



Mechanical Responses of the Human Triceps Surae after Passive “Stretching” Training of the Plantarflexors in Conditions Modulating Weightlessness

by
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The effect of a 60-day 6° head-down tilt of bed rest with and without prolonged passive muscle “stretching” training on the mechanical properties of the human triceps surae muscle was studied in 13 healthy male subjects. One group ($n = 6$; mean age 30.8 ± 3.1 years) underwent a 60-day head-down tilt, and a second group ($n = 7$; mean age 30.4 ± 1.2 years) underwent head-down tilt with prolonged passive muscle stretching. Head-down tilt without prolonged passive muscle “stretching” training showed maximal voluntary contraction declined by 34 % ($p < 0.05$) and the electrically evoked tetanic tension at $150 \text{ impulses}\cdot\text{s}^{-1}$ and isometric twitch contraction reduced by 17 % ($p < 0.02$) and 18 % ($p < 0.05$), respectively. Time-to-peak tension, and half-relaxation time of the twitch slightly decreased by 3% ($p > 0.05$), and 7 %, respectively, but total contraction time slightly increased. The difference between electrically evoked tetanic tension and the maximal voluntary contraction expressed as a percentage of electrically evoked tetanic tension (referred to as force deficiency), has also been calculated. The force deficiency increased by 61 % ($p < 0.001$). After head-down tilt with prolonged passive muscle “stretching” training, the time-to-peak tension did not change, and half-relaxation time of the twitch decreased. In addition, there was a 14 % lengthening in the total duration of the twitch. The results of prolonged passive muscle “stretching” training demonstrated a clear deterioration of voluntarily and electrically induced muscle contractions. Passive “stretching” training caused a decrease by 29 % ($p < 0.05$) in the maximal voluntary contraction. The isometric twitch contraction, and electrically evoked tetanic tension both showed reductions by 17 %, and by 19 % ($p < 0.05$), respectively. The force deficiency decreased significantly by 21 % ($p < 0.02$). The rate of rise of electrically evoked tetanic tension and feature of voluntary contractions significantly reduced during head-down tilt with prolonged passive muscle “stretching” training. These basic experimental findings concluded that prolonged passive “stretching” training of a single muscle did not prevent a reserve of neuromuscular function.

Key words: bed-rest – static (passively) stretching – contractile properties – triceps surae muscle

Introduction

Skeletal muscle demonstrates a remarkable ability to adapt to alterations in activity level. Sustained reductions in muscle activity, via spinal cord transection, hindlimb suspension, or exposure to micro-

gravity conditions, result in a rapid and dramatic reduction in muscle fiber size and contractile profile (Edgerton et al., 1995; Staron et al., 1998; Fitts et al., 2000; Koryak, 1998a, 2001; 2003; Akima et al., 2001; Kawakami et al., 2001). It is accepted that the major factor responsible for all of these changes is the sud-

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den elimination of the proprioceptive information from the muscle and tendon in response to absence of load-bearing. Accordingly, the anti-gravitational, or the postural muscle, should be the principal target for the action of unloading.

Studies simulating microgravity have shown that exercise countermeasures can attenuate, but not completely prevent the loss of muscle mass and force (Koryak, 1998a; Akima et al., 2001; Kawakami et al., 2001). The muscle groups most affected by exposure to microgravity appear to be the antigravity extensors of the knee and ankle (Akima et al., 2001). Among these, the plantarflexors seem to be the most affected (Akima et al., 2001), likely due to their greater mechanical loading under normal gravitational conditions.

Among various mechanisms, such as starting mechanisms of muscular changes, the important role belongs to updating nervous-muscular activity, as structural and functional integrity of skeletal muscle depends on normal nervous function (Pette and Vrbova, 1985). It is recognized that a primary factor responsible for changing physiological functions, is the sudden elimination of proprioceptive input from muscles and tendon in response to absence of weight loading (Leterme and Falempin, 1996). In connection to this phenomenon it is necessary, to remove loading and to increase afferent input from "working" muscles using various preventive means, from artificially created support for stops (Stump et al., 1990) up to complex forms of physical exercises (Stepantsov et al., 1972). However, not all preventive means completely prevent muscular changes (Koryak, 1998; Akima et al., 2001; Kawakami et al., 2001).

It is well known that when the skeletal muscle is maintained in a lengthened position, the changes and the rate of atrophy due to immobilization are reduced (Spector et al., 1982; Steffen, Mussacchia, 1984). Muscle stretching has been used for a long time not only in physiotherapy during the treatment and rehabilitation of patients (Kabat, 1958; Knoll and Voss, 1968) but also in preparation for athletic performance (Möller et al., 1985; Hortabagyí et al., 1985; Cornwell et al., 2001).

In light of these observations, it seems reasonable that prolonged passive, static, stretching (PPS) could be used to control muscular wasting and dysfunction that may accompany adaptation to weightlessness. There are no relative studies concerning long-term use of passive distention of a muscle and changes in contractile properties, especially of

a model in simulated weightlessness. It was, subsequently, presumed by Kozlovskaya (2001) that prolonged, passive, static, stretching of a muscle is an effective modality for counter-measuring the negative influence of weightlessness. Thus, we postulated that long-term passive, static, stretching reduces contractile properties of a muscle. This paper examines these questions in the human triceps surae muscle.

The PPS, as a methodological approach, has been used in a number of research contexts to study mechanical factors of force production (Edman, 1988), stress-relaxation characteristics of muscle (Toft et al., 1989; Taylor et al., 1990; McHugh et al., 1992; Magnusson, 1998;), neuromuscular reflex patterns (Guisard et al., 1988; Hutton, 1992), factors contributing to muscle damage (Lieber et al., 1991; Armstrong et al., 1993) mechanisms of hypertrophy (Timson, 1990) and stretching parameters relating to the development of flexibility (Toft et al., 1989; Magnusson, 1998). Despite this breadth of information, there is very little research on factors that may influence measured force in twitch or maximum contractions as affected by PPS.

