Vertical jump coordination: fatigue effects

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ABSTRACT

RODACKI, A. L. F., N. E. FOWLER, and S. J. BENNETT. Vertical jump coordination: fatigue effects. *Med. Sci. Sports Exerc.*, Vol. 34, No. 1, 2002, pp. 105–116. **Purpose:** The aim of this study was to investigate the segmental coordination of vertical jumps under fatigue of the knee extensor and flexor muscles. **Methods**: Eleven healthy and active subjects performed maximal vertical jumps with and without fatigue, which was imposed by requesting the subjects to extend/flex their knees continuously in a weight machine, until they could not lift a load corresponding to ~50% of their body weight. Knee extensor and flexor isokinetic peak torques were also measured before and after fatigue. Video, ground reaction forces, and electromyographic data were collected simultaneously and used to provide several variables of the jumps. **Results**: Fatiguing the knee flexor muscles did not reduce the height of the jumps or induce changes in the kinematic, kinetic, and electromyographic profiles. Knee extensor fatigue caused the subjects to adjust several variables of the movement, in which the peak joint angular velocity, peak joint net moment, and power around the knee were reduced and occurred earlier in comparison with the nonfatigued jumps. The electromyographic data analyses indicated that the countermovement jumps were performed similarly, i.e., a single strategy was used, irrespective of which muscle group (extensor or flexors) or the changes imposed on the muscle force-generating characteristics (fatigue or nonfatigue). The subjects executed the movements as if they scaled a robust template motor program, which guided the movement execution in all jump conditions. It was speculated that training programs designed to improve jump height performance should avoid severe fatigue levels, which may cause the subjects to learn and adopt a nonoptimal and nonspecific coordination solution. **Conclusion**: It was suggested that the neural input used in the fatigued condition did not constitute an optimal solution and may have played a role in decreasing maximal jump height achievement. **Key Words:** VERTICAL JUMPS, MOVEMENT STRATEGY, MOTOR CONTROL, TRAINING SPECIFICITY

Vertical jumping ability is a crucial skill in the performance of several sports, such as volleyball, basketball, and football. The execution of this motor task depends on the coordination of the segmental actions of the human body, which is determined by the interaction between the muscle forces (ultimately modulated by impulses sent by the central nervous system) and the net moments that have to be generated around the joints to accomplish the mechanical demands of the task. Results of kinematic and electromyographic studies have shown that vertical jumping is performed according to a robust stereotyped pattern (6). It has been shown that the timing, sequence, and amplitude of the muscle activation and joint movements are quite comparable, even when the movement is performed by different subjects (22). It has also been shown that some movement constraints (e.g., constraining the trunk segment (12)) barely disrupt the pattern of variables used to describe coordination (e.g., muscle activation).

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Rodacki et al. (28) studied coordination of vertical jump under fatigue and also suggested the existence of a consistent pattern, irrespective of the force-generating properties of the muscles. Although the activation amplitude of the knee extensor and flexor muscles increased at the end of fatiguing exercises, the pattern of the electromyographic traces remained similar to that observed before fatigue, and the subjects performed the movement as if they scaled a robust muscle activation pattern, which guided the execution of the movement, without considering the best available muscle strength.

It was suggested that during maximal vertical jumps a common drive exists that controls the agonist-antagonist muscle pair activity as a single functional entity (27,37). In that study (27), it was proposed that the modulation of this common drive resulted in similar muscle activation (EMG) between fatigued and nonfatigued jumps, but affected the peak angular velocity and peak power around the joints during the propulsive phase of the movement.

In a vertical jump simulation study, Bobbert and Van Soest (7) demonstrated that although muscle strength determines the maximal jump height achievement, actual performance depends on the control of the muscle properties. In that study, neither increasing the muscle strength of the knee

extensor muscles nor raising the strength of all muscles resulted in jump height improvement, until the muscle activation (control) was reorganized (reoptimized).

Other studies involving cycling (18), running (19), sprinting (26), lifting (31), and continuous hopping exercises (8) provide evidence that some compensatory mechanisms are used to counterbalance the loss of the muscle force-generating properties because of fatigue. This is in agreement with the arguments proposed by Van Ingen Schenau et al. (33) that changes in muscle activation timing should be accomplished to avoid deterioration of the performance when the properties of the musculoskeletal system are changed. Hence, under fatigue, defined as the inability of the neuromuscular system to sustain the required or expected power output around a joint (11), compensatory strategies may induce a reorganization of the movement structure and a new coordination pattern may appear.

Experimentally, it is not known whether or how the neuromuscular system reorganizes the pattern of maximal countermovement jumps when a particular muscle group (e.g., knee extensor or knee flexor muscles) has its force-generating properties changed (increased or decreased). Changing the force-generating properties of one component of the agonist-antagonist pair is an attractive way to test whether a common drive exists and how it influences movement organization during dynamic conditions. If a common drive guides the movement execution of maximal countermovement jumps, a unique stimulation signal would be used and muscle activation timing may be consistent, and other variables (e.g., peak angular velocity, peak net moment, and the peak net power around the joints) may vary when one component of the agonist-antagonist pair is fatigued (i.e., the muscle force-generating properties are reduced). For instance, left- or rightward phase shifts in the timing of other variables of the movement (e.g., peak angular velocity time) would be expected to occur under fatigue, whereas the muscle activation (EMG) would remain without large variations.

It is possible that a decline in performance after fatigue may be the result of (a) a change in coordination (i.e., changing the neural input), (b) a change in the functional capacity of the muscles to produce force (i.e., without changing the neural input), or (c) the combination of these two factors. In the first case, changes in both muscle activation and kinematics are likely to occur, whereas the second case may be characterized by a stable neural input (EMG), in which a different kinematic output may emerge. In other words, does the neuromuscular system adopt a new coordination pattern to account for local muscular fatigue or does it do the same thing as suffer the consequences of reduced muscular force? The aim of the present study was to investigate whether and to what extent the neuromuscular system (re)organizes and accommodates the controls used in multisegment movements when different muscle groups are fatigued. It has been hypothesized that fatigue will decrease performance and will influence the magnitude and the time of several kinematic and kinetic variables (as speculated above), but will have little effect on muscle activation pattern (EMG). Studies analyzing how the neuromuscular system adjusts the movement coordination pattern used during vertical jumps under fatigue may provide valuable information to understand motor control of multisegment movements.

METHODS

Eleven healthy male subjects (age, 23.1 ± 4.8 yr; height, 183.4 ± 6.1 cm; and body mass, 84.0 ± 13.2 kg) engaged in various sports (six volleyball players, three rugby players, and two multiple sports) and with previous experience in vertical jumping were informed of the procedures involved in this study and gave their informed consent to act as subjects, in accordance with the ACSM policy statement regarding the use of human subjects, informed consent, and approval by an ethics committee.

Experimental procedures. Subjects reported to the laboratory for two test sessions, which were separated by at least 3 d. The knee extensor muscle group was fatigued in the first session, whereas the flexor muscle group was fatigued in the second session. Fatigue was imposed by requesting the subjects to extend and flex both their knees in a knee flexor/extensor weight machine (PowerSport®, Mid Glamorgan, United Kingdom) using a self-selected pace, until they could no longer lift a load. The load used for each subject corresponded to ~50% (knee extensors) and 40% (knee flexors) of their body mass. The knee extensors were fatigued in an upright sitting posture, whereas the knee flexor muscles were fatigued in a prone posture (biceps curl). In both exercise modes, the subjects were allowed to stabilize themselves by holding on either side of the seat, and verbal encouragement was given. On average, the subjects were not able to continue the knee extension and flexion movements after 27 ± 9 and 18 ± 8 repetitions, respectively.

Isokinetic strength measurements. The isokinetic peak torques of the right knee extensor (PT_{EXT}) and flexor (PT_{FLEX}) muscles were measured using a Cybex® dynamometer (Cybex International, Medway, MA) in each session. In both sessions, before the commencement of the isokinetic assessment, the subjects were also allowed to perform a set of five submaximal contractions in the isokinetic dynamometer. After finishing the warm-up on the isokinetic dynamometer, a 3-min rest interval was imposed before the subjects performed a maximal test of five maximal voluntary repetitions to represent the nonfatigued condition. The isokinetic measurement in the fatigued condition took place at the end of each session, immediately after the countermovement jumps assessment in the fatigued condition (see below). The interval between the end of the countermovement jumps and the initiation of the isokinetic test in the fatigued condition (60–90 s) was caused by the necessity to walk the subjects through a distance of 15 m and to set the isokinetic dynamometer.

