Comparison of hormone responses following light resistance exercise with partial vascular occlusion and moderately difficult resistance exercise without occlusion

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Reeves, Greg V., Robert R. Kraemer, Daniel B. Hollander, Jordan Clavier, Craig Thomas, Michelle Francois, and V. Daniel Castracane. Comparison of hormone responses following light resistance exercise with partial vascular occlusion and moderately difficult resistance exercise without occlusion. J Appl Physiol 101: 1616–1622, 2006. First published August 10, 2006; doi:10.1152/japplphysiol.00440.2006.—Previous studies of contracting muscle with low loading and partial vascular occlusion demonstrated hypertrophy and strength adaptations similar to and exceeding those observed with traditional moderate to high resistance (Shinohara M, Kouzaki M, Yoshihisa T, and Fukuaga T. Eur J Physiol 77: 189–191, 1998; Takarada Y, Takazawa H, Sato Y, Takebayashi S, Tanaka Y, and Ishii N. J Appl Physiol 88: 2097–2106, 2000; Takarada Y, Sato Y, and Ishii N. Eur J Physiol 86: 308–314, 2002). The purpose of the study was to determine the anabolic and catabolic hormone responses to light resistance exercise combined with partial vascular occlusion. Three experimental conditions of light resistance with partial occlusion (LRO), moderate resistance with no occlusion (MR), and partial occlusion without exercise (OO) were performed by eight healthy subjects [mean 21 yr (SD 1.8)]. Three sets of single-arm biceps curls and single-leg calf presses were completed to failure with 1-min interset rest periods. Workloads of 30 and 70% one repetition maximum for each exercise were lifted for the LRO and MR trials, respectively. Blood samples were taken preexercise, postexercise, and 15 min postexercise for each experimental condition. Lactate increased significantly in the LRO and MR trials and was not significantly different from each other at any time point. Growth hormone (GH) increased significantly by 40% from pre to postexercise in the LRO session but did not change significantly during this time period in the MR and OO trials (8.3 ± 2.3 vs. 2.1 ± 1.2 and 2.6 ± 0.94 µg/l, respectively, P < 0.05). There were no changes in resting total testosterone [T; mean 15.7 ± 1.6 (SE) nmol/l], free testosterone (FT; 54.1 ± 4.5 pmol/l), or cortisol (267.6 ± 22 nmol/l) across all trials and times. In conclusion, with similar lactate concentrations suggest what may be a fiber-type shift from previously anaerobic cell dominance (high stored ATP for immediate contraction but low muscle glycogen for endurance) to fiber types more capable of sustained work. Cortisol is a hormone that prevents protein synthesis, promotes conversion of amino acids into carbohydrate, and stimulates production of protein-degrading enzymes (29). These actions are glucose sparing. Since these actions oppose the anabolic effects of GH and T, it is important to consider cortisol’s response to resistance exercise (21, 37).

Only one study has compared GH response from light resistance with partial occlusion with partial occlusion only (33), low-workload resistance with partial vascular occlusion manifested an exaggerated GH response (40 µg/l) comparable to levels measured in traditional moderate resistance exercise with short interset rest periods. Lactic acid concentrations also increased. Since lactate has been shown to have direct and indirect effects on T synthesis (26), it is possible that T is increased in response as well. Takano et al. (31) reported significant GH response to very low workloads with partial vascular occlusion compared with the same load without partial occlusion.

Increases in muscular mass have been documented with as little as 3 wk of low resistance training with partial occlusion (1). Other studies report strength adaptations and hypertrophy after training with similar protocols (30, 32, 34, 35). In an attempt to elucidate some of the possible metabolic adaptations that may be implicated as explanation for partial occlusion findings, Burgomaster et al. (6) compared the resting values of stored ATP and glycogen in muscle before and after 8 wk of low-intensity (~50% one repetition maximum (1 RM)) biceps curls isometric resistance training with and without partial occlusion. There were increases in stored glycogen and decreases in stored ATP for both groups, with a more exaggerated result in the partially occluded group. The changes in ATP and glycogen concentrations suggest what may be a fiber-type shift from previously anaerobic cell dominance (high stored ATP for immediate contraction but low muscle glycogen for endurance) to fiber types more capable of sustained work. Cortisol is a hormone that prevents protein synthesis, promotes conversion of amino acids into carbohydrate, and stimulates production of protein-degrading enzymes (29). These actions are glucose sparing. Since these actions oppose the anabolic effects of GH and T, it is important to consider cortisol’s response to resistance exercise (21, 37).

