suggest that, in addition to metabolic factors, specific recruitment patterns of the working muscles influence the physiological responses (including HR) to the challenge of exercise. In effect, input from various organ systems acts in concert to regulate, or at least modulate, the physiological and psychophysiological stress associated with extended physical activity. On a more practical level, these findings should be considered in efforts to accurately monitor exercise intensity as well as in the prescription of exercise and fitness programs.

We express appreciation to the dedicated subjects and to Dr. Clifford Henderson for reviewing the medical records of potential subjects.

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