During the isokinetic measurements, the subjects were seated in an upright posture (90 degrees between the trunk and the thigh) and secured across the pelvis and trunk by a four-point belt. Peak torque was defined as the highest torque achieved during five successive (without pause)

maximal concentric knee extension (from 90 degrees of knee flexion to 180 degrees of knee extension) and flexion (from 180 degrees of knee extension to 90 degrees of knee flexion) movements. The hamstrings to quadriceps torque ratio (H/Q) was defined as the quotient between PT_{FLEX} and PT_{EXT}. The range of motion and the movement velocity (60 $deg \cdot s^{-1}$) of the concentric contractions were preset by the experimenter. This movement velocity was selected to represent the muscle force-generating properties. Gravity correction was performed according to manufacturer's guidelines. Verbal encouragement and visual feedback of the torque traces were given during the testing.

Vertical jumps. After the first isokinetic testing, the subjects were prepared for the electromyographic and kinematic assessments (see below), received explanation and demonstration of the countermovement jumps, and performed three warm-up trials, which were followed by a set of three maximal countermovement jumps. During the countermovement jumps, subjects bent their knees to a freely chosen angle, which was followed by a maximal vertical thrust. The effect of the arms was minimized by requesting the subjects to keep their hands and arms crossed against the chest. Kinematic, force, and electromyographic data were recorded simultaneously for three maximal jumps, but only the jump that resulted in the greatest flight time was further analyzed to represent the subject's best performance in the nonfatigued condition (CMJ_1). Force data were used to determine the flight time of each jump in all conditions.

The second set of three maximal countermovement jumps took place immediately after the end of the fatiguing exercises and dismounting the knee extensor/flexor weight machine, which was positioned close to the force platform—approximately 3 m. The interval between the end of the fatiguing exercises and the countermovement jumps in the fatigued condition was kept as short as possible (< 10 s). The subjects were instructed to follow the same procedures used in CMJ_1. Again, three maximal countermovement jumps with simultaneous kinematic, force, and EMG data were recorded and the best performance jump selected to represent fatigued conditions ($CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$, respectively).

Kinematic assessment. Reflective marks were placed on the right side of the subject's body to match with the following sites: 1) fifth metatarsal joint, 2) lateral malleolus, 3) lateral femoral epicondyle of the knee, 4) the most prominent protuberance of the greater trochanter, and 5) neck at the level of the fifth cervical vertebrae. The subjects were filmed (100 Hz) using a two-dimensional kinematic optoelectric system (ELITE, BTS, Milan, Italy) and the coordinates of the marker points were filtered using spline functions. These filtered body markers defined the position of the foot (FOT), shank (SHA), thigh (THI), and upper body (TRU), and were used to calculate joint angular displacement, velocity, and acceleration of the ankle (ANK), knee (KNE), and hip (HIP). Figure 1 provides visual information of the four-segment model and also shows the joint angle conventions.

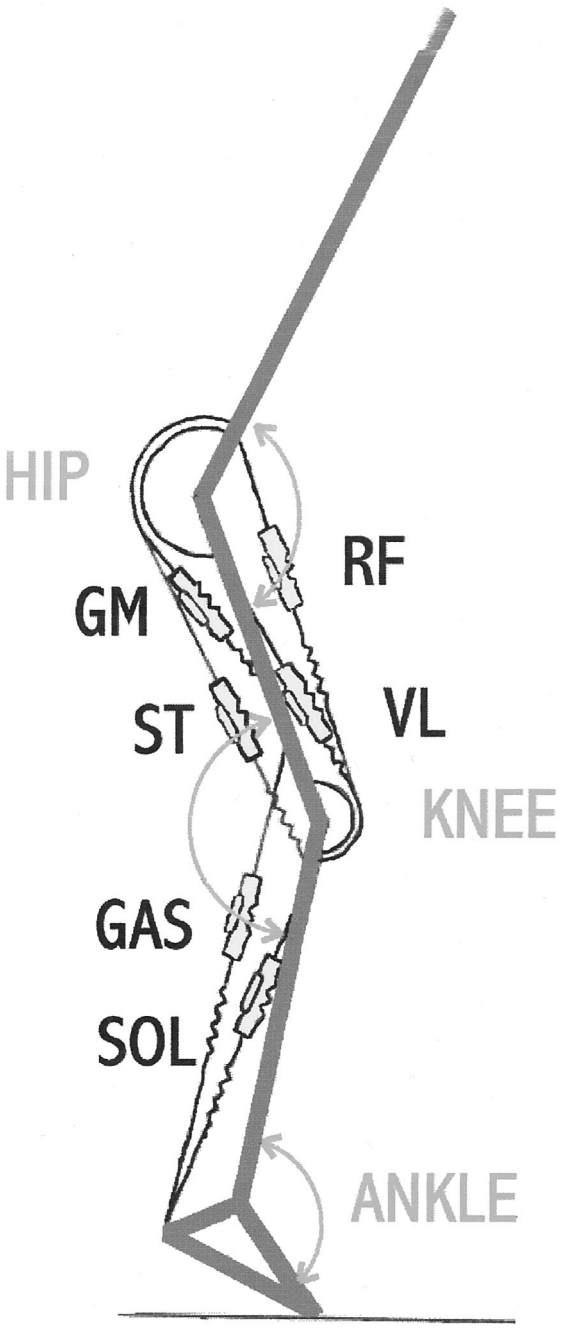

FIGURE 1—The four-body segment model and the joint angle convention. The muscles soleus (SOL), gastrocnemius (GAS), semitendinosus (ST), vastus lateralis (VL), rectus femoris (RF), and gluteus maximum (GM) are indicated.

Electromyographic assessment. Surface electromyographic signals were recorded from gastrocnemius lateralis (GAS) (over the area of the greatest muscle bulk on the lateral calf), soleus (SOL) (over the lateral edge, where the muscle protrudes below the GAS), vastus lateralis (VL) (over the area of the greatest muscle bulk just lateral to the rectus femoris on the distal half of the thigh), rectus femoris (RF) (over the midpoint between the anterior superior iliac spine and the patella superior border), semitendinosus (ST) (midway on a line between the ischial tuberosity and the medial epicondyle of the tibia), and gluteus maximus (GLU)

(over the bulkiest part of the middle of the muscle belly). The electrode placement sites followed the recommendation of Acierno et al. (1).

Electromyographic signals were obtained using disposable bipolar surface electrodes (Bio-tabs MSB® (MIE Medical Research Ltd., Leeds, UK) Ag/AgCl, with leadoff area 2.75 cm^2), placed with center-to-center distance of 1.5 cm and border-to-border distance of 1.0 cm. Reference electrodes (3M Red Dot®, type 2237 (3M Company, St. Paul, MN), Ag/AgCl with foam tape and solid gel) were used at the most distant point possible away from the electrode sites (approximately 10–15 cm), toward the most distal point of the segments. Because of the fast and explosive characteristic of the movement, all sites were covered with straps of adhesive tape to prevent disconnection and reduce movement artifacts. All test sites were identified and prepared by the same experimenter.

The electromyographic signals were preamplified in subminiature amplifiers before transmission via FM radio telemetry (459 MHz, with channel bandwidth of 1000 Hz) to a recording device no farther than 3 m away. The miniature preamplifiers (33 × 21 × 9 mm) provided a gain of 1000, bandwidth of 15 kHz, noise of less than −52 dB, common mode rejection ratio of −102 dB, and input impedance greater than 10^8 Ω. The raw electromyograms were processed into a linear envelope (EEMG). The EEMGs were calculated using the MYO-DAT® 5.0 EMG analysis package software (MIE Medical Research Ltd., Leeds, UK) by applying a second-order low-pass filter set at 6 Hz frequency. Electromyographic data were sampled at 200 Hz.

Because of technical limitations, it was not possible to record the electromyographic signals using a sampling frequency higher than 200 Hz. Therefore, the relatively low sampling frequency used in this study (200 Hz) may not have allowed the high-frequency components to be recorded adequately, which may constitute a violation of the sampling theorem. In order to assess the impact of using such resolution, we performed an assessment in which the electromyographic sampling frequency was set at 800 Hz. These data were used to generate a second data set, in which the sampling frequency was reduced to 200 Hz. Then, both data sets (800 and 200 Hz) were processed (rectified, filtered, and normalized with respect to magnitude). The average root mean square difference was 6.0 ± 0.5% (GAS, 5.9; VM, 6.6; VL, 5.5). The differences in the detection of initiation (ON) and peak (PK) instants between the two data series were small (ON, 4 to 7 ms; PK, 5 to 8 ms, respectively). Therefore, and despite constituting a certain limitation, the EMG data collected at 200 Hz can be used to represent the muscle activation pattern without obscuring relevant aspects of the biological significance of the movement coordination analyzed in this study.

Kinetic assessment. A force platform (Kistler®, model 9281B, Kistler Instruments, Winterthur, Switzerland) synchronized with the kinematic and electromyographic measurements and sampling at 1000 Hz provided force-time traces. The kinematic analysis was combined with the ground reaction forces to calculate net moments at the ankle, knee, and hip joints. The moment of inertia of each segment was estimated by using the Drillis and Contini (10) equations. Net powers around the joints were also calculated by multiplication of the net moments and joint angular velocities. The net impulse was calculated by integrating the force-time curves of the vertical component of the ground reaction forces during the positive phase of the movement (see below). Extension moments were considered positive at all joints. All kinetic data were normalized with respect to body weight (BW).