RESISTANCE TRAINING WITH MODERATE Workloads stimulates increases in muscular strength and size (17, 21, 28). Knowledge of relative contributions to these training adaptations by the anabolic hormones testosterone (T) and growth hormone (GH) is incomplete, but they are thought to be influential on the particular mechanisms that enhance hypertrophy and strength (2, 3, 5, 11, 14, 24, 27). In a previous study by Takarada et al. (33), low-workload resistance with partial vascular occlusion manifested an exaggerated GH response (40 µg/l) comparable to levels measured in traditional moderate resistance exercise with short interset rest periods. Lactic acid concentrations also increased. Since lactate has been shown to have direct and indirect effects on T synthesis (26), it is possible that T is increased in response as well. Takano et al. (31) reported significant GH response to very low workloads with partial vascular occlusion compared with the same load without partial occlusion.

In conclusion, with similar lactate concentrations suggest what may be a fiber-type shift from previously anaerobic cell dominance (high stored ATP for immediate contraction but low muscle glycogen for endurance) to fiber types more capable of sustained work. Cortisol is a hormone that prevents protein synthesis, promotes conversion of amino acids into carbohydrate, and stimulates production of protein-degrading enzymes (29). These actions are glucose sparing. Since these actions oppose the anabolic effects of GH and T, it is important to consider cortisol’s response to resistance exercise (21, 37).

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workload administered was typically low (32, 34, 35) despite the traditional view that hypertrophy occurs only as an adaptation to moderate-load protocols. The rationale for lower workloads in research investigating GH, skeletal muscle hypertrophy, and strength adaptations is partially due to previous findings that significant increases of all three occur as a result of low-intensity work combined with partial occlusion (30, 32–34). Furthermore, largely immobile patients of pulmonary or vascular disease (15) have exhibited reduced atrophy and even hypertrophy in leg muscle, suggesting that hypoxic or ischemic conditions may directly contribute to these adaptations. This particular low workload and occlusive training could be an advantage with diseased patients of compromised ambulation.

The purposes of this study were to determine the hormonal responses of GH, T, free testosterone (FT), cortisol, and lactate concentrations to low-workload resistance exercise with partial vascular occlusion and to compare these responses with those of moderate exercise without occlusion and partial occlusion alone.

MATERIALS AND METHODS

Research design. The study employed a preliminary session and three different exercise and/or partial occlusion sessions that allowed different effects of resistance load and partial vascular occlusion on anabolic and/or catabolic hormones to be compared using the same subjects. Subjects completed the four sessions on separate days. The first session served as a preliminary session to record descriptive characteristics, familiarize subjects with the equipment, and determine 1-RM maximum resistances. This was followed by either a light resistance partial occlusion (LRO) session or a moderate resistance (MR) without occlusion session, in counterbalanced fashion. The final session was always the partial occlusion-only (OO) session so that the same duration of partial occlusion could be used as in the LRO session. The duration of partial occlusion varied for each individual depending on number of repetitions performed to failure in the LRO session. All experimental sessions began at 1100, a time of day chosen to account for normal diurnal hormone secretion.

Subjects Eight adult men were recruited from the student population at Southeastern Louisiana University. The investigation was approved by the Southeastern Louisiana University Institutional Review Board and was conducted in accordance with the Declaration of Helsinki. Individual criteria for participation included age within the range of 18–30 yr and past experience in weight training of at least 1 yr so that hormonal responses were not affected by physical stress typically experienced when first engaging in resistance exercise. Exclusion from the study was based on the following criteria: 1) current use of ergogenic aids such as creatine monohydrate, herbal stimulants, or any anabolic agents such as steroids; 2) cardiac or circulatory ailments, including high blood pressure and/or hypertension (to avoid complications due to the hemodynamic alterations caused by partial vascular occlusion); 3) presence of metabolic diseases that could affect hormonal responses, e.g., diabetes and Cushing’s disease; and 4) participation in reduced-caloric-intake diets that may alter basal hormonal levels.

