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Increased rate of force development and neural drive of human skeletal muscle following resistance training

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Aagaard, Per, Erik B. Simonsen, Jesper L. Andersen, Peter Magnusson, and Poul Dyhre-Poulsen. Increased rate of force development and neural drive of human skeletal muscle following resistance training. *J Appl Physiol* 93: 1318–1326, 2002. First published July 12, 2002; 10.1152/japplphysiol.00283.2002.—The maximal rate of rise in muscle force [rate of force development (RFD)] has important functional consequences as it determines the force that can be generated in the early phase of muscle contraction (0–200 ms). The present study examined the effect of resistance training on contractile RFD and efferent motor outflow ("neural drive") during maximal muscle contraction. Contractile RFD (slope of force-time curve), impulse (time-integrated force), electromyography (EMG) signal amplitude (mean average voltage), and rate of EMG rise (slope of EMG-time curve) were determined (1-kHz sampling rate) during maximal isometric muscle contraction (quadriceps femoris) in 15 male subjects before and after 14 wk of heavy-resistance strength training (38 sessions). Maximal isometric muscle strength [maximal voluntary contraction (MVC)] increased from 291.1 ± 9.8 to 339.0 ± 10.2 N·m after training. Contractile RFD determined within time intervals of 30, 50, 100, and 200 ms relative to onset of contraction increased from 1,601 ± 117 to 2,020 ± 119 ($P < 0.05$), 1,802 ± 121 to 2,201 ± 106 ($P < 0.01$), 1,543 ± 83 to 1,806 ± 69 ($P < 0.01$), and 1,141 ± 45 to 1,363 ± 44 N·m·s^{-1} ($P < 0.01$), respectively. Corresponding increases were observed in contractile impulse ($P < 0.01$–0.05). When normalized relative to MVC, contractile RFD increased 15% after training (at zero to one-sixth MVC; $P < 0.05$). Furthermore, muscle EMG increased ($P < 0.01$–0.05) 22–143% (mean average voltage) and 41–106% (rate of EMG rise) in the early contraction phase (0–200 ms). In conclusion, increases in explosive muscle strength (contractile RFD and impulse) were observed after heavy-resistance strength training. These findings could be explained by an enhanced neural drive, as evidenced by marked increases in EMG signal amplitude and rate of EMG rise in the early phase of muscle contraction.

electromyography; neural adaptation; quadriceps muscle

"EXPLOSIVE" MUSCLE STRENGTH can be defined as the rate of rise in contractile force at the onset of contraction, i.e., the rate of force development (RFD) exerted within the early phase of rising muscle force (24, 41, 44, 46) (Figs. 1 and 2). In isolated muscle preparations, contractile RFD is obtained from the slope of the force-time curve (Δforce/Δtime), whereas, for intact joint actions, RFD is calculated as the slope of the joint moment-time curve (Δmoment/Δtime). The RFD parameter has important functional significance in fast and forceful muscle contraction. For example, fast movements such as sprint running, karate, or boxing typically involve contraction times of 50–250 ms. In contrast, it typically takes a longer time to reach maximum force in most human muscles, i.e., ≥300 ms for the elbow flexors (45) and knee extensors (46) (compare Figs. 1 and 2). During fast limb movements, therefore, the short contraction time may not allow maximal muscle force to be reached. As a result, any increase in contractile RFD becomes highly important as it allows reaching a higher level of muscle force in the early phase of muscle contraction, e.g., within the initial 100–200 ms of contraction. In addition to RFD, another important strength parameter is the total contractile impulse that can be produced within a given contraction time (9). In accordance with classic mechanical physics, the impulse, defined as the time (t)-integrated moment of force ($\int$Moment dt), is identical to the kinetic impulse (or "momentum") reached during limb movement. The latter is defined by I·w, in which I denotes the moment of inertia of the limb, and w denotes the instantaneous rotational velocity of the limb. Thus for an isolated, single-joint movement, the level of contractile impulse at any time point t directly determines the rotational angular velocity w of the distal segment at time t.