Definition of variables and data analysis. The times at which initiation of extension (IEX) and peak angular velocity (PAV) of the ankle (ANK), knee (KNE), and hip (HIP) joints were determined. IEX was defined as the first instant (frame) after a joint reaches its deepest flexion angle, whereas PAV was defined as the instant at which the greatest joint angular velocity is achieved during the propulsive phase of the movement. The difference in time at which IEX of each joint occurred was used to determine the relative timing and the sequential relationship between adjacent segments (28). In other words, if the movement of a proximal segment precedes the movement of its distal counterpart (i.e., a proximal-to-distal order), the difference between the IEX of these joints would be negative. Because the IEX was determined kinematically (from the joint angle position data), it is estimated that a mean error of 5 ms (ranging from 0 to 9 ms) may have occurred.

In order to analyze the movement sequence and temporal organization, the first and the last data points corresponding, respectively, to the beginning and take-off instants were used to define the movement duration, i.e., the contact time phase (CT) duration. The CT was fractioned in three phases: the negative phase (NEG), the transient phase (TR), and the positive phase (POS). The initiation of the NEG phase was calculated using the ground reaction force traces and was determined as the instant before the vertical force decreases continuously for a period longer than 0.02 s (two frames). The end of the NEG and the beginning of the POS phases was defined as the first instant in which the vertical velocity of the body mass center is positive. The vertical velocity body mass center was calculated by integrating the net vertical ground force data (2). The end of the POS phase was determined with the help of the force data and was defined as the instant in which the subjects lost contact with the force platform, i.e., the instant in which the vertical component of the ground reaction forces was zero—the take-off instant (TO). At the end of the NEG phase and at the beginning of the POS phase, there is a phase of transition (TR) in which there are no large changes in the knee joint angular velocity. For analysis purposes, the TR phase was determined as the period in which the knee angular velocity ranged between $+30\ deg \cdot s^{-1}$ and $-30\ deg \cdot s^{-1}$ in relation to the deepest knee flexion angle (IEX) (28).

To better compare the characteristics of the electromyographic signals, the EEMG traces were normalized with respect to the signal magnitude. For each muscle, the highest electromyographic signal value obtained during the performance of each trial was used as reference and set at 100%.

In the next sections, the processed electromyographic signals (rectified, filtered, and normalized with respect to magnitude) are referred to as EMG. The ON and PK activation of each muscle were also examined. ON was arbitrarily considered as the first instant in which the EMG traces were equal to or greater than 20% of PK, which was defined as the highest muscle activation obtained during the POS phase. The use of different criteria to determine the instant in which muscle activation was initiated (ON) may produce different results. Cross-correlation analysis (bidirectional phase shift of 15 lags; 75 ms) were applied on the EMG traces between CMJ_1 and the fatigued conditions ($CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$) to account for phase shifts between these signals.

Muscle activation was quantified during the POS phase of the movement by dividing the integrated EMG signal (the area under the muscle activation curve) by the duration of the movement phase. Changes in muscle activation were expressed as a mean percentage difference of the initial values (CMJ_1). Therefore, a positive value indicates greater muscle activity in the fatigued condition than in the nonfatigued condition, and a negative value indicates the opposite.

Knee joint stiffness was determined by calculating the coefficient of linear regression of the moment-angle relationship from the last 15 degrees of the NEG phase to the deepest excursion of the knee joint. This analysis only considered the final 15 degrees of the NEG phase because no differences were found before this point.

A *t*-test was performed to compare several variables used to describe coordination in the nonfatigued condition (CMJ_1) between the jumps performed in the first and second sessions. Because no significant differences were found ($P < 0.05$), all variables representing this condition were collapsed, and an ensemble average was used to represent the nonfatigued condition (CMJ_1). To compare the kinematic, kinetic, electromyographic, and joint stiffness data between conditions, a number of one-way ANOVAs with repeated measures were applied. When significant differences were detected, a Newman-Keuls *post hoc* test was applied. Effect size (ES) was also calculated. A Kolgomorov-Smirnov test was applied and confirmed data normality. All statistical analyses were performed in the Statistica® package software, version 5.5 (StatSoft, Inc., Tulsa, OK) and the significance level was set at $P < 0.05$.

RESULTS

Isokinetic peak torques. No significant differences in peak torque in the nonfatigued condition (PT_{EXT} and PT_{FLEX}) were found between the first and second two sessions (0.29 and 0.18 $Nm \cdot BW^{-1}$, respectively; $P > 0.05$). The fatigue protocol used in this study was proven to successfully reduce the peak torque of the knee extensor ($PT_{EXT} = 0.29 \pm 0.03\ Nm \cdot BW^{-1}$) and flexor ($PT_{FLEX} = 0.17 \pm 0.02\ Nm \cdot BW^{-1}$) muscles by 14.2% and 12.6%, respectively ($P < 0.05$; ES = 1.43 and 1.2, respectively). When these muscle groups acted as antagonists, the extensor and flexor peak torques remained unchanged (1.4% and 1.1%, respectively; $P > 0.05$) in comparison to the initial condition (ISO_1). The H/Q found in ISO_1 (0.61 ± 0.05) increased by 15.2 ± 6.1% ($P < 0.05$; ES = 1.51) after fatiguing the knee extensor muscles, and decreased by 11.4 ± 7.6% ($P < 0.05$; ES = 1.41) after fatiguing the knee flexor muscles.

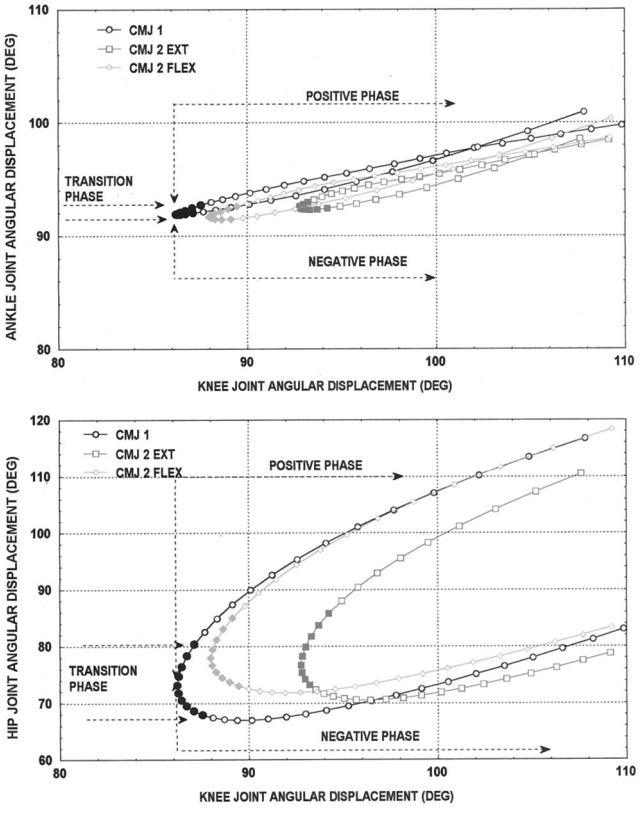

FIGURE 2—Angle-angle representation of the ankle-knee (top) and knee-hip (bottom) joint angular displacement before (CMJ_1) and after fatiguing the knee extensor ($CMJ_{2\ EXT}$) and flexor ($CMJ_{2\ FLEX}$) muscles.

Vertical jump performance. In the nonfatigued condition there was no significant difference ($P > 0.05$) between the jumps performed in the first and second sessions (0.33 ± 0.06 m and 0.33 ± 0.07 m, respectively). Fatigue reduced the ability of the subjects to jump as high as in the nonfatigued condition only in $CMJ_{2\ EXT}$. On average, the subjects were able to jump 86.1 ± 7.0% ($P < 0.05$; ES = 0.74) and 93.8 ± 4.0% ($P > 0.05$; ES = 0.16) of the maximal height jumped in CMJ_1 (0.33 ± 0.06 m) during $CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$, respectively.

Kinematics and kinetics. Fatigue did not change the total duration of the countermovement jumps between CMJ_1 and the fatigued conditions ($CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$) ($P > 0.05$). In addition, the duration of the NEG, TR, and POS phases of the movement remained unaltered ($P > 0.05$; ES < 0.22) in comparison with CMJ_1, irrespective of which muscle group was fatigued ($CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$).

