Acute Hormonal Responses to a Single Bout of Heavy Resistance Exercise in Trained Power Lifters and Untrained Men

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Abstract/Résumé

The purpose of this study was to investigate the acute responses of both stress and fluid regulatory hormones to a single bout of resistance exercise in both trained and untrained men. Seven competitive power lifters (PL) and 12 untrained subjects (UT) performed one set of the leg press exercise to exhaustion at 80% of their respective one-repetition maximum. Blood samples were obtained twice prior to exercise (at P1 and P2), immediately postexercise (IP), and at 5 minutes postexercise (5PE). Compared to P1 and P2, plasma epinephrine, norepinephrine, dopamine, atrial peptide, osmolality, and blood lactic acid increased significantly ($p \leq 0.05$) at IP. Plasma epinephrine, norepinephrine, atrial peptide, and blood lactic acid concentrations remained elevated at 5PE compared to P1 and P2.

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Plasma renin activity and angiotensin II were significantly elevated at 5PE compared to P1, P2, and IP, and this increase was significantly greater in PL compared to UT at 5PE. These data indicate that an acute bout of resistance exercise dramatically affects secretion of stress and fluid regulatory hormones.

Le but de cette étude est d'analyser les ajustements immédiats des hormones du stress et des hormones de régulation du niveau des fluides à une seule séance d'exercices de force chez des individus entraînés et d'autres non entraînés. Sept dynamophiles encore en compétition (PL) et 12 sujets non entraînés (UT) exécutent jusqu'à épuisement une série d'exercices de développé des jambes à 80% du maximum sans répétition (1-RM). Des échantillons de sang sont prélevés en deux occasions avant l'effort (P1 et P2), immédiatement après l'effort (IP), et 5 min plus tard (5PE). Comparativement à P1 et P2, les concentrations plasmatiques d'épinéphrine, de norépinéphrine, de dopamine, de peptide auriculaire, d'acide lactique et l'osmolalité sont significativement plus importantes ($p < 0,05$) à IP. Comparativement à P1 et P2, les concentrations plasmatiques d'épinéphrine, de norépinéphrine, de peptide auriculaire et d'acide lactique demeurent importantes à 5PE. Comparativement à P1, P2 et IP, l'activité rénine plasmatique et l'angiotensine II sont significativement plus élevées à 5PE, l'augmentation étant plus importante chez les PL que chez les UT. Ces observations indiquent qu'une séance d'exercices de force modifie grandement la sécrétion des hormones du stress et de celles régulatrices du niveau des fluides.

Introduction

An acute bout of resistance exercise has been shown to increase plasma concentrations of several hormones (Kraemer, 1988). Previous studies have shown elevated plasma concentrations of epinephrine (Guezennec et al., 1986; Kraemer et al., 1987), norepinephrine (Guezennec et al., 1986; Kraemer et al., 1987), dopamine (Kraemer et al., 1987), and cortisol (Kraemer et al., 1989; 1992b; 1993) in response to and for an extended period of time following resistance exercise. These acute increases are dependent on the type of resistance exercise protocol (i.e., number of sets and repetitions per set) employed (Kraemer, 1992b) and may play a significant role in the acute expression of strength, blood flow regulation, cardiac contractility, augmentation of the secretion rates of other hormones, and substrate mobilization (Kraemer, 1992a).

We know of no data examining the acute responses of hormones involved in blood pressure and fluid balance regulation to resistance exercise. The acute responses of arginine vasopressin, atrial peptide, plasma renin activity, aldosterone, and angiotensin II to submaximal and maximal endurance exercise have been extensively investigated (Convertino et al., 1981; Mandroukas et al., 1995; Mannix et al., 1990). Most of these studies have reported significant increases during exercise and several minutes into the recovery period, with the magnitude dependent on exercise intensity (Convertino et al., 1981; Mandroukas et al., 1995; Mannix et al., 1990), duration (Mandroukas et al., 1995; Perrault et al., 1989), and hydration status (Grant et al., 1996). Resistance exercise has been shown to significantly reduce plasma volume (Gordon et al., 1985) comparable to changes elicited by running or cycling at 80–95% of $\dot{V}O_2$ max (Collins et al., 1986), and may increase blood pressure to 320/250 mmHg during maximal performance of the leg press exercise (MacDougall et al., 1985). Rapid changes in body fluid volume and blood

pressure may initiate a multitude of hormonal responses involved in homeostatic control. Therefore, the purpose of this study was to document the acute response of stress-related and fluid balance regulatory hormones to a single set of exhaustive resistance exercise. A secondary purpose was to examine the influence of training status on these hormonal alterations.