Potential subjects were required to fill out a medical history questionnaire before acceptance to ensure that there were no existing health risks. Subjects read and signed written consent forms and were made aware of the potential risks of the study. Subjects were asked to refrain from resistance and cardiovascular exercise on the day before each experimental trial and to abstain from caffeine and alcohol for 12 h before each trial. Furthermore, the principle investigator provided each subject with a standardized caloric beverage (Naturite, Jacksonville, FL) to consume 3 h before each experimental session (total calories per serving: 250, carbohydrates: 40 g, protein: 9 g, fat: 6 g) and to cease all other food or caloric drink ingestion until session completion.

Preliminary session (session 1). Subjects participated in a preliminary session within which anthropometric data were assessed, and an experimental resistance load was determined from a series of 1-RM lifting attempts. Height, weight, age, and body fat percent were recorded; a three-site (chest, abdomen, thigh) skinfold measurement with skinfold calipers (Lange Instruments, Santa Cruz, CA) was used to calculate body fat percent (16). A 1-RM value was assessed (9) in the single-arm biceps curl and single-leg calf extension, closely monitored by the investigator for acceptable lifting form and complete range of motion. These two particular exercises were chosen for several reasons: 1) the biceps have been used in previous occlusive studies (6, 32) due to its ease of occlusive application and exercise familiarity; 2) the gastrocnemius represents a muscle of the lower limbs that has comparable cross-sectional area to the biceps, simple plantar flexion action, and a convenient occlusive application area proximal to the muscle; and 3) two muscle groups (though proportionately small to moderate size) represent a protocol closer to typical exercise sessions than utilizing just one muscle group.

The biceps curl was performed with the dominant arm, utilizing a free-weight dumbbell. Subjects stabilized their upper torso by grasping a stationary structure with their nondominant hand to ensure proper form and minimize momentum. Three trials of submaximal weight approximating 40, 60, and 80% of the subject’s estimated 1 RM served as specific warm-up sets, interspersed with rest intervals of 3 min. A 1-RM determination was made when the subject successfully curled the heaviest dumbbell through the full range of motion with no other body manipulation to enhance momentum or swinging. The calf extension was performed on the Body Masters (Rayne, LA) leg press device, using the same dominant side of the body. Similarly, the 1-RM determination was recorded as the highest resistance successfully plantar flexed without any assistance from the quadriceps muscles after no fewer than three warm-up sets. Experimental session workloads were then calculated as 30% 1 RM for the LRO session and 70% 1-RM for the MR session.

LRO session. A blood pressure reading was taken from the arm after 15 min of semirecumbent resting. Fifteen minutes before testing, a resting blood sample was collected from the antecubital space of the contralateral arm (nondominant side). A custom-designed narrow inflatable cuff was affixed to the dominant arm, centered in the space between the superior aspect of the biceps brachii and the inferior aspect of the anterior deltoid muscles. The cuff was inflated to a pressure of 20 mmHg below the acute edema past the vascular cuff pressure. If a pulse was not detected at the finger, the entire duration of occlusion was recorded for use as the time length of cuff maintenance in the OO period. A pulse oximeter (model 3301, Smiths Medical PM, Waukesha, WI) was employed immediately after each set to failure to ensure that blood flow was not completely halted by tissue edema past the vascular cuff pressure. If a pulse was not detected, cuff pressure was reduced ~5–10 mmHg until blood flow was detected at the finger. The entire duration of occlusion was recorded for use as the time length of cuff maintenance in the OO condition of the third experimental session (mean arm occlusion time was 341 ± 4.5 s). After cuff removal, the subject moved to the leg press device, and the inflatable cuff was then applied to the proximal portion of the lower leg on the dominant side, centered in

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the space between the superior aspect of the gastrocnemius and the inferior edge of the patella. The cuff was inflated once again, exactly 5 min after its removal from the arm, to a pressure of 40 mmHg above the arm occlusive pressure to account for the larger vasculature and muscle mass. It was maintained for the duration of three sets of calf extensions to failure and their corresponding interset rest periods. In a similar fashion to the biceps curl portion of the session, a pulse oximeter was affixed to the toe of the subject to ensure some blood flow between sets. Cuff pressure was lowered once again if a pulse was not detected. After the final set was completed, there was an additional minute of recovery while the inflated cuff remained in place (mean leg occlusion time was 387 ± 13.1 s). After deflation and removal of the cuff, the subject was asked to sit for a second blood draw within 1 min of testing completion (collected from the dominant side) and to remain seated for another 15-min period. The third and final blood draw occurred 15 min postexercise.