Many factors influence directly on contractile properties of a muscle as a result of PPS. The PPS of a muscle (less than 45 s) causes reduction of tension (i.e., muscle stiffness (Magnusson et al., 1996)) and repeated stretching increases the length of a muscle (Magnusson et al., 1996). Reduced muscle stiffness can affect of electrically evoked twitch contractions. The reduced rigidity of a muscle can change the form of twitch contractions, because greater time is necessary to stretch in-series elements (Caldwell, 1995), and increased muscle length may alter the fine balance of muscle properties and joint kinematics that combine to produce force (Lieber, Boakes, 1988). Altered force-length characteristics may influence neural activation patterns because of altered proprioceptive feedback.

The PPS of skeletal muscle in animals (Lieber et al., 1991; Armstrong et al., 1993) and humans (Avela et al., 1999; Fowles et al., 2000) significantly reduces the ability to generate force of reduction of a muscle and reduces serviceability in connection with reduction in the neural input to the muscle (Avela et al., 1999; Fowles et al., 2000). The PPS duration of 30 min causes a 13% decrease in maximum electrically evoked tetanic tension (P_0) of a muscle (Fowles et al., 2000), and during 2-h of PPS, significant decrease (61 %) in twitch force are observed (Armstrong et al.,

1993). One-hr PPS of human ankle plantarflexors is accompanied by a decrease in mechanical response (maximal voluntary force, twitch force, and total contraction time) (Avela et al., 2004), and electromyography activity of the *m. gastrocnemius* and *m. soleus* (Avela et al., 1991, 2004). The cause of this contractile impairment has often been contributed to the increased compliance of the muscle tendon unit induced by the stretch. This could then result in impaired force transfer from the muscle fibers via the tendon to the bone (Fowles et al., 2000), and thereby altering proprioceptive feedback (Avela et al., 2004).

The purpose of the present study was i) to examine the physiological adaptation (during twitch, voluntary contraction, and tetanic contractions) to a disuse condition (i.e., 60-days of simulated weightlessness, and ii) to investigate how the PPS influences the mechanical properties of ankle plantarflexors in humans during 60-days of simulated weightlessness.

A brief report of part of this work has appeared elsewhere (Koryak et al., 1997).

Methods

The experimental protocol was approved by the Russian National Committee on Bioethics of the Russian Academy of Sciences and was in compliance with the principles set forth in the Declaration of Helsinki.

Subjects

The experiments were carried out on 13 healthy male volunteer subjects (mean \pm SEM), 23-42 years of age (30.3 ± 1.5), 172-190 cm in height (179.2 ± 1.5), and 63.0-115.0 kg in body mass (75.0 ± 3.9), after explanation of the experimental protocol. The experimental protocol consisted of 10 ($n = 7$) or 8 ($n = 6$) days of pre-bed-rest ambulatory control, and 3 ($n = 8$) or 4 ($n = 5$) days after-bed-rest.

Selection of subjects was based on a screening evaluation that consisted of a detailed medical history, physical examination, complete blood count, urinalysis, resting and cycle ergometer and electrocardiogram, as well as a panel of blood chemistry analysis, which included fasting blood glucose, blood urea nitrogen, creatinine, lactic dehydrogenase, bilirubin, uric acid, and cholesterol. All subjects were evaluated clinically and considered to be in good physical condition. No subject was taking medication at the time of the study, and all subjects were nonsmokers and recreationally active, but not

especially well-trained. None of the subjects suffered from neuromuscular disease. The subjects were kept under medical observation. Each subject served as his own control.

The subjects were randomly assigned into two groups and two studies were performed. Members of first group ($n = 6$) (mean \pm SEM: age 30.8 ± 3.1 years, height 181.3 ± 2.3 cm, body mass of 79.8 ± 7.7 kg) underwent a 60-day bed-rest at a 6° head-down-tilt (HDT) position and remained maintained the HDT position without interruption for all 60-days. Throughout the study, the subjects were fed three times per day with the daily total energy intake being $\sim 3,000$ kcal for all subjects. The temperature and humidity in the room were maintained within a normal range, and the day-night cycle was regulated at 8 am and 6 pm.

Members of second group ($n = 7$) (mean \pm SEM: age 30.5 ± 1.9 years, height 180.0 ± 3.0 cm, body mass 72.0 ± 3.3 kg) wore the full assembly of the Penguin suit and were subjected to bed rest at a 6° HDT position for 2 months.

All subjects were informed about the possible risks and a signed informed consent was obtained from each subject

Experimental design

Subjects completed two experimental protocols. Each protocol involved measurement of tension properties, velocity properties, and force-velocity properties of the triceps surae muscle before and after the 60-day period of HDT (-6°) bed-rest, with and without PPS training.

Experimental protocol

Strict bed-rest

After an intensive familiarization period of 3 months before bed-rest, subjects entered the Human Research Facility of the Health Ministry Institute of Biomedical Problems. During this 60-day HDT period (experimental condition), the subjects lived 24 h-day⁻¹ in the Human Research Facility of the Health Ministry Institute of Biomedical Problems and remained the HDT position continuously for all activities, including excretory functions, showering, and eating. The -6° HDT position was chosen due to many changes in physiological responses during spaceflight which closely simulated this ground-based model (Convertino, 1996). All subjects abstained from tobacco, alcohol, caffeine, medications, and conventional exercise for the duration of the study.

Passive muscle stretching

PS the triceps surae muscle was performed by offset shock-absorbers individual suits (Penguin suit) (Barer et al., 1975). The Penguin suit was made of elastic (rubber) loading elements. Elastic cords which constituted a part of this suit provided the axial compression (from the shoulders to the feet) on weight-bearing muscles and skeleton during a prolonged period of time. The inherent value of shock-absorbers depends on personal motility in the ankle joint, made ~20-25 N, that resulted to a dorsiflexion at an angle of ~10° (i.e., at ~10° dorsiflexion relative to a 90° ankle position).

Experimental set-up and force measurements.

Isometric dynamometry

The subjects were carefully familiarized with the testing procedures of voluntary force production during several warm-up contractions preceding the actual maximal contractions, and were allowed to habituate to the electrical stimulation procedures during preliminary visits to the laboratory before definitive control measurements were taken. In addition, the subjects were trained to perform volitional contractions and to relax during electrically elicited

contractions of their dominant foot (the triceps surae muscle group). On each occasion the subjects were required to sit in a specially designed leg tendometrical set-up with a standard position (knee joint angle between tibia and sole of foot of 85–90°; see Figure 1 ref. 1995). The dynamometer and recording system used to measure the forces produced by electrical and voluntary contractions of the triceps surae muscle group have previously been described in detail (Koryak, 1995; 2001). During a recording session, the subject was seated in a chair with the right foot strapped to a foot plate. Knee joint angle between tibia and sole of foot was 85-90°. Contractile properties of the triceps surae muscle group were tested twice: 8-10 days before the beginning of bed-rest affect and 3-days after bed-rest concluded. The testing protocol was identical for both pre-bed rest and post-bed-rest testing.

