Acute Hormonal and Neuromuscular Responses and Recovery to Forced vs. Maximum Repetitions Multiple Resistance Exercises

Abstract
Acute hormonal and neuromuscular responses and recovery three days after the exercises were examined during the maximum repetitions (MR) and forced repetitions (FR) resistance exercise protocols in 16 male athletes. MR included 4 sets of leg presses, 2 sets of squats and 2 sets of knee extensions (with 12 RM) with a 2-min recovery between the sets and 4 min between the exercises. In FR the initial load was chosen to be higher than in MR so that the subject could lift 12 repetitions per set by himself. After each set to failure the subject was assisted to perform the remaining repetitions to complete the 12 repetitions per set. Thus the exercise intensity was greater in FR than in MR. Both loading protocols led to the great acute increases (p < 0.05 – 0.001) in serum testosterone, free testosterone, cortisol and GH concentrations. However, the responses in cortisol (p < 0.05) and GH (p < 0.01) were larger in FR than in MR. The decrease of 56.5% (p < 0.001) in maximal isometric force in FR was greater (p < 0.001) than that of 38.3% in MR (p < 0.001) and force remained lower (p < 0.01) during the recovery in FR compared to MR. The larger decrease in isometric strength in FR than in MR was also associated with the decreased maximal voluntary EMG of the loaded muscles. The data indicate that the forced repetition exercise system induced greater acute hormonal and neuromuscular responses than a traditional maximum repetition exercise system and therefore it may be used to manipulate acute resistance exercise variables in athletes.

Key words
Anabolic hormones · isometric force · EMG · neuromuscular fatigue · muscle damage · bodybuilding

Introduction
Heavy resistance exercise is a potent stimulus for acute increases in the concentrations of circulating hormones in young men. These acute increases are highly dependent on the type of resistance exercise protocol, i.e. number of sets and repetitions per set, length of rest period between sets and muscle mass involved employed [e.g. 16, 23].

Especially anabolic hormones (e.g. growth hormone, testosterone) influence growth and development [10]. The stress of heavy-resistance exercise when performed repeatedly (i.e. training) is a stimulus for both strength development and muscle fiber hypertrophy. This may be related, at least in part, to exercise-induced acute increase in endogenous anabolic hormones [23].

The hormonal response to resistance exercise is also associated with changes in the neuromuscular performance. Intense muscular work used typically during a heavy-resistance training leads to a momentary decrease in strength accompanied by decreases in voluntary maximal neural activation of the loaded muscles [14,17,21]. The effect of the fatiguing load on the neuro-

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Accepted after revision: November 20, 2002

Bibliography
muscular system is related not only to the intensity of exercise but also to the specific type of the fatiguing load, and the recovery time between high-intensity intermittent contractions [e.g. 17, 25]. It is speculated that there might be “an optimal” level of acute neuromuscular fatigue after each strength training session to stimulate adaptation processes. According to the principle of progressive strength training, the next training session should usually take place under the conditions where complete recovery has taken place. Therefore, the magnitude of temporary neuromuscular fatigue and the rate of recovery after intensive heavy-resistance exercise may be an indication of its effectiveness for long-term adaptations of the neuromuscular system. Recovery also involves the coordinated functioning of several physiological processes (i.e. regeneration of disrupted muscle fibers) that are heavily influenced by the availability and actions of specific hormones [29, 31, 34].

Traditionally in strength training various exercises have been performed using a so-called maximum repetition system (i.e. each set is performed to a momentary concentric failure). In order to overload the muscle progressively, the training intensity and/or volume and/or frequency should be increased periodically. Because of the risk for overtraining it is appropriate for long-term strength training purposes to prioritise the increase in the training intensity. In practical training the term “intensity” has been used to define the magnitude of the load employed or the rate of work performed [3]. In strength training the intensity can also be modified by special training systems, such as so-called “forced repetitions” as defined by Fleck and Kraemer [9]. Forced repetitions are a special resistance training system, which strength athletes, especially bodybuilders, use to increase training intensity. Forced repetitions means that, after the trainee has achieved a momentary concentric failure (i.e. a set until exhaustion has been performed), a training partner will assist by lifting or pushing the load just enough to allow the trainee to complete three to four additional repetitions. This system “forces” the trainee to continue to produce force, although he or she is already extremely fatigued. It is suggested that it is necessary to achieve the failure, i.e. the momentary muscular fatigue during the resistance exercise sets to gain maximally muscle mass and strength. Although it might be reasonable to suggest that the forced repetition training system may be an effective way to stimulate muscles, no previous studies have been conducted on acute, short- or long-term hormonal and/or neuromuscular responses of the forced repetitions training system.