MR session. The second experimental session was conducted in identical fashion to that of the LRO session except no partial vascular occlusion was applied. The session resembled the resistance protocol of traditional weightlifting for strength and muscle mass gains by employing loads of ~70% 1 RM for both biceps curls and calf extensions (dumbbells and weight stack resistances were adjusted as closely as possible to approximate 70% of the resistance lifted maximally in the preliminary session). As in the LRO session, a blood draw occurred 15 min before the beginning of the first lift. The subjects performed three sets of single-arm biceps curls interspersed with 1-min rest intervals, followed by 1 min of recovery. Three sets of single-leg calf extensions were then completed with the same resting intervals. All sets progressed to failure (which was determined by the first incomplete repetition), and good lifting form was maintained throughout. In the same manner as the LRO session, a blood draw was taken immediately following exercise and 15 min postexercise.

OO session. The protocol of the OO session followed the exact time intervals of the LRO session without any actual load lifting. A pretrial blood draw began the session, and the inflatable cuff was affixed to the same upper arm location with a pressure of 20 mmHg below the acute systolic pressure. The subject was asked to stand for the duration of time recorded from the biceps curl portion of the LRO session, including the interset rest periods and final recovery minute. The cuff was then deflated and removed, and the subject was seated with legs in the supine position, emulating the posture maintained during the calf extension portion of experimental session 1. Within the same time period recorded in session 1, the cuff was applied to the exact position of the lower leg used in LRO and inflated to 40 mmHg above the OO arm occlusion pressure. In a similar manner, the cuff remained inflated at the leg for the duration recorded in LRO. At the completion of this, the subject was seated normally, and the next blood draw was obtained. Another 15 min of relaxed sitting were followed by the final blood draw.

Analyses. Serum concentrations of GH, T, and cortisol were determined by chemiluminescent assay (Immulite, Diagnostics Products, Los Angeles, CA) and FT was determined by radioimmunoassay (Diagnostic Systems Laboratories, Webster, TX), while lactate concentration was determined using a colorimetric, enzymatic method (Trinity Biotech, St. Louis, MO) and analyzed spectrophotometrically. Hematocrit was determined using a microhematocrit method. Hemoglobin was analyzed with a colorimetric, enzymatic assay (Trinity Biotech, St. Louis, MO) and analyzed spectrophotometrically. Intra-assay variability coefficients of variation for GH, T, FT, and cortisol were 8.8, 7.6, 5.44, and 5.6%, respectively. Intra-assay coefficient of variation was <5.0%.

Trial (3) × time (3) repeated-measures ANOVAs were used to determine changes in hormone and lactate values across time and between trials. Dependent t-tests were used for post hoc analyses where appropriate. Analyses were conducted using SPSS 10.0 for Windows software.

RESULTS

Anthropometric and descriptive characteristics of the subjects for age, height, weight, and body fat were mean 21.8 yr (SD 1.4), 179.2 cm (SD 6.5), 85.9 kg (SD 6.5), and 9.6% (SD 3.4), respectively. Mean values of biceps curl and calf press 1 RM as well as 30 and 70% resistance loads used for the LRO and MR session, respectively, are shown in Table 1. Mean values for number of repetitions performed per set in the LRO and MR session are shown in Table 2.

There were no significant shifts in plasma volume for the three trials. Plasma volume shifts (means ± SE) were −0.5 ± 2.3, 0.9 ± 1.8, and 7.4 ± 3.1% from preexercise to postexercise for the LRO, MR, and OO trials, respectively. Plasma volume shifts were 2.8 ± 1.5, 3.7 ± 1.3, and −0.4 ± 2.5% from postexercise to 15 min postexercise for the LRO, MR, and OO trials, respectively. Thus all changes in plasma volume were under 10%.