Methods

SUBJECTS

Seven male power lifters (PL) (mean ± *SD* age = 24.7 ± 3.8 yrs, height = 177.4 ± 12.3 cm, body mass = 95.6 ± 17.5 kg, body fat = 11.7 ± 4.7%) and 12 untrained (UT) men (age = 26.6 ± 5.9 yrs, height = 179.4 ± 6.5 cm, body mass = 79.5 ± 9.2 kg, body fat = 10.6 ± 3.6%) volunteered for this study. All PL had competed in drug-free powerlifting competitions and reported no use of anabolic drugs in their career. Untrained subjects were physically active, healthy men who had not participated in any formal exercise training program over the past 2 years but did do manual labor in their profession. Subjects were fully informed of the testing procedures prior to the study and signed an institutionally approved consent form. All participated in several familiarization sessions in the laboratory prior to testing. Standardized strength testing procedures were used to obtain subjects' one-repetition maximum (1-RM) during a preliminary session (Kraemer and Fry, 1995). In addition, percent body fat was determined via a 7-site skinfold test using the equations of Pollock et al. (1980) and Siri (1961).

EXPERIMENTAL DESIGN

The bilateral leg press was the exercise used in this study (Universal Gladiator, Irvine, CA) because it is stresses large muscle groups of the lower body and can alter the physiological stress on the body similar to other modalities (e.g., cycling, treadmill). Briefly, to begin the exercise, the subject sat upright with feet parallel and flat on the pedal surface with the knees flexed at a 90° angle. The concentric phase consisted of hip and knee extension until a knee angle of 180° was reached, and the eccentric phase consisted of the return to the beginning exercise position. After a warm-up, subjects were instructed to perform as many repetitions as possible with a load corresponding to 80% of their respective 1-RM. Repetitions were performed continuously with no rest in between, using a full range of motion monitored by goniometers interfaced to a computer.

Blood samples were drawn via teflon cannula inserted into an antecubital vein. Two preexercise samples were obtained, immediately after cannula insertion (P1) and following 30 minutes of equilibration (P2). In addition, samples were obtained immediately after exercise (IP) and 5 minutes postexercise (5PE). The cannula was kept patent via an isotonic saline drip (30 ml · hr^{-1}). All blood samples were obtained with the subjects seated. Subjects ingested 750 ml of water 1/2 hour prior to the first blood sample. They refrained from food, beverages, and physical activity for 8 hours prior to the testing protocol.

The methods for assessing peak arterial blood pressure have previously been reported (Fleck and Dean, 1987). Briefly, an Allen's test was performed on the nondominant arm to ensure subject safety. The anterior distal forearm surface of

the nondominant arm was bathed with an iodine solution. Following administration of a subcutaneous local anesthetic (1% Lidocaine), the radial artery was cannulated utilizing a 20-gauge angiocath. A standard intra-arterial blood pressure monitoring kit (Sorenson, model MK3-03NVF, Sorenson Research, Salt Lake City) and pressure transducer (Stratham P23 ID) were used to connect the angiocath to a polygraph (Grass Instrument, Quincy, MA). The entire blood pressure monitoring system was statistically calibrated against a mercury manometer and verified to be linear between 0 and 300 mmHg. During the exercise test, the subjects were not allowed to grip the hand-support rails of the Universal leg press machine with the nondominant arm.

ANALYTICAL METHODS

Venous blood samples were obtained using a plastic syringe connected to a three-way stopcock adapter on the catheter and were immediately transferred into Vacutainers™. The blood was immediately centrifuged at 2,000 × g for 20 minutes and plasma samples were stored at –80 °C until analysis. Whole-blood lactic acid was analyzed in duplicate using an enzymatic-amperometric method (24 YSI Lactate Analyzer, Yellow Springs, OH). Blood glucose concentrations were determined in duplicate via a 23-glucose analyzer (YSI, Yellow Springs, OH). Blood was analyzed for hemoglobin using the cyanmethemoglobin spectrophometric method at 540 nm (Sigma Chemical, St. Louis). Hematocrit was determined in triplicate using a standard microcapillary technique. Plasma osmolality was measured in triplicate on unfrozen plasma on a Westcor vapor-pressure osmometer (Logan, UT). Percent change in plasma volume was calculated from the hemoglobin and hematocrit values by the method of Dill and Costill (1974). Plasma volume shifts were less than 10%. The hormonal data were corrected for plasma volume shifts prior to statistical analysis.