This study examined the influence of the exercise intensity to acute hormonal and neuromuscular responses and short-term recovery. For that purpose the responses of the forced repetitions training system were compared to those produced by the traditional maximum repetitions training protocol. The experimental training session was created in both loading conditions so that it included multiple exercises, leg press, squat and knee extension, as usually used in training for the leg extensor muscles in athletes like bodybuilders.

**Material and Methods**

**Subjects**

Sixteen healthy physically active men volunteered to participate in this investigation (mean ± SD; age = 26.8 ± 3.5 y, height = 180 ± 6 cm, weight = 81 ± 9 kg, body fat = 13.8 ± 3.5%). Each subject had recreational experience with resistance training but none were competitive strength athletes. No medication was taken by the subjects, which would have been expected to affect physical performance. Each subject was informed of the potential risks and discomforts associated with the investigation and all the subjects gave their written informed consent to participate. The Ethics Committee of the University of Jyväskylä approved the study.

**Experimental design and loading protocols**

The subjects were familiarized with the experimental testing procedures on the control day about 1 week before the measurements. Anthropometrical measurements and resistance load verifications for each experimental exercise were also determined at this time. The percentage of body fat was estimated from measurements of skinfold thickness [6].

During the control day three blood samples were obtained from each subject. One blood sample was drawn in the morning after twelve hours of fasting and approximately eight hours of sleep for the determination of basal serum hormone concentrations. Two blood samples were also drawn within 1/2 h without exercise at the same time of day that each subject would later undertake his heavy resistance loading protocols to determination of normal diurnal variation of serum hormone concentrations.

The experimental design comprised two loading sessions separated by two weeks performed at the same time of day (Fig. 1). Recovery of the loading sessions was examined for three days after both loadings. The first one of the loading sessions was a so-called maximum repetition (MR) protocol. MR included 4 sets of leg press (David 210, from a knee angle of 59.5 ± 2.0° to 180° = knee straight), 2 sets of squats (Smith machine, from a knee angle of 70° to 180°) and 2 sets of knee extensions (David 200, from a knee angle of 59.4 ± 1.6° to 180°) with a two-minute recovery between the sets and four minutes between the exercises. All the sets were performed with the maximum load possible for 12 repetitions (12 RM). The second loading session was a so-called forced repetition (FR) protocol. In FR the loading protocol was the same as in MR, but the initial load was set approximately 15% higher than in MR so that the subject could not perform twelve repetitions without assistance and would require assistance during the last three to five repetitions. The foot positions and exercise machine settings were identical in both loadings. In FR the exact force of the assistance was measured. In the squat the assistant and the subject stand on the separate force plates. The force plates were used to measure the accurate force of the assistance given by the assistant while “lifting” the barbell with his hands during the concentric phase of the squat. In the leg press and knee extension exercises the handle force dynamometer was attached to the foot support and was used to measure the accurate force given by the assistant during his “pulling” action while the subject was extending the knees. The assistant was the same person in all measurements. The external
force produced by the assistant during the concentric phases of the exercises was analysed and then subtracted from the total volume of the work (loads × sets × reps) to determine actual total work performed by the subject in the FR loading. The deepness of the squats was controlled by a sound signal. The duration of the concentric phases of dynamic muscle actions was measured by an electronic goniometer placed on the knee joint.