The magnitude and the time of the kinematic and kinetic variables analyzed during the NEG phase of the movement (Figs. 2 and 3) were similar in both fatigue conditions ($CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$) and did not differ significantly ($P > 0.05$). In $CMJ_{2\ EXT}$, the knee joint was less flexed

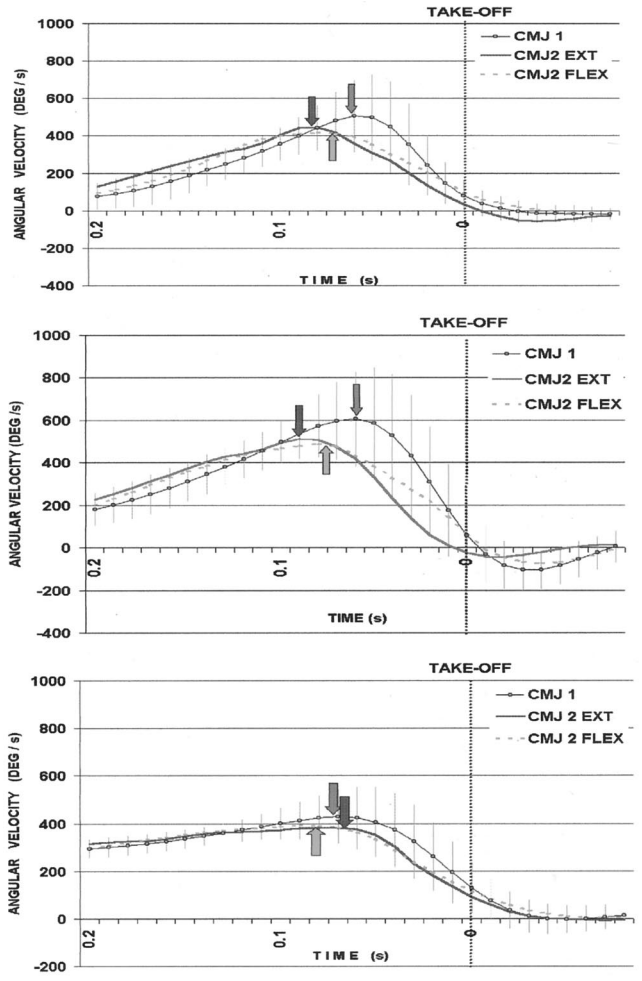

FIGURE 3—Ensemble average of the ankle (top), knee (middle), and hip (bottom) angular velocity before (CMJ_1) and after fatiguing the knee extensor ($CMJ_{2\ EXT}$) and flexor ($CMJ_{2\ FLEX}$) muscles. The standard deviation (± 1 SD) of CMJ_1 is represented. The take-off instant is indicated by the *dotted vertical line*, and the *arrows* highlight the peak angular velocity time.

during its deepest position ($P < 0.05$; ES = 0.78) and the excursion of the body mass center was shallower ($P < 0.05$; ES = 0.74) than in CMJ_1. The angular displacement of the ankle, knee, and hip joints across the experimental conditions are shown in Figure 2.

The delays between IEX_{HIP}–IEX_{KNE} and IEX_{KNE}–IEX_{ANK} indicated the existence of a proximal-to-distal order, in which the hip was consistently the first joint to extend and was followed by knee and ankle joint extensions (Table 1), irrespective of the fatigue condition. The differences between IEX_{KNE} and IEX_{ANK} revealed a variable pattern where, in some cases, ankle extension preceded knee extension. The statistical analyses of these delays suggest that the proximal-to-distal order was not influenced by muscle group fatigue ($P > 0.05$; ES = 0.23).

Figure 3 shows the joint angular velocity of the ankle, knee, and hip. Peak positive joint angular velocity was reduced at the knee and hip joints ($P < 0.05$; ES = 0.50), when the knee extensor muscles were exercised ($CMJ_{2\ EXT}$). The peak knee positive angular velocity occurred earlier ($P < 0.05$; ES = 1.31) in $CMJ_{2\ EXT}$ than in CMJ_1, and no significant differences ($P > 0.05$) were found in other joints. Fatiguing the knee flexor muscles ($CMJ_{2\ FLEX}$) did not change the ankle, knee, or hip peak joint positive angular velocity magnitude or time ($P > 0.05$).

Fatiguing neither the knee extensor ($CMJ_{2\ EXT}$) nor the flexor muscles ($CMJ_{2\ FLEX}$) decreased the net peak power, in comparison with the nonfatigued condition (CMJ_1) ($P > 0.05$; ES = 0.34 and 0.36, respectively). The net peak power of the ankle and knee joints occurred 25.7% ($P < 0.05$; ES = 0.90) and 18.9% ($P < 0.05$; ES = 1.04) earlier in $CMJ_{2\ EXT}$ than in CMJ_1. No significant changes in the temporal characteristics of the net peak power around the hip joint were detected ($P > 0.05$; ES = 0.35) between CMJ_1 and $CMJ_{2\ FLEX}$. The net impulse determined during the positive phase in CMJ_1 was reduced by 8.5% in $CMJ_{2\ EXT}$ ($P < 0.05$; ES = 0.38), whereas no significant changes were found in $CMJ_{2\ FLEX}$ ($P > 0.05$; ES = 0.15).

Figure 4 shows the average stiffness of the knee joint. Knee joint stiffness was calculated during the final 15 degrees of knee flexion, at the end of the NEG phase. Fatiguing the knee flexor muscles ($CMJ_{2\ FLEX}$) did not change the stiffness of the knee, which was similar to that observed in CMJ_1 ($P > 0.05$; ES = 0.46). On the other hand, knee joint stiffness increased ($P < 0.05$; ES = 1.2) during the final part of the NEG phase of the movement in $CMJ_{2\ EXT}$.

Electromyographic analyses. Muscle activation of the knee extensor muscles (VL and RF) increased by 39.0% ($P < 0.05$; ES = 1.4) in $CMJ_{2\ EXT}$ and by 18.8% ($P < 0.05$; ES = 1.2) in $CMJ_{2\ FLEX}$ in relation to CMJ_1 during the POS phase of the movement. Muscle activation of the biarticular flexor muscles of the knee (ST) increased by 29.6% ($P < 0.05$; ES = 1.0) in $CMJ_{2\ EXT}$ and increased by 25.4% ($P < 0.05$; ES = 1.2) in $CMJ_{2\ FLEX}$ in comparison with the control condition. The activation of the SOL, GAS, and GM muscles in $CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$ remained without large variations and did not differ significantly ($P > 0.05$; ES = 0.3) in relation to CMJ_1. ON and PK did not differ significantly ($P > 0.05$; ES = 0.2) in comparison with CMJ_1, in all muscles and fatigue conditions ($CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$) (Figs. 5 and 6). Cross-correlation analysis (Table 2) showed that the highest correlation occurred at approximately zero phase lag, indicating that no significant phase shifts occurred between the experimental ($CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$) and the control (CMJ_1) conditions.

DISCUSSION

Isokinetic peak torques and H/Q ratio. The equivalent isokinetic peak torque of the knee extensor and flexor muscle groups between the first and second sessions in the nonfatigued condition suggested a good reproducibility of the measurements when the performers were tested on different days. The H/Q ratio of approximately 0.6 found in the nonfatigued condition is in agreement with other studies using low-speed testing (13). The protocol used to fatigue the knee extensor/flexor muscles was proven as a successful way to manipulate (increase or decrease) the H/Q ratio.

TABLE 1. Kinematic and kinetic characteristics of countermovement jumps before (CMJ_1) and after fatiguing the knee extensor (CMJ_2 EXT) and flexor (CMJ_2 FLEX) muscles.[a]