Plasma epinephrine, norepinephrine, and dopamine were determined using a Waters™ high-performance liquid chromatography system (Millipore, Milford, MA) and electrochemical detection (Kraemer et al., 1991) from a preliminary aluminum oxide extraction. A reverse phase Waters plasma catecholamine column with a Water mobile phase was used. All analytical and extracation procedures used were those recommended by the Waters Chromatography Division (Milford, MA). Plasma samples were run in duplicate with an internal standard, 3,4-dihydroxybenzylamine, and corrected for the percent recovery. The mean recovery was 89 ± 7%.

Plasma cortisol and aldosterone concentrations were assayed in duplicate using solid-phase ^{125}I-radioimmunoassay techniques (Diagnostic Products, Los Angeles) (De Souza et al., 1989). All samples from each subject were analyzed in the same assay to avoid interassay coefficients of variation and were thawed only once for each assay procedure. All radioimmunoassay intra-assay and interassay coefficients of variance were <5 and 10%, respectively.

Blood for arginine vasopressin and renin was obtained with a precooled EDTA plastic Vacutainer (Sarstedt, Princeton, NJ) inserted into the three-way stopcock attached to the indwelling cannula. Blood was then centrifuged at 2,000 × g for 20 minutes at –4 °C. Arginine vasopressin was extracted from plasma with Sep-Pak C_{18} cartridges (Water Associates, Milford, MA) and measured via radioimmuno-

assay methods originally described by Goetz et al. (1981) and modified by Wang et al. (1983). Plasma renin activity was determined by the method of Haber et al. (1969) using a solid phase ^{125}I-radioimmunoassay (Clinical Assays, Cambridge, MA).

Angiotensin II concentrations were determined via radioimmunoassay with preliminary C_{18} extraction and were separated using pretreated C_{18} (5 ml of methanol and 1% trifluoroacetic acid) separating columns (Waters Associates, Milford, MA). The eluant was evaporated under vacuum. The angiotensin II antibody was brought to 1 ml with assay buffer and frozen in 100 microliter aliquots. One ml of charcoal in assay buffer was used to separate bound from unbound ^{125}I-labeled angiotensin II.

Atrial peptide concentrations were determined via radioimmunoassay with preliminary C_{18} extraction using a commercially available ^{125}I-ligand and antisera (Peninsula Labs, Belmont, CA) similar to previous studies (Kraemer et al., 1988). Atrial peptide was separated using pretreated C_{18} (200 mg, equilibrated with 100% acetonitrile and 1% trifluoroacetic acid) separating columns (Peninsula Labs) with a series of washes (1% trifluoroacetic acid and 60% acetonitrile in 1% trifluoroacetic acid). The eluant was evaporated under vacuum. There was no interassay variance as all samples were assayed at the same time. The mean recovery of radioactively labeled atrial peptide following extraction was 88 ± 4%.

STATISTICAL ANALYSES

Performance data between groups was analyzed using a *t*-test, and a 2 × 4 (group × time) analysis of variance (ANOVA) was used to analyze plasma osmolality, glucose, lactic acid, and all hormonal data. When appropriate, Tukey post hoc comparisons were used to determine pairwise differences between trained and untrained subjects. Pearson product-moment correlation coefficients were calculated between selected acute hormonal data and peak arterial blood pressure measures to determine pairwise bivariate relationships. Statistical power calculations ranged from 0.78 to 0.80. Significance in this study was set at $p \leq 0.05$. Data were expressed as the mean ± the standard deviation.

Results

Concerning performance data, the PL 1-RM leg press was significantly greater than UT (299.5 ± 28.7 vs. 156.0 ± 16.6 kg). However, the number of repetitions performed to exhaustion at the relative intensity of 80% 1-RM did not differ between groups (PL = 21.0 ± 1.8; UT = 20.3 ±5.7).