**Neuromuscular measurements**

An electromechanical dynamometer was used to measure maximal voluntary isometric force of the bilateral leg extension action at a knee angle of 107° before, after two and four sets of leg press, after squats and after knee extensions as well as 24 h, 48 h and 72 h after the loadings. Electromyographic activity (EMG) was recorded from the agonist muscles vastus lateralis (VL) and vastus medialis (VM) of the right leg during the maximal isometric action. Bipolar surface electrodes (Beckman miniature-sized skin electrodes 650-437, Illinois, USA) with 20 mm interelectrode distance were employed. The electrodes were placed longitudinally over the muscle belly on the motor point area determined by an electrical stimulator (Neuroton 626). The positions of the electrodes were marked on the skin by small ink dots to ensure the same electrode positioning in each test during the experimental period [15]. EMG signals were recorded telemetrically (Glonner Biomech 2000) and stored on magnetic tape (Racall 16) and to the computer with CODAS computer system (Dataq Instruments, Inc.). EMG signal was amplified (by a multiplication factor of 200, low-pass cut-off frequency of 360 Hz 3dB-1) and digitized at a sampling frequency of 1000 Hz. EMG was full-wave rectified, integrated (iEMG in mV×s) and time normalized. The activity (iEMG) of the VL and VM was averaged and analysed in the maximal force phase (500–1500 ms) of the isometric muscle actions [17].
Blood collection and analyses

Blood samples were drawn from the antecubital vein via multiple venipunctures for the determination of serum total and free testosterone, cortisol and growth hormone concentrations. Finger tip blood samples were drawn for the determination of blood lactate. Hemoglobin and hematocrit were also determined to estimate changes in plasma volume (PV). During the loading session blood samples were drawn before, after two and four sets of leg press, after squats and after knee extensions as well as 15 and 30 min after the loadings. In the morning of the first and second day after the loadings, fasting blood samples were obtained for the determination of basal hormone concentrations. Venous blood samples were drawn for the determination of serum creatine kinase (CK) activity before as well as 24, 48 and 72 h after the loadings at the same time of day that each subject had done his heavy resistance loading protocols. All blood samples were obtained at the same body position of the subject.

Serum samples for the hormonal analyses were kept frozen at −20 °C until assayed. Serum testosterone concentrations were measured by the Chiron Diagnostics ACS:180 automated chemiluminescence system using ACS:180 analyzer (Medfield, MA). The sensitivity of the testosterone assay was 0.42 nmol/l, and the intra-assay coefficient of variation was 6.7%. The concentration of serum free testosterone was measured by radioimmunoassays using kits from Diagnostic Products Corp. (Los Angeles, CA). The sensitivity of the free testosterone assay was 0.52 pmol/l, and the intra-assay coefficient of variation was 3.8%. The assays of serum cortisol were carried out by radioimmunoassays using kits from Farnos Diagnostica (Turku, Finland). The sensitivity of the cortisol assay was 0.05 nmol/l, and the intra-assay coefficient of variation was 4.0%. Concentrations of growth hormone were measured using radioimmunoassay kits from Pharmacia Diagnostics (Uppsala, Sweden). The sensitivity of the GH assay was 0.2 µg/l, and the intra-assay coefficient of variation was 2.5 – 5.0%. All the assays were carried out according to the instructions of the manufacturers. All samples for each test subject were analysed in the same assay for each hormone [15]. Hormone concentrations were not corrected for plasma volume changes since the target tissues sense the actual molar concentrations [22]. Blood lactate concentrations were determined using a Lactate kit (Roche, Germany). Serum CK activity was determined using a Creatine Kinase kit (Roche, Germany).

Recovery after the loadings

The rate of recovery after the MR and FR loadings was studied at 24 h, 48 h and 72 h after the loadings. The measurements were done at the corresponding time of the day as the subject’s heavy resistance loading protocols. The recordings of maximal isometric force and concurrent EMG evaluated the recovery of the neuromuscular performance after the loadings. Serum CK activity, subjective muscle soreness (DOMS) and muscle swelling were also determined as markers of muscle disruption possibly caused by the resistance exercises. DOMS was rated on a scale of 0 (no pain) to 10 (maximum pain) for the overall muscle soreness of the quadriceps muscles. To examine muscle swelling, the thickness of the vastus lateralis was measured at the middle of the thigh with ultrasound (US) (Aloka SSD-2801s). The US measurements were taken twice at each time point and the mean of the two measurements was used in the statistical analysis. Acute muscle swelling was also measured by examining effects of the resistance exercise on the thickness of the loaded muscles. Blood samples for the determination of basal hormone concentrations were drawn from each subject after twelve hours of fasting and approximately eight hours of sleep in the first and second mornings after the loadings.