	CMJ_1	CMJ_2 EXT	Variation (%)	CMJ_2 FLEX	Variation (%)
NEG duration (ms)	615 ± 82	605 ± 132	–1.6	623 ± 123	+1.3
TR duration (ms)	47 ± 9	43 ± 6	–8.5	48 ± 8	+2.1
POS duration (ms)	319 ± 47	325 ± 50	+1.8	328 ± 76	+2.8
Displacement, BMC (% standing still)	68.8 ± 10.9	60.7 ± 11.6[b]	–11.7	67.3 ± 132	–2.1
Impulse ($N \cdot s^{-1} \cdot BW^{-1}$)	300.7 ± 69	279.0 ± 72.4[b]	–7.2	289.7 ± 67.5	–3.6
Knee stiffness ($Nm \cdot BW^{-1} \cdot deg^{-1} \cdot 10^{-3}$)	14.4 ± 5.8	21.4 ± 13.1[b]	+48.6	17.1 ± 7.6	+18.7
Ankle					
IEX joint angle (deg)	90.6 ± 5.3	91.3 ± 6.6	+0.7	89.9 ± 4.7	–0.7
IEX time (ms)	268 ± 42	284 ± 70	+5.9	281 ± 66	+4.8
PAV, POS ($deg \cdot s^{-1}$)	594 ± 141	587.3 ± 120	–1.1	584.2 ± 125	–1.7
PAV time, POS (ms)	67 ± 20	90 ± 36[b]	+34.3	73 ± 31	+12.0
Peak power, POS ($W \cdot BW^{-1}$)	2.01 ± 0.58	1.89 ± 0.46	–5.9	1.95 ± 0.38	–2.9
Time to peak power (ms)	70 ± 20	88 ± 28[b]	+25.7	77 ± 28	+10.0
Knee					
IEX joint angle (deg)	89.5 ± 12.4	91.5 ± 10.4[b]	+2.2	85.2 ± 10.3	–4.8
IEX time (ms)	313 ± 49	321 ± 55	+2.5	324 ± 83	+3.5
PAV, POS ($deg \cdot s^{-1}$)	700.6 ± 119	640.3 ± 106[b]	+8.6	683.3 ± 121	–2.5
PAV time, POS (ms)	66 ± 19	91 ± 34[b]	–37.8	77 ± 31	+16.6
Peak power, POS ($W \cdot BW^{-1}$)	1.65 ± 0.59	1.52 ± 0.64	–7.8	1.61 ± 0.68	–2.4
Time to peak power (ms)	116 ± 21	138 ± 35[b]	+18.9	123 ± 24	+6.0
Hip					
IEX joint angle (deg)	65.4 ± 12.7	69.9 ± 15.7	+5.5	70.0 ± 13.8	+7.0
IEX time (ms)	387 ± 35	392 ± 46[b]	+1.3	397 ± 59	+2.5
PAV, POS ($deg \cdot s^{-1}$)	472.7 ± 61	437.1 ± 67[b]	–7.5	446.3 ± 63	–5.5
PAV time, POS (ms)	74 ± 21	69 ± 41	–6.7	84 ± 30	+13.5
Peak power, POS ($W \cdot BW^{-1}$)	1.42 ± 0.44	1.27 ± 0.52	–10.5	1.26 ± 0.42	–11.2
Time to peak power (ms)	204 ± 61	202 ± 71	–1.0	209 ± 66	+2.4
Delay between IEX_{HIP}–IEX_{KNE} (ms)	74 ± 13	71 ± 11	–4.0	73 ± 14	–1.3
Delay between IEX_{KNE}–IEX_{ANK} (ms)	45 ± 40	37 ± 67	–17.7	43 ± 68	–4.4

CT, contact time; NEG, negative phase; POS, positive phase; BMC, body mass center; IEX joint angle, initiation of the joint extension; IEX time, time to initiation of the joint extension; PAV, peak angular velocity; PAV time, time to peak angular velocity.

[a] The percentage of variation is expressed in relation to the changes in the fatigued condition (CMJ_2) in relation to the initial condition (CMJ_1). The time of the peak expressed in relation to take-off instant, which was set as zero.

[b] Significant differences between experimental conditions ($P < 0.05$).

Although the decrease in the peak torque of the knee extensor and flexor muscles (14.2 and 12.6%, respectively) may not be considered as "large," these significant declines were interpreted as satisfactory, since changes imposed after strengthening training program (viewed as changes in the opposite direction, i.e., gains in peak torque) rarely surpass these values. It can be speculated that the changes in H/Q at the time of performing maximal countermovement jumps in the fatigued condition were even greater than that revealed by the isokinetic peak torque testing because of the relatively large interval (60 to 90 s) allowed between the countermovement jumps and the isokinetic strength assessments. The discussion and the results of this study are derived from the fatigue level achieved in this experiment, and the reader should bear in mind that different fatigue protocols and other fatigue levels may invoke different responses.

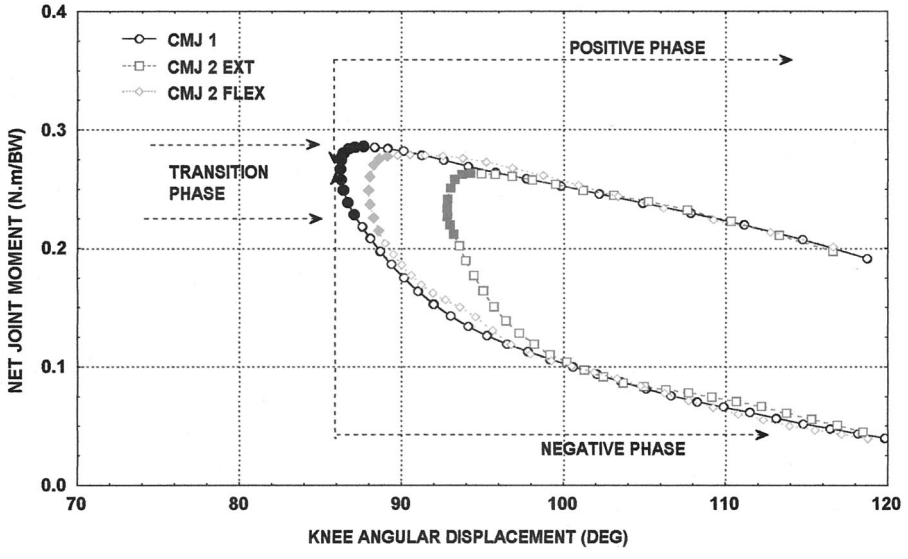

FIGURE 4—The knee joint stiffness in nonfatigued (CMJ_1) and fatigued (CMJ_2 EXT and CMJ_2 FLEX) conditions. The negative (NEG), transient (TR), and positive (POS) phases are indicated.

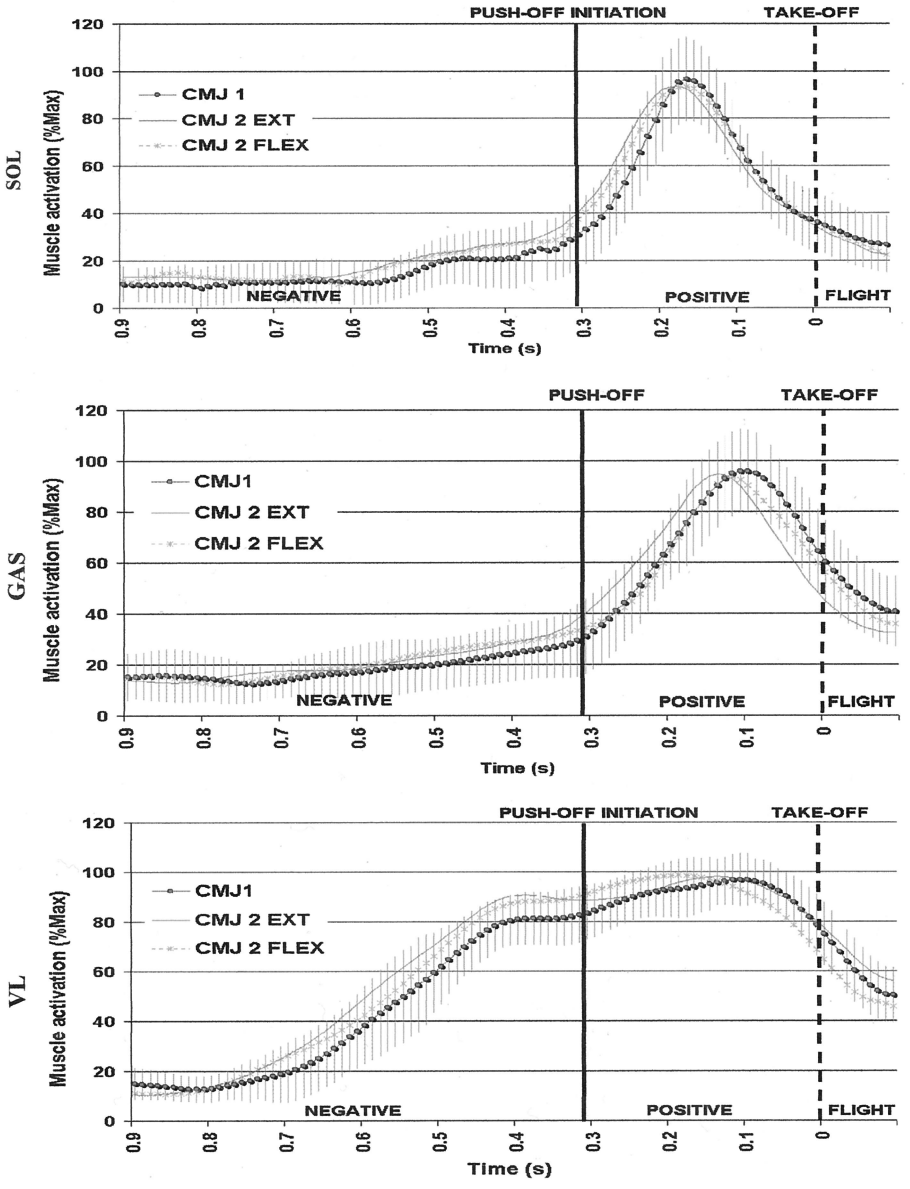

FIGURE 5—The electromyographic signals from soleus (SOL), gastrocnemius (GAS), and vastus lateralis (VL). The take-off instant is indicated. The initiation of the push-off phase of the nonfatigued condition (CMJ_1) is represented. The muscle activation was normalized with respect to the maximal activation during the movement in each jump condition. The standard deviation of the countermovement jumps performed in the CMJ_1 is represented. $CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$ are the knee extensor and flexor muscles in the fatigued condition, respectively.