ACUTE CHANGES IN BLOOD GLUCOSE AND LACTIC ACID

Blood glucose showed a significant rise at IP compared to P1 in UT. There were no significant changes in PL or significant differences between groups at any point. Blood glucose levels returned to baseline values at 5PE for both groups. The data (mean ± *SD*) for glucose (mmol/L) were as follows: ***For PL***: P1 = 5.4 ± 0.7; P2 = 5.7 ± 0.4; IP = 5.6 ± 0.6; 5PE = 5.2 ± 0.7. ***For UT***: P1 = 5.1 ± 0.6; P2 = 5.5 ± 0.6; IP = 5.8 ± 0.6; 5PE = 5.4 ± 0.5. Blood lactic acid rose significantly in both groups

at IP compared to P1 and P2. For PL there was a significantly greater rise at 5PE compared to P1 and P2. For UT there was a significantly greater rise at 5PE compared to P1, P2, and IP. Data for lactic acid (mmol/L) were as follows: ***For PL***: P1 = 1.9 ± 0.7; P2 = 1.6 ± 0.4; IP = 4.9 ± 1.8; 5PE = 7.1 ± 1.7. ***For UT***: P1 = 1.8 ± 0.4; P2 = 1.8 ± 0.8; IP = 5.2 ± 2.2; 5PE = 7.3 ± 2.9. There were no differences between groups at any time.

ACUTE PLASMA CATECHOLAMINE AND CORTISOL RESPONSE

Figure 1 shows the acute responses of epinephrine, norepinephrine, and dopamine. Both groups had significant increases in epinephrine, norepinephrine, and dopamine at various time points. Epinephrine was significantly elevated at IP for both groups, but was only elevated at P2 for PL and 5PE for UT compared to P1. Plasma norepinephrine was significantly greater at P2, IP, and 5PE for both groups compared to P1. However, PL displayed greater concentrations at IP. Plasma dopamine only showed insignificant increases in UT at P2 and IP, but was significantly increased at IP in PL. Cortisol showed no significant changes at any time point, and no significant differences between groups at any time. Data for cortisol were as follows: ***For PL***: P1 = 409 ± 103; P2 = 434 ± 192; IP = 345 ± 106; 5PE = 315 ± 114. ***For UT***: P1 = 398 ± 151; P2 = 452 ± 189; IP = 428 ± 178; 5PE = 377 ± 149.

PLASMA OSMOLALITY, RENIN ACTIVITY, AND OTHER HORMONES

Figure 2 shows the acute changes in plasma osmolality, renin activity, angiotensin II, and atrial peptide. Plasma osmolality (Panel A) was significantly increased at IP compared to P1 and P2 in both groups. In UT, osmolality was greater at IP compared to 5PE. There were no differences between groups. Plasma renin activity and angiotensin II (Panels B and C) concentrations did not change in UT. However, both significantly increased in PL at 5PE compared to IP (angiotensin II and renin activity) and P2 (plasma renin activity). There was a significant difference at 5PE between groups, as PL showed a greater response than UT. Plasma atrial peptide (Panel D) did not change between P1 and P2 for either group. There were significant increases at IP and 5PE for both groups compared to P1 and P2. Plasma atrial peptide concentrations at P1 in UT were significantly greater than PL. Plasma aldosterone concentrations did not change significantly at any time point in either group.

Data (mean ± *SD*) for aldosterone (pmol/L) were as follows: ***For PL***: P1 = 286± 154; P2 = 229 ± 107; IP = 3196 ± 110; 5PE = 301 ± 104. ***For UT***: P1 = 260 ± 145; P2 = 257 ± 175; IP = 262 ± 110; 5PE = 290 ± 109. Plasma arginine vasopressin was not statistically different at P1 and P2. Data for arginine vasopressin (pg/ml) were as follows: ***For PL***: P1 = 0.8 ± 0.5; P2 = 0.9 ± 0.5; IP = 4.9 ± 3.2; 5PE = 3.07 ± 2.7. ***For UT***: P1 = 2.6 ± 2.4; P2 = 2.1 ± 1.2; IP = 3.1 ± 3.5; 5PE = 4.6 ± 4.5. There were increases at IP in PL and a moderate increase at 5PE in both groups, but these increases were also not significant.