Statistical analyses

Standard statistical methods were used for the calculation of means, standard deviations (SD), standard errors (SE) and Pearson bivariate correlation coefficients. The changes in the variables over time from the pre-level were analysed using general linear model (GLM) analysis of variance with repeated measures. Differences between the variables within each time point were analysed utilizing paired-samples of t-tests. The p < 0.05 criterion was used for establishing statistical significance.

Results

Loading

According to the design of the study, the mean (± SD) load was 13.2 ± 11.2% higher in all FR sets than in MR sets (p < 0.001) (Fig. 2). The average duration of the concentric phases was 28.4% longer in FR (1537 ± 467 ms) than in MR (1100 ± 257 ms) (p < 0.001). In general, assistance was given for 6.0 ± 3.2 repetitions of the 12 repetitions sets in the FR loading. The total volume of the work (loads × sets × reps) in MR was 12 616 ± 1807 kg and in FR 14 126 ± 1744 kg (p < 0.001). However, when the amount of external force produced by the assistant during the concentric phases of the FR repetitions was taken into account, the actual total volume of FR did not differ from that of MR.

Acute neuromuscular responses

Isometric force

Significant decreases (p < 0.05 – 0.01) occurred in maximal isometric force in both loading protocols (Fig. 3). Maximal isometric force decreased during the entire course of the loading in MR down to 61.7 ± 9.3% (from 3024 ± 463 N to 1859 ± 383 N, p < 0.001) and in FR down to 43.5 ± 12.2% (from 3031 ± 498 N to 1290 ± 360 N, p < 0.001). The decrease in isometric force was greater (p < 0.01 – 0.001) in FR than in MR during the entire
course of the loadings. The decreased isometric force remained lower \((p < 0.01)\) in FR than in MR for 24 h (Fig. 3). In FR maximal isometric force was still lower \((p < 0.05)\) at the third day after the loading as compared to the pre-level.

**EMG activity**

There were no significant changes in the maximum integrated EMG of the isometric action at any time points in MR, but FR led to the decreases \((p < 0.05 - 0.01)\) in the EMG (Fig. 3). The changes in maximal isometric force and the changes in EMG correlated with each other after the entire FR loading \((r = 0.51, p < 0.05)\). The EMG was significantly lower \((p < 0.05 - 0.01)\) in FR compared to MR after the second and fourth leg press sets and after the squat sets. There were no statistically significant alterations in EMG activity during the recovery as compared to the pre-level or between the loadings.

**Blood lactate**

The blood lactate concentration increased up to \(14.2 \pm 3.2\) mmol/l \((p < 0.001)\) and up to \(15.0 \pm 2.8\) mmol/l \((p < 0.001)\) after the entire course of the MR and FR loadings, respectively. There were no significant differences between the loadings.

**Plasma volume**

PV decreased after the entire loading by \(-11.8 \pm 6.8\% \ (p < 0.001)\) and \(-14.4 \pm 3.3\% \ (p < 0.001)\) in MR and FR, respectively, with no significant differences between the loadings.

**Acute hormonal responses**

**Control samples**

There were no significant differences in the concentrations of serum hormones examined between the two control blood samples drawn during the control day with no exercise (data not shown).

**Exercise samples**

Serum testosterone concentrations increased after the entire course of the MR and FR loadings from \(22.5 \pm 7.0\) nmol/l up to \(25.2 \pm 9.2\) nmol/l \((p < 0.05)\) and from \(23.2 \pm 7.9\) nmol/l up to \(26.8 \pm 7.5\) nmol/l \((p < 0.05)\), respectively (Fig. 4). After the MR loading serum testosterone concentrations decreased significantly below the pre-level \((p < 0.05, \text{post } 30\text{ min})\).