Electrical stimulation

To stimulate the tibial nerve, an active monopolar electrode--an active electrode (cathode)-- 1 cm in diameter was located in the popliteal fosse, the location of lowest resistance; the passive electrode (anode), 6 x 4 cm in size, was positioned on the lower one-third of the surface of the thigh. The large earth electrode (7.5 x 6.5 cm) was located on the

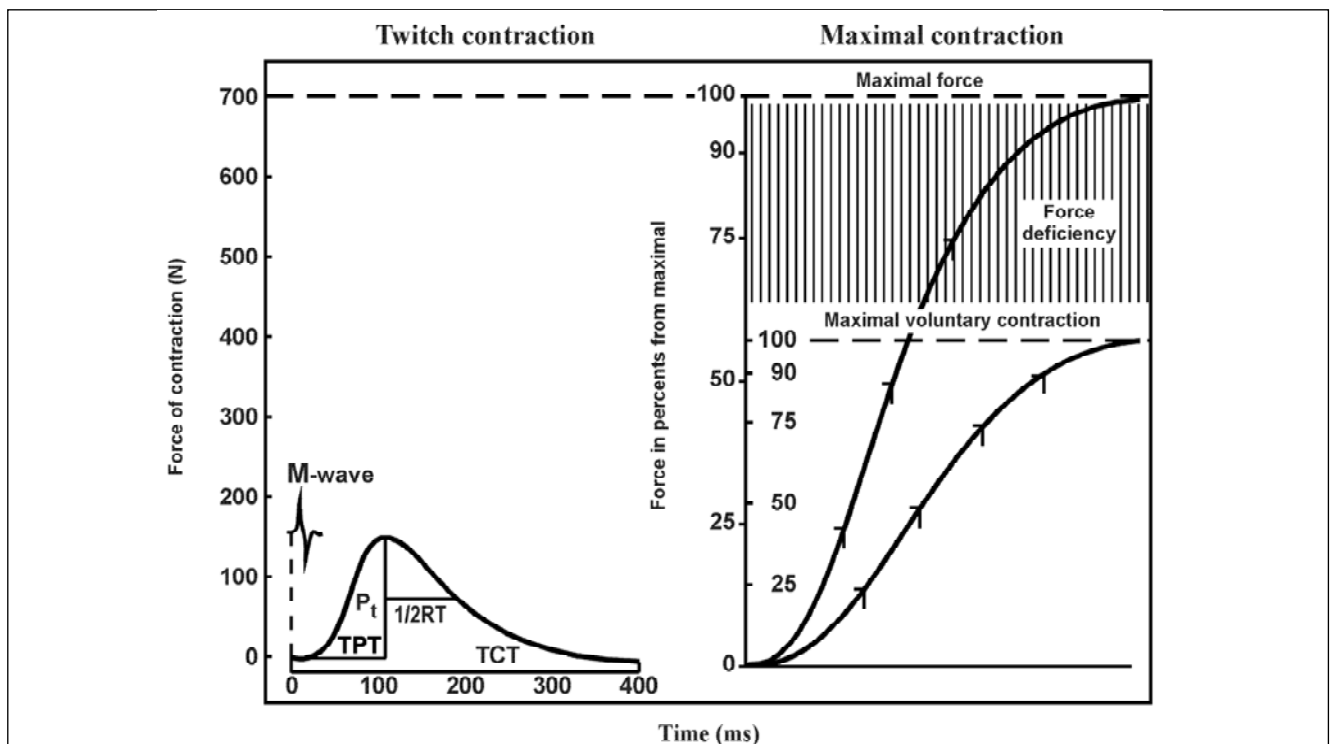


Figure 1

Measurements of mechanical response parameters in the isometric twitch contraction curve (left) and in electrically evoked titanic tension and voluntary muscle tension development (right).

TPT, a time-to-peak; 1/2 RT, a half-relaxation time; TCT, a total contraction time; P_t, a twitch force.

proximal portion of the leg between the pick-up and stimulating electrodes.

Bipolar Ag-AgCl surface electrodes, with an electrode spacing of 2 cm, were placed over the soleus muscle, parallel to the Achilles tendon, below the gastrocnemius muscle. The inter-electrode impedance was less than 5 k Ω .

The stimulation was performed by supramaximal voltage rectangular wave pulses (voltage was 30–40 % greater than minimal voltage at which a maximal M-wave of the muscle had been obtained for the first time) of 1-ms duration and with a frequency of 150 impulses·s⁻¹ (Koryak, 1995).

Tension properties

Isometric twitches were elicited with brief rectangular wave pulses (1 ms), at 30s intervals and by progressively increasing voltage until maximal force (P_t) was obtained (Figure 1).

On double stimulation, when the second impulse was generated at intervals of 3, 4, 5, 10, 20, and 50 ms (Koryak, 1995), the maximal amplitude (strength) of the muscle contraction was determined.

After an appropriate period of rest, subjects performed a maximal voluntary contraction (MVC) of the triceps surae muscle group on the instruction "to exert maximal contraction". Between 2–3 maximal concentrations were usually recorded for each subject until maximal force concentration was

obtained. In each set, the contractions were performed at a rate of 0.5·s⁻¹. There was a 1-min rest between the sets. The MVC was determined as the highest value of voluntary force recorded during the entire contraction. The force was recorded on a magnetic tape.

The maximal force (P_o) was estimated by the tendonogram from the evoked contraction in response to an electrical titanic stimulation of the nerve, innervating the triceps surae muscle group, with a frequency of 150 impulses·s⁻¹ (Koryak, 1995).

The difference between P_o and MVC expressed as a percentage of the P_o value and referred to as force deficiency (F_d) has also been calculated (Koryak, 1995; 2003).