BLOOD PRESSURE

Peak arterial systolic (SBP) and diastolic blood pressures (DBP) for both groups obtained at IP were 285.8 ± 34.4 and 216.6 ± 46.4 mmHg, respectively (PL: SBP =

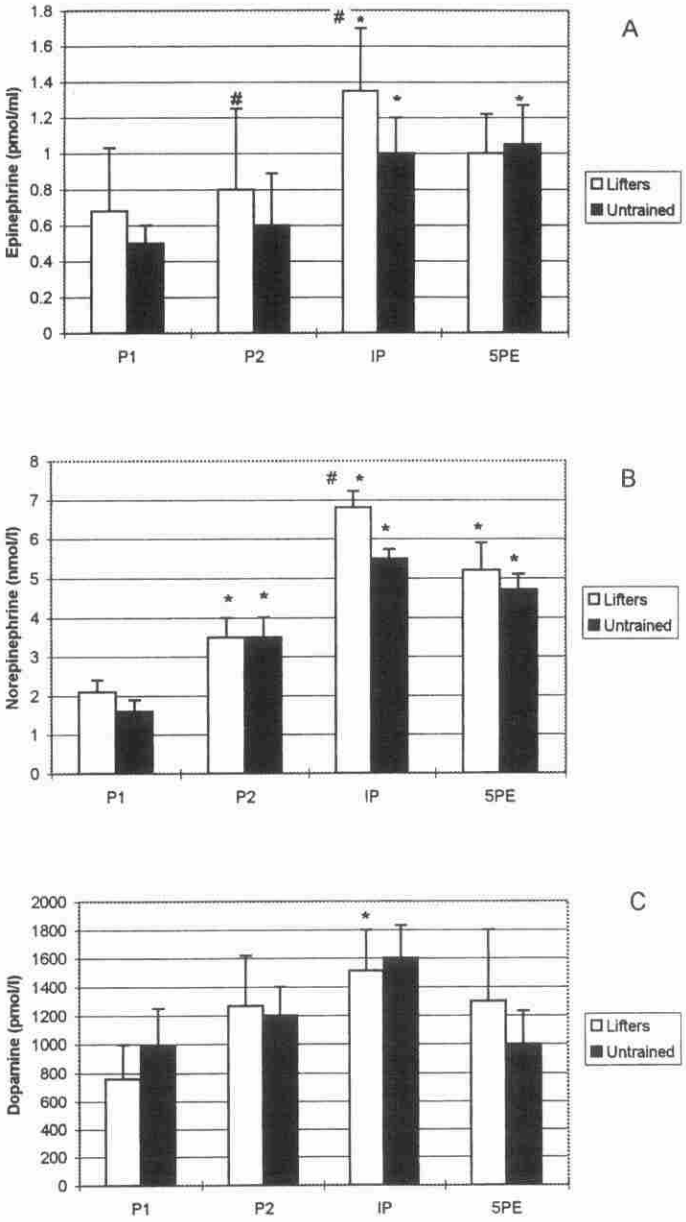

Figure 1. Plasma epinephrine, norepinephrine, and dopamine. (**A**) Changes in epinephrine (pmol/L). *Signif. increase from P1 and P2 for both groups; #Signif. difference between PL and UT. (**B**) Changes in norepinephrine (nmol/L). *Signif. increase from P1 in both groups; #Signif. difference between PL and UT. (**C**) Changes in dopamine (pmol/L). *Signif. increase in PL vs. P1. No differences observed between groups.