Serum free testosterone concentrations increased after the entire course of the MR and FR loadings from \(53.8 \pm 13.9\) pmol/l up to \(72.5 \pm 27.9\) pmol/l \((p < 0.001)\) and from \(58.3 \pm 18.1\) pmol/l up to \(79.1 \pm 25.7\) pmol/l \((p < 0.01)\), respectively (Fig. 4). After the FR loading serum free testosterone concentrations decreased significantly below the pre-level \((p < 0.05, \text{post } 30\text{ min})\). There were no significant differences between the MR and FR loadings.

Serum cortisol concentrations increased after the entire course of the MR and FR loadings from \(0.36 \pm 0.12\) \(\mu\text{mol/l}\) up to \(0.53 \pm 0.13\) \(\mu\text{mol/l}\) \((p < 0.001)\) and from \(0.35 \pm 0.11\) \(\mu\text{mol/l}\) up to \(0.65 \pm 0.11\) \(\mu\text{mol/l}\) \((p < 0.001)\), respectively (Fig. 5). The acute responses in cortisol \((p < 0.05)\) were larger in FR than in MR after the squat and knee extension sets as well as 15 and 30 minutes after the loadings. The changes in maximal isometric force and the changes in serum cortisol concentrations correlated with each other after the entire FR loading \((r = -0.55, p < 0.05)\).
**Serum GH concentrations** increased from 1.0 ± 1.9 µg/l up to 23.6 ± 15.2 µg/l (p < 0.001) and from 0.3 ± 0.8 µg/l up to 28.6 ± 16.3 µg/l (p < 0.001) after the entire course of the MR and FR loadings, respectively. The relative changes in GH concentrations were greater (p < 0.05 – 0.01) in FR than in MR already after the four sets of leg press, and remained larger throughout the entire loading and the recovery of 30 min (Fig. 6). The changes in blood lactate and the changes in serum GH concentrations correlated with each other in both MR (r = 0.66, p < 0.01) and FR (r = 0.55, p < 0.05) loadings after the two sets of knee extensions.

**CK-activity and muscle soreness**

CK-activity peaked at 24 hours after MR and FR from 128.4 ± 114.8 IU/l up to 237.8 ± 127.7 IU/l (p < 0.01) and from 67.9 ± 55.7 IU/l up to 251.8 ± 168.4 IU/l (p < 0.001), respectively. Subjective muscle soreness ratings peaked at 48 hours after MR and FR from 0.2 ± 0.3 up to 3.4 ± 2.0 (p < 0.001) and from 0.2 ± 0.3 up to 3.7 ± 2.9 (p < 0.001), respectively.

**Thickness of the vastus lateralis**

VL thickness increased after the entire course of the loadings by 9.3 ± 5.1% (p < 0.001) and by 11.8 ± 5.7% (p < 0.001) in MR and FR, respectively. The values decreased gradually during the recovery period in both loadings.

**Basal hormone concentrations**

Basal hormone concentrations were unaltered compared to the control day morning values after the loadings (Table 1), except for the increased free testosterone values at 48 hours after the MR (p < 0.01) and FR (p < 0.05) loading protocols.

**Discussion**

The primary findings of this study were that both maximum repetition and forced repetition loading protocols led to the great acute hormonal responses. There were no differences between the loadings in the acute testosterone and free testosterone responses, while the cortisol and relative GH responses were greater in FR than in MR. Both loading protocols led also to major neuromuscular fatigue observable with the acute decreases in isometric force associated with high blood lactate concentration, while only FR led to the decrease in the maximal voluntary EMG of the loaded muscles. Also the recovery in maximal isometric force after the loading was slower in FR than MR.