There was a significant time effect, trial effect, and time × trial interaction for lactate (P < 0.001; Fig. 1). The pooled trial means for postexercise and 15 min postexercise lactate concentrations were significantly higher than preexercise values. Post hoc analysis for individual conditions showed that lactate increased significantly in the LRO and MR trials from preexercise to postexercise (P = 0.00 for both comparisons) but not in the OO session (P = 0.67). Moreover, lactate concentrations at 15 min postexercise were significantly higher than preexercise values in the MR (P = 0.00) but not for the LRO or OO conditions; however, 15 min postexercise lactate concentrations in the MR and LRO trials were not significantly different from each other. Examination of post hoc power estimates indicated that lactate primarily varied as a function of time (n² = 0.76, observed power = 1.0), followed by time × trial interaction (n² = 0.62, observed power = 1.0) and trial (n² = 0.6, observed power = 0.99).

There was a significant time effect (P < 0.001), trial effect (P < 0.05), and time × trial interaction for GH (P < 0.001). Pooled trial means revealed a significant increase from preexercise to postexercise values, as well as preexercise to 15 min postexercise values. Post hoc analyses indicated that the LRO condition elicited a significant increase in GH from preexercise to postexercise (P = 0.012) and approached but was not significant from preexercise to 15 min postexercise exercise (P = 0.07; Fig. 2). For MR the mean GH concentrations from preexercise to postexercise and from preexercise to 15 min postexercise approached significance (P = 0.16, P = 0.06, respectively). LRO postexercise GH concentrations were significantly greater.

Table 1. Biceps curl and calf press 1 RM, 30% 1 RM, and 70% 1 RM

<table>
<thead>
<tr>
<th>Exercise</th>
<th>1 RM, kg</th>
<th>30% 1 RM, kg</th>
<th>70% 1 RM, kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps curls</td>
<td>23.3 (2.5)</td>
<td>7.4 (1.1)</td>
<td>16.2 (2.3)</td>
</tr>
<tr>
<td>Calf press</td>
<td>130.7 (16.1)</td>
<td>39.5 (5.1)</td>
<td>91.8 (10.5)</td>
</tr>
</tbody>
</table>

Values are means (SD) for 8 subjects; 1 RM, one repetition maximum.
than OO postexercise ($P = 0.013$), but MR postexercise was not significantly greater than OO postexercise ($P = 0.791$). Thus the LRO trial produced greater GH responses than both the MR and OO trials. Power estimates indicated the highest value for time (0.98) compared with time x trial interaction and trial (0.79 and 0.62, respectively). The $\eta^2$ values were comparatively similar among all effects at 0.27, 0.27, and 0.3 for time, interaction, and trial, respectively.

There was no significant time effect for $T$ concentrations (Fig. 3). Furthermore, no trial and time x trial interaction was revealed. The $\eta^2$ and observed power for time, time x trial, and trial, respectively, were 0.03/0.13, 0.04/0.12, and 0.09/0.22. There was no significant time effect for FT concentrations (Fig. 4). Additionally, no significant trial and time x trial interaction was found. In a similar manner to $T$, $\eta^2$, and observed power for time, interaction, and trial were 0.05/0.21, 0.06/0.21, and 0.09/0.21, respectively, for FT.

There were no significant time, interaction, or trial effects for cortisol (Fig. 5). The $\eta^2$ and observed power for time, interaction, and trial were 0.08/0.36, 0.16/0.55, and 0.06/0.14, respectively.

**DISCUSSION**

In the present study, we compared the anabolic and catabolic hormone responses to light resistance exercise and partial vascular occlusion with those from moderate workload without occlusion (deemed to produce similar metabolic stress) and occlusion only. As designed, we found similar lactate responses between the LRO and MR trials, indicating the same metabolic stress produced by both trials. However, LRO elicited a greater GH response than MR, but $T$, FT, and cortisol were not affected. Lactate response was a particular point of interest because of the reported direct effects on GH and $T$ secretion and due to use of partial vascular occlusion, which is known to trap local lactate production and limit its metabolism (13). We hypothesized that lactate concentration would increase to a greater extent in the LRO session compared with the OO session and that it would match or exceed the response measured in the MR session. These two points were supported because the LRO session lactate response did in fact match and exceed those measured in the MR session and the OO session, respectively. Although the LRO and MR sessions resulted in similar lactate concentrations, there was a greater GH response to the LRO sessions. We think that this provides evidence that the difference in GH responses between the two trials was not due to lactate but from other mechanisms. Moreover, it corroborates that the metabolic stress produced during the two sessions was similar, which was in accord of the methodological design of the two sessions.