Vertical jump performance. Vertical jump height achievement in the CMJ_1 (0.33 m) was comparable to other studies, in which subjects jumped using an equivalent technique (e.g., Hortobágyi et al. (20)). The pronounced decrease in the maximal countermovement jump performance that occurred by fatiguing the knee extensor muscles suggests that this group possesses greater importance than its antagonist counterpart. This is not surprising if one takes into account that the positive work done by the knee extensors (49% of the net amount of work done) is much greater than that reported around the ankle (23%) and hip joints (28%) (21). Additionally, some studies (23,27) have estimated that the knee flexor muscle group exerts a moment of force that accounts for 11 to 17% of the resultant joint moment, which is substantially less than that generated by the knee extensors (the main force generators).

Kinematics and kinetics. Fatiguing the knee flexor muscles did not change significantly the kinematic or kinetic variables at any joint level, during either the negative or positive phases of the movement. Despite most flexor muscles of the knee being biarticular (hamstrings), and assuming that fatigue affected the ability of these muscles to generate torque at both joints they span, the initiation of the trunk segment extension remained relatively unaltered. In part, this emphasizes the arguments that the gluteus maximus, which was not fatigued in this study, is the strongest hip joint extensor and performs most work necessary to extend the hip joint, while the hamstrings "make negligible contributions to the joint angular accelerations" (25).

Fatiguing the knee flexor muscles did not produce significant changes at the knee joint level in any variables assessed in this study (see Figs. 2 and 3). It is apparent that the level of fatigue imposed on the knee flexor muscles ($\geq$ 12.6% decrease in PT_{FLEX}) was not adequate to compel the neuromuscular system to reorganize the segmental motion. It is possible that after "fatiguing," these biarticular muscles were still able to exert a large enough flexor moment about the knee to help reduce the extensor moment at this joint to

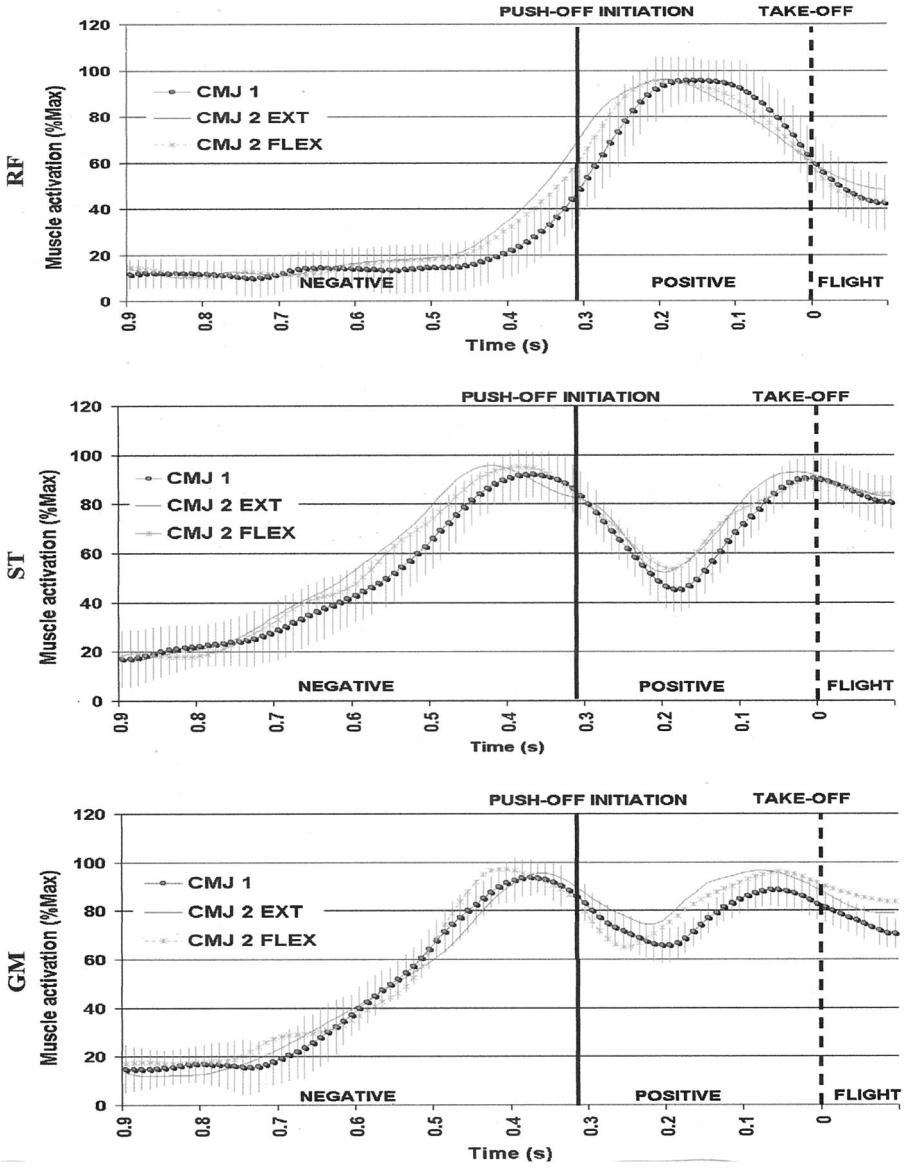

FIGURE 6—The electromyographic signals from rectus femoris (RF), semitendinosus (ST), and gluteus maximum (GM). The take-off instant is indicated. The initiation of the push-off phase of the nonfatigued condition (CMJ_1) is represented. The muscle activation was normalized with respect to the maximal activation during the movement in each jump condition. The standard deviation of the countermovement jumps performed in the CMJ_1 is represented. $CMJ_{2\ EXT}$ and $CMJ_{2\ FLEX}$ are the knee extensor and flexor muscles in the fatigued condition, respectively.

delay the initiation of joint extension. Therefore, it can be speculated that the hamstrings muscles were still able to play a role (fine-tune regulation) at the knee joint level under the fatigue level used in this study.

Fatiguing the knee extensor muscles caused the subjects to adjust several kinematic and kinetic variables of the movement, which included a reduced joint angular displacement of the knee joint, a decreased knee and hip peak joint angular velocity, and an increased knee joint stiffness. In addition, peak knee joint angular velocity and peak knee power during the positive phase of the movement occurred earlier when the knee extensor muscles were fatigued than in the control condition.

Increased joint stiffness in stretch-shortening cycle has been considered as an efficient way to potentiate the positive phase of countermovement jumps (15). In this study, the increased stiffness detected at the end of the negative phase may have contributed to the smaller knee joint angular displacement and to keep a short coupling time between the eccentric and the concentric phases of the movement (9). The potentiation that occurs after fatigue, i.e., increased reuse of the stored elastic energy and reflex sensitivity that accompany increased joint stiffness (15), may explain the similar duration of the movement phases between experimental and control countermovement jumps (36). This may also account for the similar temporal structure of movement, which remained relatively unaltered after fatiguing the knee extensor muscles.

Rather than influencing the early stages of the movement (NEG phase), where most variables did not change with fatigue (see Figs. 2 and 3), the decline in the ability of the extensor muscles to generate force was more reflected during the final part of the movement, as the take-off approached. This is in agreement with previous studies (24,32), in which concentric contractions have been reported as more affected by the loss of the force-generating properties of the contractile components than eccentric contractions. Since each step in the chain of the events for muscle contraction could be a site for fatigue, it is difficult to determine the exact mechanisms that account for the

TABLE 2. Average (± SD) changes in muscle activation amplitude, phase shift, and the cross-correlation values of six muscles assessed before (CMJ_1) and after fatiguing the knee extensor (CMJ_2 EXT) and flexor (CMJ_2 FLEX) muscles.[a]

	CMJ_2 EXT			CMJ_2 FLEX		
Muscle	Muscle Activation (% CMJ_1)	Phase Shift (ms)	Cross-Correlation	Muscle Activation (% CMJ_1)	Phase Shift (ms)	Cross-Correlation
SOL	4.1 ± 3.3	5 ± 1.9	0.94 ± 0.03	3.5 ± 3.1	2 ± 0.7	0.97 ± 0.02
GAS	6.1 ± 4.0	12 ± 1.6	0.95 ± 0.04	5.2 ± 4.7	6 ± 1.7	0.96 ± 0.04
VL	39.2 ± 9.5[b]	13 ± 1.7	0.96 ± 0.02	18.5 ± 5.0[b]	14 ± 1.6	0.98 ± 0.03
RF	39.0 ± 6.6[b]	15 ± 2.9	0.93 ± 0.04	19.3 ± 6.3[b]	14 ± 2.0	0.93 ± 0.05
ST	29.5 ± 5.1[b]	10 ± 1.8	0.93 ± 0.05	25.1 ± 7.0[b]	9 ± 1.9	0.94 ± 0.03
GM	5.2 ± 2.9	6 ± 1.0	0.97 ± 0.07	6.2 ± 4.3	8 ± 1.3	0.98 ± 0.02

[a] The variation in muscle activation is expressed in percentage and is relative to the nonfatigued condition (% CMJ_1). Positive cross-correlation indicates a leftward phase shift, whereas a negative value indicates a rightward phase shift.
[b] Significant differences ($P < 0.05$).

reduced ability of the muscles to generate force (e.g., central and/or peripheral fatigue; see Fitts (14)). Hence, rather than debating the possible sources and causes of fatigue, the present study focuses on the consequences of fatigue on movement coordination.