Velocity properties

From the maximal twitch response, the force (P_t), the time-to-peak tension (TPT), half-relaxation time (1/2 RT), and total contraction time (TCT), respectively defined as the time elapsed from the moment of stimulation to peak force, the time from peak to half-force, and the time from the moment of stimulation to the total muscle relaxation. The skin temperature was 35° C, maintained by an infrared lamp and a sensor on the skin over the triceps surae muscle group. The accuracy of measurement was 2 ms.

Force-velocity properties

The rate of development of increased muscle ten-

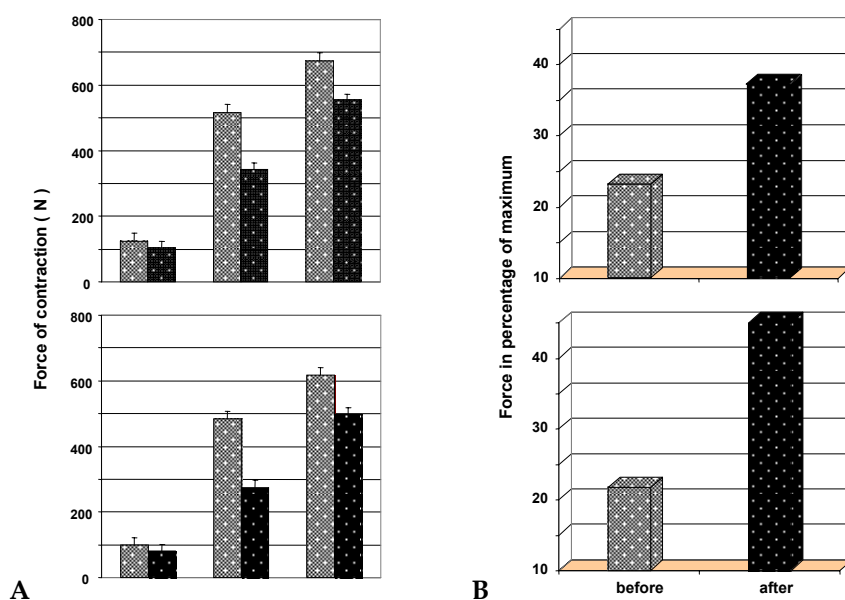


Figure 2

- A. The effect of 60-day head-down tilt (HDT) (top panel) and HDT with prolonged passive "stretching" training (bottom panel) on maximal voluntary contraction (MVC), evoked tetanic tension at 150 impulses·s⁻¹ (P_o), and on tension of the maximal twitch (P_t) of the triceps surae muscle.
- B. Maximal voluntary contraction force as % P_o after 60-day 6° head-down tilt (HDT) (top panel) and HDT with prolonged passive "stretching" training (bottom panel).

Table 1

Mechanical properties of skeletal muscle in the control group and after 60-day bed-rest, with and without prolonged passive muscle "stretching" training

	TPT, ms	1/2 RT, ms	TCT, ms	dP_{vc}/dt , $N \cdot ms^{-1}$	dP_{ec}/dt , $N \cdot ms^{-1}$
<i>Bed-rest without passive "stretching" training (n = 6)</i>					
control	145.3 ± 2.3	97.0 ± 6.8	486.0 ± 17.7	4.75 ± 0.4	7.46 ± 0.4
after	140.3 ± 2.2	90.0 ± 6.8	492.2 ± 20.5	3.40 ± 0.6	7.55 ± 0.5
<i>Bed-rest with passive "stretching" training (n = 7)</i>					
control	132 ± 3.4	97.8 ± 5.6	437.0 ± 32.5	3.83 ± 0.3	8.24 ± 0.4
after	132 ± 5.9	86.3 ± 4.7	497.5 ± 26.3	1.28 ± 0.5	7.65 ± 0.2

sion was calculated from the tendogram isometric voluntary contraction after the instruction "to exert the fastest and greatest tension" using a relative scale (i.e., the time of reaching 25, 50, 75, and 90 % of maximum tension (Koryak, 1995). Similarly, the increasing rate in evoked contractions in response to electric stimulation of the nerve, with a frequency of 150, was determined (Koryak, 1995). The accuracy of measurement was 2 ms.

The maximal rates of voluntary contractions (dP_{vc}/dt), and tetanic (evoked contraction) force development (dP_{ec}/dt) were obtained by differentiation of the analog signal.

Statistical Analyses

Standard descriptive statistics were calculated for baseline levels of MVC, P_t , P_o , F_d , TPT, 1/2 RT, TCT, and the rates of tension development by the voluntary and electrically evoked isometric contractions, with results presented as means ± SEM. The differences between two values were evaluated by Student *t*-tests, when sample sizes were sufficient in numbers. A statistical significance level was chosen at $p < 0.05$. The values given in each experimental group were presented as means. The percent changes of pre-bed-rest and post-bed-rest were calculated.

Results

Bed-rest without PPS training

Force properties. Compared with before bed-rest, contractile properties of the muscle was decreased (Figure 2, A; top panel). Thus, isometric P_t decreased by a mean of 17.3 % (mean ± SEM: pre 124.6 ± 7.8 N compared to post 103.0 ± 10.8 N; $p < 0.05$) MVC decreased by a mean of 33.5 % (mean ± SEM: pre 516.0 ± 42.2 N compared to post 343.4 ± 37.3 N; $p < 0.01$), and P_o was substantially reduced by a mean of

18.0 % (mean ± SEM: pre 765.0 ± 52.0 N compared to post 553.2 ± 52.0 N; $p < 0.02$). MVC was constantly less in comparison with P_o . Compared with before bed-rest and after 2 months of bed-rest, MVC was decreased by 76.9 and 62.8 %, respectively. This indicates, first, that the power capabilities of the neuromuscular apparatus in voluntary reduction are used briefly and, secondly, the degree of voluntary use of power capabilities of the muscle is essentially reduced in conditions of microgravitation. This is confirmed by an increase in F_d . Compared with before bed-rest, the F_d was increased by a mean of 61 % (mean ± SEM: pre 23.1 ± 4.8 % compared to post 37.2 ± 6.6 %; $p < 0.02$) after 2 months of bed-rest, respectively (Figure 2, B; top panel).