The present study was, to our knowledge, the first one to examine the exercise and recovery profiles of acute hormonal responses when the exercise regimen included several multiset exercises for the same muscle group (i.e. leg press, squat, and knee extensions for the quadriceps femoris) as used in typical hypertrophic type of strength training. Forced repetitions are a special resistance training system, which is used to increase the intensity of training. It may not be common among strength trainers to perform forced repetitions in every set and more than three to four of them per set, but in the present study the number of the forced repetitions was intentionally high in order to create the different experimental conditions between the two loadings. The MR loading was performed first in the experimental design.
due to practical reasons. In this way it was possible to obtain the true strength level of the subjects at the moment and to create maximal loading for both loading protocols. It cannot be excluded, but there are no serious reasons to believe that the acute hormonal or neuromuscular responses could be affected by the previous loadings in the present study. However, contrary to acute hormonal and neuromuscular responses the muscle damage markers as CK-activity, subjective muscle soreness and muscle swelling could be diminished on the second experimental loadings via prophylactic effect of the previous loading [28].

Testosterone promotes muscle hypertrophy via enhancing protein synthesis and may also contribute to force production by its potent influence on neural mechanism (e.g. increased neurotransmitter synthesis) [4,27,29,30]. Heavy resistance exercise-induced acute increases in serum testosterone concentration may be caused by the influence of the increased circulation in the testicles, activation of the sympathetic nervous system, increased lactate accumulation and/or luteinizing hormone concentrations [7,8,18]. Changes in the plasma volume and the increased clearance of the circulating testosterone may also, at least in part, explain the exercise-induced testosterone response. In the present study serum testosterone and free testosterone concentrations increased significantly during both loading protocols with no significant differences between the loadings. The contribution of free testosterone represents the amount of bioactive testosterone, which can act directly with androgen receptors in the target tissue (e.g. skeletal muscle) to mediate changes of the function of a muscle cell via enhanced protein synthesis. Regardless of the mechanism(s) of an exercise-induced increase of serum testosterone concentrations, the skeletal muscle will be exposed to an elevated peripheral testosterone concentration and thus the likelihood of possible interactions with potential muscle cell receptors could increase. Furthermore, the increase of testosterone concentration in serum has been connected to up-regulation of androgen receptors [5]. In the present study, serum testosterone concentrations decreased 30 minutes after MR below the pre-level, which is probably caused by the lowered LH response of the testicles due to the exercise-induced increase in serum testosterone concentration. Interestingly, serum total testosterone concentration turned to a downward trend already after the squat exercise, especially in FR. This may be due to smaller activated muscle mass in the knee extension exercise, lowered blood lactate concentration, failure in testosterone production and/or increase in its hepatic clearance. Contrary to total testosterone, serum free testosterone increased throughout the entire loading. The concentration of serum free testosterone was also increased on the second morning after both loadings as compared to the control day value. That is perhaps an indication of a compensation mechanism of the hormonal system against the exercise-induced stress.

Cortisol influences, among others, the metabolism of amino acids and glucose. Cortisol increases gluconeogenesis and recycling of proteins. Amino acid availability is an important regulator of the muscle protein metabolism [2]. In the postexercise recovery period cortisol contributes to maintain sufficient rates of glycogen synthesis, protein turnover and supply of protein synthesis by amino acids [33]. On the other hand, the exercise-induced increase in cortisol concentration may inhibit the secretion of GnRH [26] and/or the increased level of ACTH may compete with LH of the androgen receptors of Leydig cells [1]. In the present study the exercise-induced cortisol response was significantly greater in FR than in MR. The exercise response occurred in FR after the four sets of leg press and in MR after the squat, when the threshold for the cortisol response was probably exceeded [32]. Exercise-induced cortisol response may be due to glycolytic demands of the exercise, stimulated effect of catecholamines and/or a consequence of neural control of muscle work. The exercise response of endocrine action is triggered by the central motor command and the responses are further supported by positive feedback influences from proprio- and metaboreceptors in muscles [20]. In the present study, the changes in maximal isometric force and changes in EMG as well as the changes in maximal isometric force and the changes in serum cortisol concentration correlated significantly between each other in FR. This may partly support the hypothesis for the influence of neural control of the muscle work to exercise-induced cortisol response.