The present study revealed an early occurrence (leftward shift) in the time of the peak angular velocity around the knee and ankle joints after fatiguing the knee extensor muscles in comparison with the control countermovement jumps. Similar shifts were also observed in the net peak power around the knee joint. These findings will be discussed with the help of the electromyographic data.

Electromyography. The EMG traces found in the control countermovement jumps are qualitatively similar to those reported in the literature (e.g., Bobbert and Van Ingen Schenau (6) and Viitasalo et al. (35)). The increased activation that occurred immediately after the respective series of fatiguing exercises (see Table 2) is in agreement with other studies (26,35) and has been attributed to several factors such as the recruitment of new motor unit pools (38) and firing rate synchronization (4). These mechanisms have been generally interpreted as an attempt by the neuromuscular system to compensate for the failure to produce the same force output.

It is difficult to determine whether these adjustments in muscle activity constitute a physiological and/or cognitive strategy of the neuromuscular system to accommodate changes in muscle force-generating properties. However, the adjustments in muscle activation that are generally observed after a period of training (17) suggest that changes in muscle activation magnitude during maximal explosive movements are likely to be a physiological response of the neuromuscular system rather than a voluntary or cognitive adjustment. The results presented by Psek and Cafarelli (27), in which a common drive controls the activation of an agonist-antagonist pair as a functional entity, is also further evidence that muscle activation magnitude regulation of movements involving production of high force level is mediated by a spinal cord mechanism. A comprehensive discussion of this issue is beyond the scope of this study.

Hamstring coactivation is an important factor in maintaining knee joint stability (3) and has been shown to increase in high-velocity contractions as a protective mechanism (30). In countermovement jumps, where the knee peak positive joint angular velocity is high (700 to 1000 $deg \cdot s^{-1}$ (6,35)), strong antagonistic activity is expected during the final part of the movement. If the subjects do not decelerate the knee joint before full extension (via hamstrings cocontraction), the considerable amount of rotational energy achieved at the final part of the push-off phase would expose the soft tissues to damaging hyperextension. Despite playing a role stabilizing the knee joint, the increased hamstring coactivation detracts from the resultant (extensor) moment and may explain the early peak angular velocity and peak power that occurred after fatiguing the knee extensor muscles.

It is not known if the greater hamstrings activation that occurred when the knee flexor group was fatigued is an attempt of the neuromuscular system to sustain the rotational energy of the trunk segment as high as possible. Increasing the rotational energy around the hip joint would increase the amount of energy that could be transferred to the knee joint via rectus femoris (6). This would also explain the high activation of the rectus femoris and the small (nonsignificant) decrease in hip joint angular velocity during the push-off phase of the movement when the knee extensor muscles were fatigued.

Setting aside the differences in the magnitude of the electromyographic signals, which were interpreted in the present study as a physiological response, the traces recorded after fatigue were similar to those recorded during the control condition. The electromyographic traces in the fatigued condition were an amplified scale of the traces observed without fatigue, where the temporal characteristics remained relatively unchanged, irrespective of which muscle group was fatigued (see Figs. 5 and 6). This is in agreement with the speed-sensitive hypothesis (16), which suggests that faster limb movements are produced by increasing the magnitude of the neural pulse, whereas the duration of the pulse remains relatively constant. The similarities between the electromyographic traces and the absence of a significant phase shift between jump conditions indicates the existence of a stereotypical neural input, irrespective of which muscle had its force-generating property changed. These findings support the hypothesis that a stable coordination pattern exists. Therefore, changes in the kinematics and performance of the movement are likely to be the result of changes in the muscle force-generating properties

(e.g., contractile component failure) rather than changes in the temporal characteristics of the neural input.

The findings of the present study are consistent with simulation (7) and experimental (5) studies, where the execution of explosive movements was proven to rely on preprogrammed muscle stimulation patterns, which cannot be promptly changed, irrespective of the muscle properties. Bobbert and Van Soest (7) showed that muscle strength gains were not reflected in jump height achievement until the control of the muscle properties were reoptimized. These findings suggest the existence of a link between the capability of the muscles to generate force and the set of commands (neural input) used by the neuromuscular system to perform the movement and achieve maximal performances. In this study, fatiguing a muscle set (knee extensors/flexors) did not cause the neuromuscular system to reorganize the temporal characteristics of the controls (neural input). This is in agreement with the arguments of Van Zandwijk et al. (34) that maximal movements require a unique set of control signals yielding maximal performances.

It has been suggested that control reorganization occurs only after a period of practice, where the subjects are allowed to repeatedly solve the task requirements and learn how to control their changed muscle properties to improve their jump height performance. This may not be the case of maximal vertical jumps, which are not always practiced continuously under fatigue conditions (e.g., during a volleyball game). In other activities, in which the movement is frequently practiced under fatigue circumstances, as a natural consequence of the long duration and the nature of the activity, reorganization is likely to occur to prevent potential disruption of the movement pattern and performance (33). Indeed, several studies have reported muscle stimulation and/or segmental movement pattern changes under fatigue (cycling (18), running (19), sprinting (26), and lifting (31)). Note that most of these movements are repeatedly performed under fatigue conditions (e.g., running and sprinting) during training and/or competition situations. When trained under these circumstances, the subjects may be familiar with the coordinative exigencies required to accommodate the force strength decline and may perform the movement "optimally," i.e., using a coordination strategy that would allow them to make use of the best available muscle strength.

The long duration of the lifting movement (0.7 s) studied by Sparto et al. (31) may have allowed the subjects to use the information of the mechanical capacities of the muscles to restructure the motor command at low level so that the kinematic pattern could be relatively adapted during early stages of the movement. This is not the case of countermovement jumps, where the ballistic characteristic and the short duration of the propulsive phase do not allow the subjects to tune the control signals as they perform the movement (29).

In the case of the study performed by Bonnard et al.

(8), it can be speculated that the submaximal nature of the task (hopping) and the fact that this continuous exercise did not involve extensively all major muscles of the lower limbs allowed the subjects to compensate for the effects of fatigue and sustain the height of the jumps. Van Zandwijk et al. (34) demonstrated that the control signals used in maximal and submaximal vertical jump performances are strongly related, but differ with respect to the activity of the biarticular muscles, which are modulated differently (temporal shifting and amplitude changes).

Although the coordination strategy has been shown to be constant even after fatigue, only the transient (acute) effects of fatigue were examined in the present study. It is not known whether the neuromuscular system would reoptimize the neural input when the movement is continually practiced under fatigue conditions (i.e., under chronic fatigue conditions). The simulation study performed by Bobbert and Van Soest (7) provided some insight into the mechanisms of adaptation that occur when the movement controls (i.e., the neural input) are changed. They showed that increases in performance after changing the muscle force-generating properties (i.e., increased muscle strength) were achieved only when the neural input was reoptimized. Bobbert and Van Soest (7) suggested that improvements in performance would occur after a period of practice if the subjects have the opportunity to learn and adjust their coordination pattern (i.e., neural input) to the new force-generating properties of the muscles (i.e., a chronic adaptation). If the same effect exists under fatigue conditions, then repeatedly practicing countermovement jumps under the acute effects of fatigue may induce the subjects to adopt a new coordination strategy that may reinforce a coordination pattern (i.e., a chronic adaptation) that is not optimal for maximal performances in a nonfatigued state. As a consequence, training programs inducing repetitive use of control strategies, in which the properties of the muscles are not taken into account or are not specific, may produce unsatisfactory results or even reduced jump height performance. Further studies are necessary to investigate experimentally whether and how the neuromuscular system reorganizes/reoptimizes the neural input and the movement pattern after a period of practice (training) in which the capacity of the muscles to generate force is changed (increased or decreased).

CONCLUSION

In conclusion, it has been shown that vertical jump performance is affected by fatigue of the knee extensor muscles, but not by fatigue of knee flexors. Despite the change in effective muscle force and therefore jump height, there was no change in the temporal characteristics of the muscle activation pattern as indicated by surface EMG. This suggests that the same movement strategy was followed before and after fatigue. On the basis of the arguments proposed by Bobbert and Van Soest (7) that the neural input has to be adjusted to take into account the muscle force-generating properties to produce maximal performances, it is speculated that the coordination strategy (neural control) used after fatigue was no longer optimal for the muscle strength available.