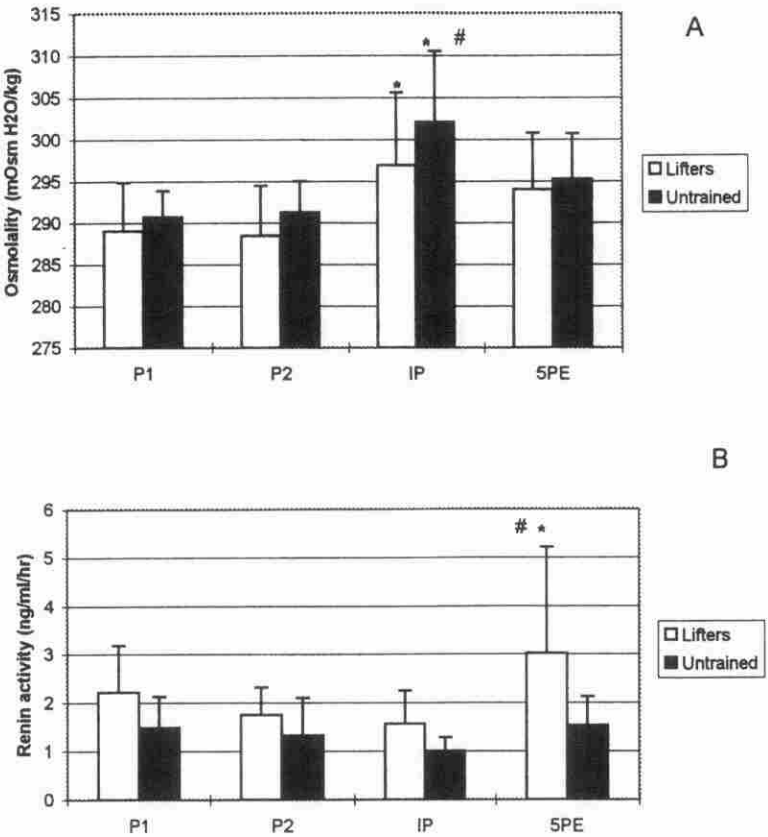

Figure 2a. Changes in osmolality, renin activity, angiotensin II, and atrial peptide. (**A**) Changes in osmolality (mOsm H_2O/kg). *Signif. increase from P1 and P2 in both groups. #Signif. difference between UT and PL at IP compared to 5PE. (**B**) Changes in plasma renin activity (ng/ml/hr). *Signif. increase from P2 and IP for PL. #Signif. difference between PL and UT. No differences in UT.

304.0 ± 53.9 mmHg; DBP = 230.5 ± 80.5 mmHg; UT: SBP = 278.5 ± 23.1 mmHg; DBP = 211.1 ± 28.9 mmHg). No significant differences were observed between groups. Combined group analysis of relationships showed a significant bivariate correlation between plasma norepinephrine and SBP ($r = 0.61$) at IP.

Discussion

The primary finding in this study was that a single acute bout of resistance exercise elicits a significant response of several stress related and fluid regulatory hormones. Plasma concentrations of these hormones have been shown to be sensitive to changes in blood pressure during resistance training (Fleck and Dean, 1987). A

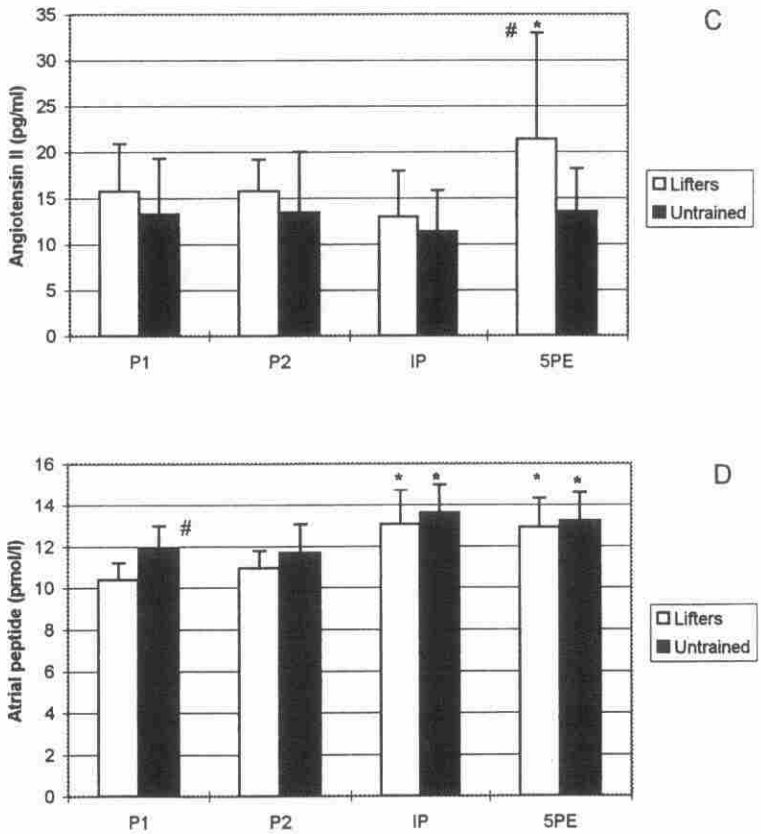

Figure 2b. Changes in osmolality, renin activity, angiotensin II, and atrial peptide. (**C**) Changes in angiotensin II (pg/ml). *Signif. increase from IP in PL. #Signif. difference between PL and UT. No differences in UT. (**D**) Changes in atrial peptide (pmol/L). *Significant increase at IP and 5PE vs. P1 and P2 in both groups. #Signif. difference between groups at P1.

review of previous studies revealed that sets of the leg press performed to volitional fatigue at intensities of 70 and 80% 1-RM increased blood pressure to a significantly greater extent than 95 and 100% 1-RM (Fleck and Dean, 1987). Both the intensity and duration of the set, coupled with the large muscle mass used, are important variables for eliciting a significant increase in arterial blood pressure. Therefore the load corresponding to 80% 1-RM for the leg press was chosen to provide a significant stress in order to show changes in the fluid regulatory and stress hormonal systems.