There was no change in lactate concentration between any time points across the OO session. It was thought that application of partial vascular occlusion would limit the removal of resting lactate and that it would elicit a plasma concentration greater than that observed before cuff application. This was not observed during the OO session, nor was there change in GH

![Fig. 1. Lactate concentrations during the experimental sessions for the light resistance partial occlusion (open bars), moderate resistance (hatched bars), and partial occlusion only (stippled bars) sessions. Values are means ± SE. Pre, preexercise; Post, postexercise; 15 Post, 15 min postexercise. *Significant difference compared with the partial occlusion only session for the same time period, $P < 0.05$. †Significant difference compared with preexercise values, $P < 0.05$.](image1)

![Fig. 2. Growth hormone concentrations during the experimental sessions for the light resistance partial occlusion (open bars), moderate resistance (hatched bars), and partial occlusion-only (stippled bars) sessions. Values are means ± SE. *Significant difference compared with preexercise values, $P < 0.05$. †Significant difference compared with the moderate resistance and partial occlusion only sessions for the same time period, $P < 0.05$.](image2)

![Fig. 3. Total testosterone changes during the experimental sessions for light resistance partial occlusion (open bars), moderate resistance (hatched bars), and partial occlusion-only (stippled bars) sessions. Values are means ± SE. There was no significant difference for each treatment over time.](image3)

Table 2. Number of repetitions performed per set of biceps curls and calf presses in the LRO and MR sessions

<table>
<thead>
<tr>
<th></th>
<th>Biceps Curls</th>
<th></th>
<th>Calf Presses</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Set 1</td>
<td>Set 2</td>
<td>Set 3</td>
<td></td>
</tr>
<tr>
<td>LRO</td>
<td>23 (2.5)</td>
<td>9.6 (1.9)</td>
<td>7.8 (2.5)</td>
<td></td>
</tr>
<tr>
<td>MR</td>
<td>10.2 (1.9)</td>
<td>5.4 (1.1)</td>
<td>4.2 (1.4)</td>
<td></td>
</tr>
</tbody>
</table>

Values mean (SD) for 8 subjects. LRO, light resistance occlusion; MR, moderate resistance; OO, no occlusion.
levels. Regardless of the partial vascular occlusion, the lack of active muscle contraction in the OO session likely reduced lactate formation due to the negligible demand for oxygen and substrate metabolism.

In a study by Takarada et al. (33), the researchers reported a GH increase 290 times that of resting levels in response to a light resistance leg-extension protocol and partial occlusion, a concentration \( \sim 34 \mu g/l \). In the present study, GH increased 41 times more than resting levels to \( \sim 8 \mu g/l \), an increase less than that in the Takarada et al. study but still a strong response. There are a few possible explanations for the lower GH response measured in the present study compared with the study by Takarada and colleagues. First and foremost, the biceps brachii and the gastrocnemius and/or soleus muscles recruited in this protocol design are much smaller in cross-sectional area than the quadriceps group employed in the earlier study, and the metabolic stress induced by partial vascular occlusion would be less widespread and potentially attenuate some of the lactate response to muscular work. Peak lactate levels in the present study reached \( \sim 2.2 \) mmol/l, whereas those in the study by Takarada et al. reached 4.4 mmol/l. Furthermore, the stimulus for hormone response provided by afferent feedback from the relatively low mechanical stress (due to small-muscle recruitment) would be less in our study compared with larger active muscle groups. Second, the 5-min rest period between the two exercises performed by the present study’s subjects was not present in the single exercise protocol utilized by Takarada et al.; this length of inactivity may have reduced GH response and/or enhanced GH clearance, resulting in lower values. Despite protocol differences, our second hypothesis was supported; GH response to LRO did rise to a greater extent than MR from baseline levels.