The mean changes in the force of the triceps surae muscle group contraction during double stimulation, as a function of interpulse interval (in paired stimulation of the nerve), when the second impulse was applied 3, 4, 5, 10, 20, and 50 ms after the first, are presented graphically in Figure 3 (top panel). The greatest force of contraction under these conditions was at intervals of 4-10 ms before and 4-20 ms after HDT, and decreases or increases in the interval were accompanied by a considerable decline ($p < 0.01-0.001$). However, the curve location pattern at the same interpulse interval was changed: a relative rise of force contraction after 60-day HDT was significantly higher, by comparison with the control value ($p < 0.05-0.01$).

Velocity properties. The change in data of mean time isometric twitch contraction, as a reverse value to contraction velocity for the triceps surae muscle group after a 60-day HDT effect, are given in Table 1. As is seen from the data analysis, the remaining subjects exposed to HDT conditions were accompanied by a statistically insignificant decrease of muscle contraction and an increased relaxation velocity. Thus, TPT and 1/2 RT decreased slightly by a mean

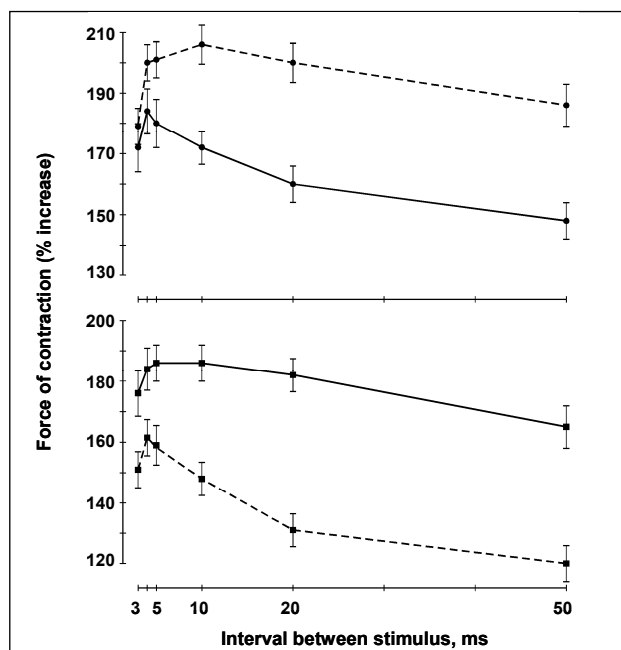


Figure 3

The effect of 60-day 6° head-down tilt (HDT) (*top panel*) and HDT with prolonged passive “stretching” training (*bottom panel*) on the maximal force contraction of triceps surae muscle in response to supramaximal twin stimuli at 3, 5, 10, 20, and 50 ms.

of 3.4 % (mean \pm SEM: pre 145.3 ± 2.3 ms compared to post 140.3 ± 2.2 ms), and by a mean of 7.2 % (mean \pm SEM: pre 97.0 ± 6.8 ms compared to post 90.0 ± 6.8 ms), respectively. Additionally, TCT small insignificantly increased by a mean of 1.2 % (mean \pm SEM: pre 486.0 ± 17.7 ms compared to post 492.2 ± 20.5 ms; $p > 0.05$).

Force-velocity properties. The decrease in the MVC (33.5 %) was associated with a significant slowing in the rate of tension developed during a voluntary “explosive” isometric contraction (Figure 4, *top panel*) and by a decrease of maximal dP_{vc}/dt (pre-bed-rest, 4.75 ± 0.4 N \cdot ms $^{-1}$ vs. post-bed-rest, 3.40 ± 0.6 N \cdot ms $^{-1}$) when measured in absolute terms (Table 1). The normalized values (% of MVC) of dP_{vc}/dt declined by 18.7 %. This may be seen in the decrease in convexity of the force-time curve estimated according to relative scale.

However, analysis of the force-time curve of the electrically evoked contractions did not reveal significant differences (Figure 4, *top panel*) and maximal dP_{ec}/dt (pre-bed-rest, 7.46 ± 0.4 N \cdot ms $^{-1}$ vs. post-bed-rest, 7.55 ± 0.5 N \cdot ms $^{-1}$). The normalized values (% of MVC) were increased by 23.6 % ($p < 0.05$).

Bed-rest with PPS training

Force properties. Compared with conditions before HDT, contractile properties of the muscle was decreased (Figure 1, A; *bottom panel*). Thus, isometric P_i decreased by a mean of 23.4 % (mean \pm SEM: pre 108.9 ± 29.4 N compared to post 83.4 ± 11.8 N; $p < 0.05$), MVC decreased by a mean of 33.4 % (mean \pm SEM: pre 428.7 ± 31.4 N compared to post 285.5 ± 53.0 N; $p < 0.05$), and P_o was reduced by a mean of 24.3 % (mean \pm SEM: pre 691.6 ± 34.3 N compared to post 523.9 ± 76.5 N; $p < 0.05$). MVC was constantly less in comparison with P_o . Compared with before bed-rest and after 2 months of bed-rest MVC was reduced 38.0 and 45.5 %, respectively. This indicates, first, that the power capabilities of the neuromuscular apparatus in voluntary reduction are used briefly and, secondly, degree of voluntary use of power capabilities in the muscle is essentially reduced in conditions of microgravitation. This is confirmed by an increase in F_d . Compared with conditions before HDT, the F_d was increased by a mean of 24.1 % (mean \pm SEM: pre 38.2 ± 2.5 % compared to post 47.4 ± 4.9 %; $p < 0.02$) after 2 months of bed-rest, respectively (Figure. 2, B; *bottom panel*).

The mean changes in the force of the triceps surae muscle group contraction during double stimulation, when the second impulse was applied 3, 4, 5, 10, 20, and 50 ms after the first, are presented graphically in Figure 3 (*bottom panel*). The greatest force of contraction under these conditions was at intervals of 4-20 ms before and 4-5 ms after HDT with use the PS, and decreases or increases in the interval were accompanied by a considerable decline ($p < 0.01-0.001$). However, the curve location pattern at the same inter-pulse interval was changed: a relative rise of force contraction after 60-day HDT was significantly lower, by comparison with the control value ($p < 0.05-0.01$).