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REFERENCES

1. ACIERNO, S. P., R. V. BARATTA, and M. SOLOMONOV. *A Practical Guide to Electromyography for Biomechanists.* New Orleans: Louisiana Medical Centre, Bioengineering Laboratory, Louisiana State University, 1995, pp. 4–8.
2. BACA, A. A comparison of methods for analysing drop jump performance. *Med. Sci. Sports Exerc.* 31:437–442, 1999.
3. BARATTA, R., M. SOLOMONOW, B. H. ZHOU, D. LETSON, R. CHUINARD, and R. D'AAMBROSIA. Muscular co-activation: the role of the antagonist musculature in maintaining knee stability. *Am. J. Sports Med.* 16:113–122, 1988.
4. BIGLAND-RITCHIE, B. EMG/force relations and fatigue of human voluntary muscle contractions. *Exerc. Sci. Sports Rev.* 9:75–117, 1981.
5. BOBBERT, M. F., and E. D. DE BRUIN. Training of muscle strength and control in vertical jumping. *Proceedings of the 3rd Annual Congress-European College of Sport Science, Manchester, UK,* 1998. Liverpool, UK: Centre for Health Care Development, 1998, p. 15.
6. BOBBERT, M. F., and G. J. VAN INGEN SCHENAU. Co-ordination in vertical jumping. *J. Biomech.* 21:249–262, 1988.
7. BOBBERT, M. F., and A. J. VAN SOEST. Effects of muscle strengthening on vertical jump height: a simulation study. *Med. Sci. Sports Exerc.* 26:1012–1020, 1994.
8. BONNARD, M., A. V. SIRIN, L. ODDSON, and A. THORSTENSSON. Different strategies to compensate for the effects of fatigue revealed by neuromuscular adaptation processes in humans. *Neurosci. Lett.* 166:101–105, 1994.
9. BOSCO, C., J. TIHANYI, F. LATTERI, G. FEKETE, P. APOR, and H. RUSKO. The effect of fatigue on store and re-use of elastic energy in slow and fast types of human skeletal muscle. *Acta Physiol. Scand.* 128:109–117, 1986.
10. DRILLIS, R. J., and R. CONTINI. *Technical Report No. 1166–03.* New York: New York University, 1966, pp. 3–4.
11. EDWARDS, R. H. T. Human muscle function and fatigue. In: *Human Muscle Fatigue: Physiological Mechanisms,* R. Porter and J. Whelan (Eds.). London: Pitman Medical, 1981, pp. 1–18.
12. ELORANTA, V. Effect of postural and load variation on the co-ordination of the leg muscles in concentric jumping movement. *Electromyogr. Clin. Neurophysiol.* 36:59–64, 1996.
13. FIGONI, S. H., C. B. CHRIST, and B. H. MASSEY. Effects of speed, hip and knee angle and gravity on hamstring to quadriceps torque ratios. *J. Orthop. Sports Phys. Ther.* 9:287–291, 1988.
14. FITTS, R. H. Cellular mechanisms of muscle fatigue. *Physiol. Rev.* 74:49–94, 1994.
15. GOLLHOFER, A., P. V. KOMI, M. MIYASHITA, and O. AURA. Fatigue during stretch-shortening exercises: changes in mechanical performance of human skeletal muscle. *Int. J. Sports Med.* 8:71–78, 1987.
16. GOTTLIEB, G. L., D. M. CORCOS, and G. C. AGARWAL. Strategies for the control of single mechanical degree of freedom voluntary movements. *Behav. Brain Sci.* 12:189–210, 1989.
17. HAKKINEN, K., M. ALEN, and P. V. KOMI. Effect of explosive type strength training on isometric force and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. *Acta Physiol. Scand.* 125:587–600, 1985.
18. HAUTIER, C. A., L. M. ARSAC, K. DEGHDEGH, J. SOUQUET, A. BELLI, and J. LACOUR. Influence of fatigue on EMG/force ratio and co-contraction in cycling. *Med. Sci. Sports Exerc.* 32:839–843, 2000.
19. HEISE, G. D., D. W. MORGAN, H. HOUGH, and M. CRAIB. Relationship between running economy and temporal EMG characteristics of bi-articular leg muscles. *Int. J. Sports Med.* 17:128–133, 1996.
20. HORTOBÁGYI, T., N. J. LAMBERT, and W. P. KROLL. Voluntary and reflex responses to fatigue with stretch-shortening exercise. *Can. J. Sport Sci.* 16:142–150, 1991.
21. HUBLEY, C. L., and R. P. WELLS. Work-energy approach to determine individual joint contributions to vertical jump performance. *Eur. J. Appl. Physiol.* 50:247–254, 1983.
22. JACOBS, R., and G. J. VAN INGEN SCHENAU. Control of an external force in leg extensions in humans. *J. Physiol.* 457:611–626, 1992.
23. KELLIS, E., and V. BALTZOPOULOS. The effects of antagonist moment on the resultant knee joint moment during isokinetic testing of the knee extensors. *Eur. J. Appl. Physiol.* 76:253–259, 1997.
24. MACINTYRE, D. L., R. W. DARLENE, D. M. LYSTER, I. J. SZASZ, and D. C. MCKENZIE. Presence of WBC, decrease strength and delayed soreness in muscle after eccentric exercise. *J. Appl. Physiol.* 80: 1006–1003, 1996.
25. PANDY, M. G., and F. E. ZAJAC. Optimal muscular co-ordination strategies for jumping. *J. Biomech.* 24:1–10, 1991.
26. PINNIGER, G. J., J. R. STEELE, and H. GROELLER. Does fatigue induced by repeated dynamic efforts affect hamstring muscle function? *Med. Sci. Sport Exerc.* 2:647–653, 2000.
27. PSEK, J. A., and E. CAFARELLI. Behaviour of coactive muscles during fatigue. *J. Appl. Physiol.* 74:170–175, 1993.
28. RODACKI, A. L. F., N. E. FOWLER, and S. J. BENNETT. Multi-segment coordination: fatigue effects. *Med. Sci. Sports Exerc.* 33:1157–1167, 2001.
29. SCHMIDT, R. A. *Motor Control and Learning: A Behavioural Emphasis.* Champaign, IL: Human Kinetics, 1982, pp. 48–75.
30. SNOW, C. J., J. COOPER, A. O. QUANBURY, and J. E. ANDERSON. Antagonist co-contraction of knee extensors during constant velocity muscle shortening and lengthening. *J. Electromyogr. Kinesiol.* 5:185–192, 1995.
31. SPARTO, P. J., M. PARNIANPUOR, T. E. REINSEL, and S. SIMON. The effect of fatigue on multi-joint kinematics, co-ordination, and postural stability during a repetitive lifting test. *J. Orthop. Sports Phys. Ther.* 25:3–12, 1997.
32. TESCH, P. A., G. A. DUDLEY, M. R. DUVOISIN, B. M. HATHER, and H. T. HARRIS. Force and EMG signal patterns during repeated bouts of concentric and eccentric muscle actions. *Acta Physiol. Scand.* 138:263–271, 1990.
33. VAN INGEN SCHENAU, G. J., A. J. VAN SOEST, F. J. M. GABREËLS, and M. W. I. M. HORSTINK. The control of multi-joint movements relies on detailed internal representations. *Hum. Movement Sci.* 14:531–538, 1995.
34. VAN ZANDWIJK, J. P., M. F. BOBBERT, M. MUNNEKE, and P. PAS. Control of maximal and sub-maximal vertical jumps. *Med. Sci. Sports Exerc.* 32:477–485, 2000.
35. VIITASALO, J. T., K. HÄMÄLÄINEM, H. MONONEN, A. SALO, and J. LAHTINEN. Biomechanical effects of fatigue during continuous hurdle jumping. *J. Sports Sci.* 11:503–509, 1993.
36. VIITASALO, J. T., A. SALO, and J. LAHTINEN. Neuromuscular functioning of athletes and non-athletes in the drop jump. *Eur. J. Appl. Physiol.* 78:432–440, 1998.
37. WEIR, J. P., D. A. KEEFE, J. F. EATON, R. T. AUGUSTINE, and D. M. TOBIN. Effect of fatigue on hamstring co-activation during isokinetic extensions. *Eur. J. Appl. Physiol.* 78:555–559, 1998.
38. WITTEKOPFT, G., E. SCHAAF, and H. TAUBENHEIM. Use of electromyography for quantification of local muscular fatigue following a known strength-endurance load. In: *Biomechanics VI-A,* E. Asmussen and K. Jorgensen (Eds.). Baltimore: University Park Press, 1978, pp. 185–193.