There was a similar trend of GH elevation from preexercise to postexercise for both the MR and OO conditions. Despite a lack of statistical difference, it is interesting to note that partial occlusion alone, without any voluntary muscular activity, tended to cause a rise in GH concentration to a similar extent as that of moderate workload resistance exercise utilizing similar muscle mass, as was hypothesized. We speculate that use of larger muscle groups and/or bigeminal limb activity would have produced significant increases in GH in both the MR and OO trials. Further investigation is required to elucidate the particular mechanism responsible, but we can surmise that partial occlusion induced hypoxic conditions (which promotes lactate formation) and trapped the locally produced lactate from circulating away to the liver and other tissues, which may then actively promote GH secretion (10, 12).

Although it has been shown that lactate may directly stimulate T synthesis in Leydig cells (26), T and FT concentrations after all three exercise conditions were virtually unchanged. The protocol was expected to elicit a less prominent change in T compared with changes in GH, but the negligible alterations suggest that the volume and/or intensity of the protocols with and without partial vascular occlusion were not sufficient to elicit T and FT changes. Kraemer et al. (23) demonstrated increases in T after 8 exercises of either 5 or 10 repetitions per set and low rest periods, a protocol of considerably greater volume of exercise and consistent 1-min rest periods. Since testosterone secretion appears to follow a dose-response mechanism (7), it is possible that this design was not of sufficient intensity to promote T release.

The response of cortisol was important to determine since the T-to-cortisol ratio has been implicated in resistance-exercise-induced anabolism (4). Cortisol responses to exercise have been previously demonstrated to occur primarily after high-intensity exercise as opposed to low to moderate levels (22). This study employed low to moderate workloads, and therefore we hypothesized that cortisol would not change as a result of any trial condition. A further rationale for this hypothesis was from previous findings that showed no cortisol response to hypoxic conditions (25); our use of the vascular cuff promoted a similar effect on the intramuscular environment and was supported since no significant change was observed.

In conclusion, the use of external cuff application proximal to working muscle, combined with light resistance exercise promotes GH response that exceeds or matches the amount elicited from moderate resistance exercise without partial occlusion. Thus this is an important finding since traditional thinking suggests that GH concentration will not rise to any appreciable extent unless moderate to heavy resistance loads are utilized. The study employed small muscle mass and low as well as moderate loads that would be of a lesser intensity and muscle volume than those used in many resistance exercise regimens. Although employing partial vascular occlusion with

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**Fig. 4.** Free testosterone changes during the experimental sessions light resistance partial occlusion (open bars), moderate resistance (hatched bars), and partial occlusion-only (stippled bars) sessions. Values are means \( \pm SE \). There was no significant difference for each treatment over time.

**Fig. 5.** Cortisol changes during the light resistance partial occlusion (open bars), moderate resistance (hatched bars), and partial occlusion-only (stippled bars) sessions. Values are means \( \pm SE \). There was no significant difference for each treatment over time.
greater muscle mass and larger loads than used in the present study would have the practical barriers of pain, subject compliance and multiple occlusion sites, its effects remain to be determined. Nevertheless, it is interesting that GH responses to the LRO trial were similar to those in previous studies produced with high-volume resistive exercise (19, 20). T was not involved in the occlusion and light resistance response. This is significant since it has been shown previously that very light resistance exercise with occlusion can result in muscular hypertrophy (30, 32, 34, 35); thus GH may be a contributor to the known adaptation, but T, at least acutely with light loading, does not appear to play a role. Tremblay et al. (38) demonstrated a T response after resistance exercise for three groups of varying training status: sedentary, resistance, and endurance trained. The sedentary group elicited the greatest response, followed closely by the resistance-trained group, and the lowest response occurred in the endurance-trained group. Based on these findings, we do not believe greater T or lactate responses would have resulted from a group of endurance-trained or sedentary subjects. More research is needed to determine the degree to which exercise load and muscle mass utilized affect these responses.

The present protocol is different from the previous protocols investigating partial occlusion and endocrine responses (33, 36) in that an OO session was included for all subjects, smaller muscle groups were utilized, and LRO was shown to have much greater impact on GH release compared with MR. Moreover, this is the first study to document no change in T, FT, or cortisol responses to LRO. With the possible benefits of GH action on muscle and bone tissue, these findings could provide further evidence to support methods of inducing metabolic stress for individuals not capable of tolerating overload to promote tissue adaptations. This eventually may be of benefit to clinical applications of rehabilitative resistance exercise for orthopedic conditions, because the obstacle of compromised muscular strength in many patients would be ameliorated with use of LRO.

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GRANTS

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