Velocity properties. The change in data of mean time isometric twitch contraction, as a reverse value to contraction velocity for the triceps surae muscle group after a 60-day HDT effect, are given in Table 1. As is seen from the data analysis, the remaining subjects exposed to HDT conditions were accompanied by a statistically insignificant decrease of muscle contraction and an increased relaxation velocity. Thus, TPT showed no significant changes (mean \pm SEM: pre 132 ± 3.4 ms compared to post 132.0 ± 5.9 ms), but 1/2 RT decreased slightly by a mean of 11.2 % (mean \pm SEM: pre 97.8 ± 5.6 ms compared to post 86.3 ± 4.7 ms), and TCT increased by a mean of

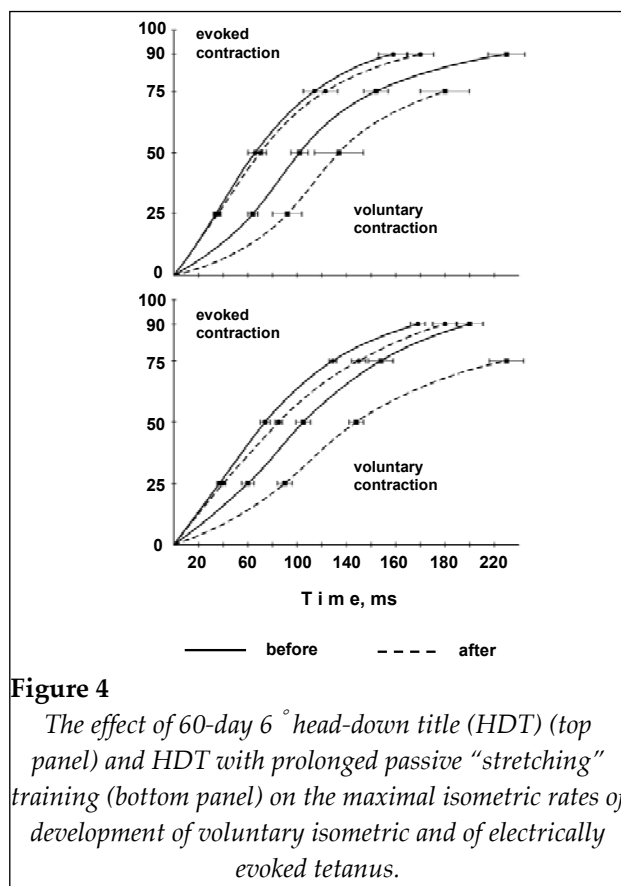


Figure 4

The effect of 60-day 6° head-down tilt (HDT) (top panel) and HDT with prolonged passive "stretching" training (bottom panel) on the maximal isometric rates of development of voluntary isometric and of electrically evoked tetanus.

13.7 % (mean \pm SEM: pre 437.0 ± 32.5 ms compared to post 497.5 ± 26.3 ms; $p < 0.05$).

Force-velocity properties. The analysis of results showed time increases for isometric reduction after HDT. Firstly, the rate of tension developed during a voluntary "explosive" isometric contraction reduction was much less than in electrically evoked contractions, which caused reduction, and secondly, force-time curves essentially differed (Figure 4, bottom panel). The decrease in the MVC (43.0 %) was associated with a significant reduction in the rate of tension developed during a voluntary isometric contraction (Figure 4, bottom panel), which was significantly decreased (66.6 %) from maximal dP_{vc}/dt (pre-bed-rest, 3.83 ± 0.3 N \cdot ms $^{-1}$ vs. post-bed-rest 1.28 ± 0.5 N \cdot ms $^{-1}$) when measured in absolute terms (Table 1). The normalized values (% of MVC) of dP_{vc}/dt increased by 12.5 % ($p < 0.05$). This may be seen in the decrease in convexity of the force-time curve estimated according to the relative scale.

However, analysis of the force-time curve of electrically evoked contractions showed a decrease in the developmental rate of the triceps surae muscle group during isometric tension (Figure 4, bottom panel) and was reduced (by 7.2 %) with maximal dP_{ec}/dt (pre-bed-rest 8.24 ± 0.4 N \cdot ms $^{-1}$ vs. post-bed-

rest 7.65 ± 0.2 N \cdot ms $^{-1}$) when measurements were executed in absolute sizes. Normalized (% MVC) values of dP_{ec}/dt increased by 14.9 % ($p < 0.05$).

Discussion

Bed rest without prolonged passive muscle "stretching" training

The major goal of the present study was to determine the influence of long-term simulated microgravity on the *in vivo* functional properties of the extensor muscles of the foot (triceps surae muscle group) in healthy young males. In the present investigation, the most striking changes in contractile properties of the skeletal muscle occurred after the 6° HDT.

The results showed that a slow contracting muscle group (Vandervoort, McComas, 1983; Koryak, 1994) was affected by disuse, and extended the previous findings concerning the effects of short-term voluntary immersion (Koryak, 1995) and long-term bed-rest (Koryak, 1994b; 1995a), and of long-term spaceflights (Koryak, 2003) on the mechanical characteristics of the triceps surae muscle group. Significant increases in maximal isometric twitch TPT and decreased 1/2 RT and TCT were maintained during a 120-day HDT. A likely explanation of the change in TPT in the disused limbs is a relatively greater atrophy of type I fibres (slow twitch motor units), which has been found to comprise the majority of the triceps surae muscle group (Johnson et al., 1973). However, since disuse produces muscle atrophy, both in fast and slow twitch skeletal muscles and, in addition, have been shown to cause fibre type-specific changes in the contractile properties (Gardetto et al., 1989), other factor(s) may be affected that alter fibre type composition.

The rapid nature of isometric changes (i.e., twitch duration) may be related to alterations in sarcoplasmic reticulum (SR) function (Briggs et al., 1977). The primary factor (mechanism) in the explanation of these changes may be a reduction in the rate at which Ca $^{2+}$ is dissociated from the myofibrillar proteins (Briggs et al., 1977). Dissociation would occur more slowly if the rate of Ca $^{2+}$ re-uptake by SR was decreased. Such a decrease has been found following disuse (Kim et al., 1982). A reduced rate of Ca $^{2+}$ dissociation from myofibrillar proteins might be expected not only to increase the time course of the twitch response, but also to allow more force to be generated, since cross-bridges will continue to be

formed while Ca^{2+} is available in the sarcoplasm. The tendency for P_t to decrease in this study cannot be explained. These effects on SR would be difficult to observe, as the effects on P_t would be masked by atrophy, but are of interest on the assumption that the twitch changes are due to SR alterations. The changes in the kinetics of the mechanical responses to paired stimulation (cf. Figure 2, top) might also be explained by altered development of Ca^{2+} kinetics in the muscles used in the experiment. The reduced twitch duration in the triceps surae muscle group might, in part, result from lower P_t obtained in this muscle (cf. Figure 1, top).