GH has anabolic effects on the muscle cell by increasing transport of amino acids in the muscle cell and increasing protein synthesis. GH is an anabolic hormone, and therefore, heavy resistance exercise-induced increased secretion of GH may be important for the process of training-induced muscle hypertrophy [24]. In the present study, serum concentrations of GH increased greatly after both loadings but the relative change in the GH response was significantly greater in FR than in MR. Exercise-induced increase in serum GH concentrations measured by the immunoactive method (molecular size 22kDa) can be explained mainly by hypoglycemia and the stimulatory effect of the motor cortex and the sympathetic nervous system of the hypothalamus. Further explanation might also be an increased acidity in the muscle caused by anaerobic muscle work, which stimulates metaboreceptors and sendsafferent feedback to the central nervous system and hypothalamus leading to an increased secretion of GH [11,12,19]. This is supported by the present study showing that serum GH concentrations correlated with blood lactate concentrations in both loadings. In the present study, the deceased maximal voluntary isometric actions were also associated with the decreased EMG activity of the loaded muscles during the FR loading. Therefore greater relative increase in GH concentrations in FR may indicate the importance of the central motor command to the exercise-induced GH response.

The investigation of the rate of recovery after the heavy resistance exercise may be advantageous to estimate a proper strength training frequency and/or intensity and/or volume. Especially the forced repetitions resistance exercise system is supposed to cause muscular soreness [9]. Mechanical load due to resistance exercise may cause some level of myofibrillar disruptions to the activated muscles. In the present study maximal isometric force, subjective muscle soreness, muscle swelling and CK activity were used to estimate the level of muscle damage caused by the heavy resistance exercise and rate of recovery after the loadings. Maximal isometric force was significantly more lowered in FR than in MR during the recovery and isometric force remained significantly lowered even on the third day after FR. There were no significant alterations in the maximum iEMG during the recovery period. Thickness of the vastus lateralis (i.e. muscle swelling) increased during the present loading session and returned
to the pre-exercise level on the next day. Although the loading regimens were strenuous, the muscle damage markers as CK-activity and subjective muscle soreness did not increase greatly after the present kinds of high load and low velocity types of dynamic muscle actions.

The load was greater in FR especially at the beginning of the loading session than in MR. However, when the force of the assistance was reduced from the total load, there was no significant difference between FR and MR in the total work that the subjects had performed by themselves. Nevertheless, FR led to a greater decrease in maximal isometric force than MR. That may be due to a greater accumulation of lactic acid to working muscles during FR and/or the decrease in the ability to activate motor units (especially type II) by the central nervous system [17]. The larger increase in blood lactate concentration and the greater decrease in EMG in FR compared to MR supports this suggestion. It has been demonstrated earlier that the decrease in maximal strength during high-intensity fatiguing strength training sessions may be associated with the decrease in the maximal voluntary neural activation of the exercised muscles [14,17]. The present FR protocol may produce greater peripheral and central fatigue compared to MR where fatigue was caused mostly by the peripheral factors. The greater blood lactate concentration may be due to a longer concentric phase of the dynamic muscle actions in FR than in MR as a consequence of the higher load used in FR. Therefore, blood circulation may decrease in the activated muscles and acid metabolic waste products accumulated in the muscle cells [13]. The larger decrease in isometric force associated with decreased neural activation of the loaded muscles and the higher GH response in FR than in MR indicate that the usage of the forced repetitions protocol may be beneficial for the development of muscle mass and muscle strength during strength training. On the other hand, the lowered recovery of force production in FR may indicate an increased risk for overtraining if the training frequency is kept too high and/or the overall volume of each training session is too high.

In summary, the present data showed that FR compared to MR loading led to greater stress for the neuromuscular system shown through the larger decrease in isometric force and iEMG of the loaded muscles. Furthermore, the recovery of the isometric force was slower after the FR than MR loading. Because the degree of acute neuromuscular fatigue and the time needed for recovery may differ considerably between the MR and FR loading protocols, there is a need to optimize the contents and the frequency of different training sessions in order to create proper strength training programs to match the individual requirements of athletes. The FR loading stimulated the hormonal system, especially the secretion of GH, more than MR did. Whether the repeated usage of the multiple FR protocol is associated with larger gains in muscle mass or strength during prolonged strength training, and how any differential gains are related to acute and long term endogenous anabolic adaptations needs to be examined.

References