To our knowledge, there have been no studies on the acute responses of atrial peptide, arginine vasopressin, aldosterone, renin activity, and angiotensin II to resistance exercise. Previous studies have focused on aerobic modes of exercise

in which acute plasma increases in atrial peptide (Convertino et al., 1981; Mannix et al., 1990; Tanaka et al., 1986), arginine vasopressin (Convertino et al., 1981; Perrault et al., 1989; Tanaka et al., 1986), renin activity (Perrault et al., 1989; Staessen et al., 1987; Tanaka et al., 1986), aldosterone (Mannix et al., 1990; Perrault et al., 1989; Staessen et al., 1987), and angiotensin II (Staessen et al., 1987) were reported to be dependent on exercise intensity (Mannix et al., 1990; Perrault et al., 1989; Staessen et al., 1987), duration (Mandroukas et al., 1995; Perrault et al., 1989), and hydration status (Grant et al., 1996). Low to moderate exercise intensities have produced little or no change in atrial peptide, aldosterone, renin activity, and arginine vasopressin, but intensities greater than 40% of $\dot{V}O_2$ max have produced the most pronounced increases (Convertino et al., 1981; Perrault et al., 1989). Considering that most resistance exercise protocols are high in intensity, it is reasonable to hypothesize that resistance exercise may produce similar responses. The present study supported this contention as plasma atrial peptide, renin activity, and angiotensin II did show similar responses at both IP and 5PE.

The present study showed significant increases in atrial peptide at IP and 5PE. The magnitude of release was not as great as previously shown during high-intensity aerobic training with a high volume of total work (Perrault et al., 1989). Perrault et al. (1989) and Tanaka et al. (1986) have reported a proportional increase in atrial peptide concentrations with increases in $\dot{V}O_2$ ($r = 0.80$). The intensity in the present study was high, but it may be that the relatively short duration of exercise and low total work may not have been sufficient to elicit similar responses observed with aerobic training sessions lasting at least 15 minutes (Perrault et al., 1989). No difference was found between PL and UT. This has also been observed when aerobically trained individuals were compared with untrained individuals (Fellmann, 1992).

There were no significant decreases in plasma volume for either group in the present study. Decreases in plasma volume have been reported during aerobic (Freund et al., 1991) and resistance exercise (Collins et al., 1986; Gordon et al., 1985). The protocol used in the present study was most likely too brief and of insufficient total work to elicit a significant decrease in plasma volume (Kraemer et al., 1988; 1989). Thus, the magnitude of plasma volume shift was not great enough to elicit significant hormonal responses to the initial physical stress.

Interestingly, plasma osmolality was significantly greater at IP for the power lifters, and at IP and 5PE for the untrained subjects, despite an insignificant change in plasma volume. Typically, plasma osmolality increases curvilinearly as a consequence of graded aerobic exercise whereas plasma volume has been shown to decrease linearly (Convertino et al., 1981). Similar findings have been reported previously. Convertino et al. (1981) found a disproportionate increase in plasma osmolality despite only a 3.5% decrease in plasma volume. Thus, our data show that changes in plasma osmolality are initiated early in response to the first set of a resistance exercise protocol and are disproportionate in comparison to changes in plasma volume.

The lack of change in plasma volume may have contributed to the lack of changes observed with arginine vasopressin and aldosterone. In addition, previous investigations have shown that atrial peptide secretion (Goetz et al., 1986; Perrault et al., 1989) and angiotensin II (Grant et al., 1996; Wade, 1984) may be involved in the regulation of arginine vasopressin and aldosterone concentrations. Atrial pep-

tide concentrations increased in both groups and angiotensin II increased significantly only in PL at 5PE in the present study. Thus, the possibility that these hormones were involved with arginine vasopressin regulation cannot be excluded. In addition, atrial peptide has been reported to inhibit aldosterone concentrations (Weidmann et al., 1989). It is possible that the elevated atrial peptide concentrations observed in the present study suppressed plasma aldosterone concentrations. No differences were observed between groups in the present study for either arginine vasopressin or aldosterone. These results support previous findings in which no differences were observed between endurance-trained and untrained subjects (Freund et al., 1987).