These changes in twitch tension may reflect alterations in muscle extensibility and in temperature. A decrease in muscle extensibility, which has been observed after disuse (Goldspink et al., 1974) would be the changes in isometric P_t . It may be speculated that in this experiment, bed-rest was the cause of a decrease in muscle extensibility. It has been well-documented that body temperature fluctuations cause muscle temperature changes, thus influencing their contractile properties (Davies and Young, 1983). Temperature falls up to 5°C are within physiological limits. The subjects' body temperatures during this study were monitored continuously and were within the normal range (36.4°C - 36.6°C). It has been shown that bed-rest, but not *dry* immersion, can cause a slight decrease in body temperature (Volkov and Molchanova, 1977). Nevertheless, the decreased value was so insignificant that it could not be associated with a change in the propagation velocity of the action potential along the muscle fibre (membranes) (Christova et al., 1986), and can accordingly be disregarded as a factor in the present case.

The decrease in P_t observed in these experiments after long-term 6° HDT is in agreement with previous results which have shown a decrease of maximal force during voluntary contractions and electrically evoked contractions (Koryak, 1995; 2003). It was found that there was in all four subjects a proportionately identical decrease in P_t and in P_o at 150 Hz. After 6° HDT, the electrically evoked contractions (P_o) decreased significantly at a rate of 24 % of normal. The P_o is a direct measure of the force-generating capacity of a muscle and has been considered to reflect the number of active interactions between actin and myosin (Close, 1972). Disuse has been reported to produce a decline in P_o by other workers (Duchateau and Hainaut, 1987; 1990; Koryak, 1995; 2001-2003). This decline could reflect a decrease in

the number of active cross-bridges and be expected to decrease the work capacity.

Two hypotheses may be suggested to account for the observation. First, the total number of cross-bridges could have been smaller after the period of disuse. Second, the force output per cross-bridge could have been decreased. However, Steven et al. (1990) showed that when it was expressed per cross-sectional area (CSA), the force was unchanged after disuse. This would indicate that the first hypothesis of a decrease in the maximal number of cross-bridges was more appropriate to our results, rather than a change in density. Thus, the decline in the triceps surae muscle group P_o could have been directly correlated with the decrease of the muscle fibre diameter and with muscle atrophy.

In addition, the decline in P_o of the whole muscle would suggest that long-term 6° HDT may deleteriously affect one of the steps in excitation-contraction coupling (Duchateau and Hainaut, 1987). Possibilities include alterations in the sarcolemma action potential, the T-tubular charge movement, and/or direct effects on the SR Ca^{2+} release channel. Alternatively, the disuse-induced muscle atrophy may enlarge the extracellular space, such that the tension per whole muscle decreases more than per fibre cross-sectional area (CSA).

The mechanisms responsible for the loss of force with disuse are not well understood, but they cannot include decreases in the CSA of slow- and fast-twitch muscle fibres of the muscle. Muscle atrophy, therefore, probably contributed to the loss of force. Hikida et al. (1989) showed that relative changes in muscle and fibre sizes were less than the relative change in force, and that changes in the ultra-structure may diminish the force output ability of skeletal muscle during and following long-term exposure to microgravity.

The much larger (36 %) reduction in MVC, when compared to the insignificant changes in P_o after a 120-days HDT (24 %), may indicate an inability of the central nervous system to activate the triceps surae muscle group normally. Whether this was due to a lack of motivation on the part of the subjects, or to an involuntary reduction in neural drive, is difficult to distinguish. The subjects certainly appeared well-motivated and had no discomfort or knee stiffness before performing the test, which could have accounted for the low MVC. The increase in force deficiency (Figure 1, top, right) would suggest a decline in central drive in the control of voluntary muscle by the motor nerve system. In fact, during MVC, the

electromyogram activity has been found to be significantly changed by inactivity itself (Duchateau and Hainaut, 1987). Moreover, observation of amplitude changes after inactivity has suggested that fewer motor units were activated in disused muscle (Fuglsang-Frederiksen and Scheel, 1978), and maximal firing frequency of motor unit has been found to be decreased (Duchateau and Hainaut, 1990). It has been thought that a decrease in maximal firing rate could be explained by changes in proprioceptive afferents on the motoneurons (Mayer et al., 1981). This suggests that in future studies, human cognition must be taken for initial physiological states of the muscles that are to be disused to access the extent to which neural and muscle function is affected by loss of voluntary movement.

The rate of rise of evoked contraction in response to electrical stimulation of the nerve with a frequency of 150 impulses·s⁻¹, calculated according to a relative scale, changed very little due to HDT. This observation agrees with the data obtained earlier by Witzmann et al. (1982), who showed that there were no significant changes in the force-velocity characteristics of rat soleus, extensor digitorum longus or superior, and vastus lateralis muscles after 21-days of immobilization, or in human triceps surae muscle group after 120-days of HDT (Koryak, 1995; 1998a), and is consistent with the observed relative constancy of the mechanics of the tetanus and current (cross-bridge) theories of muscle contraction (Ranatunga, 1982). It would, therefore, seem reasonable to conclude that disuse, for example HDT, in women patients has little effect on either cross-bridge cycling or myosin activity (Close, 1972).

Bed rest with prolonged passive muscle "stretching" training

The major goal of the present study was to evaluate if the physiological alterations of the human triceps surae muscle group, induced by HDT conditions, could immobilize the ankle plantaris muscle in a passive stretched position. Our finding showed that HDT conditions induced important decreases in contractile properties of the triceps surae muscle group, in agreement with our earlier observations (Koryak, 1995; 2001; 2003) concerning the effects of voluntary immobilization on the mechanical characteristics of the twitch, and voluntary contractions of the triceps surae muscle group.

It has previously been shown that an acute bout of passive muscle stretching can impede maximal

force production (Avela et al., 1999; 2004; Fowles et al., 2000). In addition, prior stretching can also compromise the performance of a skill for which success is dependent on the rate of force production or power, rather than just the ability to maximize force output (Cornwell et al., 2001; Young, Behm, 2003). Our results show that prolonged passive muscle stretching (~ 5 h/day for 60 days) resulted in a 17 % loss in P_t , a 43 % loss in MVC, and a 19 % loss in P_o . Decreases in MVC were more significant in comparison with earlier results (Avela et al., 1999; Fowles et al., 2000). Thus, it appears that pre-performance stretching exercises might negatively impact skills that require multiple, repetitive high power outputs, in addition to those that depend mainly on maximizing a single output of peak force or power.

The loss in force was partly due to reduced activation and partly due to compromised muscle force-generating capacity. Therefore, it is possible to assume that there was a deficiency of force and damage-related impairment in force transfer from the muscle fibers to the tendon. Damage could have taken place in the portions of the myotendinous junction, a location that has been shown to be susceptible to acute injury because of stress concentration at the ends of the tapered muscle fibers (Garrett et al., 1987). In result, it is possible to expect infringement of communication between of muscle stiffness and compressed answers, and as a result, it will decrease fascia stiffness to a point that could result in reduced force production (Avela et al., 1999; Fowles et al., 2000).

Except for changes in rigidity of a muscle, as a result of long passive stretching, it is possible to assume change, and the connecting fabric caused by damage of a muscle, will be the additional factor causing a decrease of generating of force (Lieber et al., 1991; Armstrong et al., 1993). Creatinekinase enzyme activity is used as a marker of exercise-induced muscle damage, and an increase by 250 % was established after strong and rapid stretching (Ashmore et al., 1988), but showed only a 62 % increase after 17 min of PPS (Smith et al., 1993).

When discussing the neural mechanisms which underlay muscle stretching, changes in afferents on the muscle motoneuron pool are commonly considered (Delwaide, 1973; Robinson et al., 1982; Etnure and Abracham, 1986). A depression of motor pool excitability during a stretch of a muscle has been previously reported (Paillard, 1955; Gottlieb and Agarwal, 1973; Robinson et al., 1982). Robinson et al.

(1982) observed 23-50 % decreased in the soleus muscle H-reflex amplitudes during passive dorsiflexion of the foot. Several neuromuscular feedback responses could contribute to activation failure after PPS, namely, the Golgi tendon reflex, mechanoreceptor (type III afferent) and nociceptor pain feedback (type IV afferent), and/or fatigue responses. The Golgi tendon reflex is an autogenic inhibition that occurs when the Golgi tendon organs, located at myotendon junctions, detect high force combined with muscle lengthening. The Golgi tendon organs' feedback inhibits agonist activation to lower force production and reduce potentially injurious strain on the muscle. However, an extremely intense stretch is necessary to activate Golgi tendon organs (Houk et al., 1971). Moreover, Golgi tendon organ discharge rarely persists during maintained muscle stretch, and the inhibitory effects are momentary (Alter, 1996).

The experimental results indicate that the PPS modality not only avoids the excitatory afferents from muscle spindles (Matthews, 1972), but there is also an inhibition of the motoneuron pool during PS. The inhibition could result directly from inhibitory afferents originating from Golgi tendon organ (Eccles et al., 1957; Houk et al., 1980; Pierrot-Deseilligny and Mazieres, 1984) or from muscle spindle secondary afferents (Hutton et al., 1973; Romano and Schieppati, 1987). It has also been proposed that motoneuron excitability can be indirectly depressed by Ia presynaptic inhibition of Ia afferents (Burke and Ashby, 1972; Delwaide, 1973).

Decreased contractile properties (MVC and P_0) after long-term PPS in HDT conditions maybe connected with discomfort or pain due to a extended stretching of a muscle. Mechanoreceptor (type III afferent) and nociceptor pain feedback (type IV afferent) may reduce central drive (Bigland-Ritchie et al., 1986; Mense and Meyer, 1985). Any perceptions of discomfort or pain were not present during the post-stretch MVCs, so the sensation of stretch and discomfort as a cause of temporary activation failure would have to be short-lived. Some subjects commented that their muscle "just didn't want to contract", despite maximal voluntary effort.

Irrespective of the mechanism responsible for time loss in activation (reduced MVC), indicates that the reduced muscle force-generating capacity was caused by additional factors, as opposed to a decrease in motor activity in HDT conditions (Koryak and Kozlovskaya, 1994). Factors that may affect force-generating abilities after stretch may be due to changes in the length-

tension relationship and/or plastic deformation of connective tissue, which could affect force generation during a maximal contraction. This effect was experimentally confirmed by B-mode ultrasound, which measures muscle fascicle lengths. Kawakami et al. (1995) observed muscle fascicle elongation of 8, 8, and 2 mm for the soleus, lateral gastrocnemius, and medial gastrocnemius muscles, respectively.

Change of characteristics *length-strength*, caused by passive maximal stretching of the triceps surae muscle group, influences a pattern of nervous activity. Fowles et al. (2000) found essential reduction of activity of motor units in electromyograms. Moreover, the stimulation of the nervous system, caused by a passive stretching, depends on the general stretching muscle-tendon complex (Avela et al., 2004).

This experiment simulated an intense maximal stretch far beyond what an athlete may attempt before activity or as part of a flexibility training program. The intensity and duration of stretching required to produce lasting stiffness changes in muscle are unknown (Magnusson et al., 1996). By its viscoelastic nature, muscle has a strong tendency to return to its resting or genetically and biomechanically determined length. It may be questionable to oppose this tendency with the use of intense stretching to enhance performance, when performance can be compromised by altering the fine dynamic balance of neural, architectural, and electrophysiological factors that exists in muscle to create force. Further testing with a stretching protocol more similar to that regularly performed in the athletic context should be evaluated under the controlled conditions of this study.

In summary, PPS impairs both electrically and voluntarily induced muscle contractions. This impairment is mainly related to modification of contractile material behavior. In addition, altered contractile properties seem to affect proprioceptive feedback and, therefore, the motor unit activation in proportion to the contractile failure. Similar changes in muscle parameters related to neural behavior have also been observed during long-lasting stretch-shortening cycle exercise (Nicol et al., 1996; Avela et al., 2001).

Thus, the present research showed that long-term PPS of the ankle plantarflexors, in conditions of reduced mechanical unloading, does not prevent simplification of contractile properties. Decrease in MVC is caused partly by reduction in activity of a muscle, and partly by the ability to generate force.

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