Plasma renin activity and angiotensin II concentrations significantly increased only for the power lifters at 5PE. There was a significant difference at 5PE between groups, as PL showed a greater response than UT. Previously, no studies have examined the response of the renin-angiotensin system to an acute bout of resistance exercise. High correlations between renin activity and angiotensin II have been reported during aerobic exercise (Staessen et al., 1987). Atrial peptide has been shown to stimulate the renin-angiotensin system (Perrault et al., 1989). Considering that both groups experienced similar increases in atrial peptide, the greater response observed in the power lifters may have been due in part to the greater norepinephrine response at IP. Greater sympathetic stimulation of β-adrenergic receptors of the juxtaglomerular cells has been shown to be a potent stimulator of plasma renin activity (Mannix et al., 1990; Perrault et al., 1989). It is possible that the greater norepinephrine response observed at IP in the power lifters may have stimulated the renin-angiotensin system to a greater extent.

The plasma catecholamine response to heavy resistance exercise has been similar to heavy anaerobic sprint and cycle exercise and is dependent on the force of muscle contraction, amount of muscle tissue stimulated, and rest periods between sets and repetitions (Kraemer, 1988). The results of the present study support previous findings of elevated catecholamine concentrations during an acute bout of resistance exercise (Kraemer et al., 1987; 1993). Kraemer et al. (1987) reported significant elevations of epinephrine and norepinephrine through 5 minutes postexercise using 3 sets at 10-RM with 10-sec rest intervals. Guezennec et al. (1986) also reported significant elevations in epinephrine and norepinephrine following both submaximal (i.e., 6 sets of 8 reps at 70% 1-RM) and maximal weight training sessions (one set to failure at same workload). Although only one set was performed in the present study, the heavy resistance and high anaerobic nature of the set appeared to present a sufficient stimulus for catecholamine secretion, which supports the rapid response of this neural-hormonal axis to physical stress (Kraemer et al., 1991).

Interestingly, plasma norepinephrine concentrations showed a significant increase at P2 compared to P1. This "anticipatory rise" was observed in a previous study by Kraemer et al. (1991), who reported an increase in epinephrine prior to the initiation of maximal cycle exercise ranging from 115 to 318% of $\dot{V}O_2$ max. The greatest increase occurred prior to initiation of the highest intensity tests. This anticipatory response may be part of the body's psychophysiological adjustment for performance of a maximal-effort exercise stress. This response may be elicited hours prior to physical stress and may be accentuated prior to acute exercise (Triplett-McBride et al., 1998).

Plasma cortisol concentrations did not change significantly from rest in the present study. The acute response of plasma cortisol during resistance exercise has been shown to be variable with increases (Guezennec et al., 1986; Kraemer et al., 1992; 1993) or no changes reported (Kraemer et al., 1993). Furthermore, the time course of plasma cortisol changes may be longer than 5 minutes into recovery (Kraemer et al., 1993). It appears that the type of resistance training protocol used may be the most important variable influencing peripheral concentrations of cortisol, as well as the time frame used to evaluate these response patterns (Kraemer et al., 1993).

In summary, this study demonstrated that a single acute bout of resistance exercise significantly increased the plasma concentrations of atrial peptide, renin activity, angiotensin II, epinephrine, norepinephrine, and dopamine. Changes in plasma catecholamines reflect the stress associated with resistance training. The small magnitude of plasma volume change may have contributed to the lack of significant change observed with arginine vasopressin. The acute increase in atrial peptide may have modulated the response of renin activity, angiotensin II, and aldosterone. The increase in plasma renin activity and angiotensin II at 5PE in the power lifters may have been stimulated by increased catecholamine concentrations. The hormonal response patterns between trained and untrained lifters differed with respect to epinephrine, norepinephrine, renin activity, and angiotensin II concentrations.

It appears that resistance training elicits a multitude of hormonal effects that attempt to maximize performance while maintaining homeostatic control of fluid volume and blood pressure. This study emphasizes the rapidity with which many hormones respond to an initial physical stress and yet form a basis for subsequent alterations and interactions of further exercise. Such initial response patterns must be kept in mind when studying various resistance exercise protocols in order to understand the physiological progression of endocrine regulation.

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