Our laboratory has previously observed a marked increase in the maximal contractile moment and power generated during fast, nonisokinetic knee extension after a prolonged period of heavy-resistance strength training (7). In addition, the maximal joint moment recorded during fast isokinetic limb movements was

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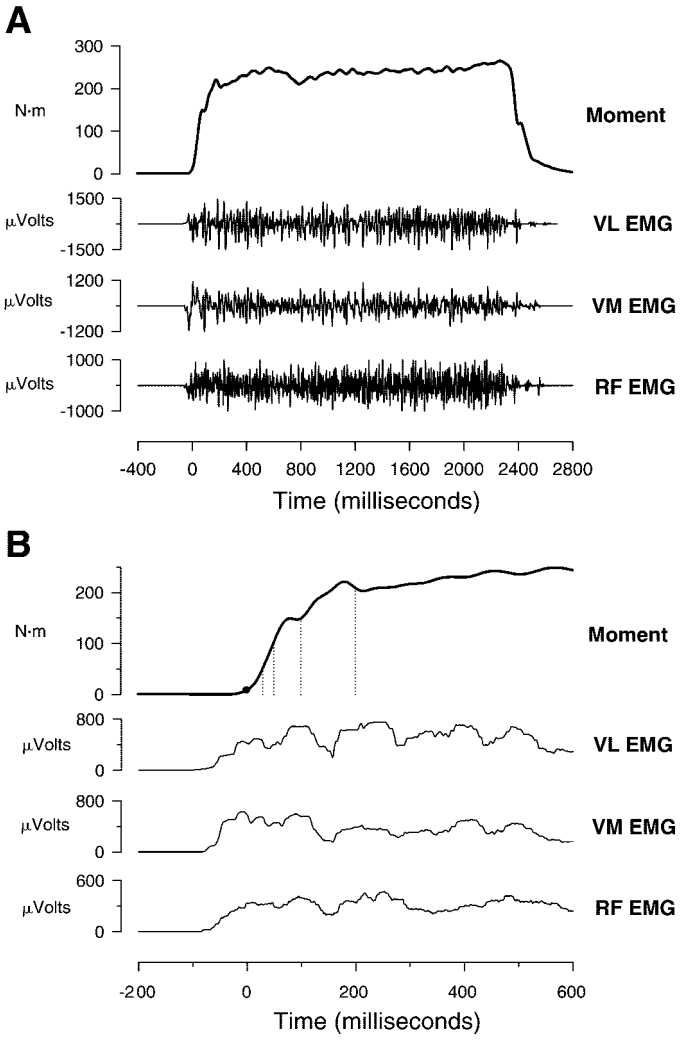

Fig. 1. *A*: moment (*top* trace) and raw electromyography (EMG) signals recorded during maximal isometric contraction of the quadriceps femoris muscle. VL, vastus lateralis; VM, vastus medialis; RF, rectus femoris. Time = 0 corresponds to the onset of muscle contraction. All moment and EMG signals were recorded at 1,000-Hz sampling rate. Contractile rate of force development (RFD) was defined as the slope of the moment-time curve (Δmoment/Δtime) derived at time intervals of 0–30, 0–50, 0–100, and 0–200 ms relative to the onset of contraction (see *B*). *B*: moment (*top* trace) and filtered EMG signals (moving root-mean-square filter, 50-ms time constant); same trial as in *A*. Note the change in axis range. Dotted vertical lines indicate time intervals of 30, 50, 100, and 200 ms relative to the onset of contraction.

positively related to the relative content of type II myosin heavy chain (MHC) isoforms in the quadriceps [vastus lateralis (VL)] (1) and triceps surae (lateral gastrocnemius) muscle (29). Based on these observations, it was suggested that contractile RFD is a major determinant of the maximal force and velocity that can be achieved during fast limb movements (1, 5).

RFD is inherently of major importance for athletes engaged in sports that involve an explosive type of muscle action. However, RFD may also play an important role in other populations. For example, in the elderly individual, the ability to exert a rapid rise in muscle force may reduce the incidence of falls related to the impaired control of postural balance with increasing age.

Contractile RFD may be influenced by the level of neural activation (20), muscle size, and fiber-type (MHC isoform) composition (27). Based on electromyography (EMG) recordings, an enhanced neuromuscular drive has been demonstrated after heavy-resistance strength training (3, 21–26, 30, 36, 47), and a "parallelism" between EMG and RFD has previously been postulated to exist (31). Accordingly, concurrent adaptations in efferent neural drive and contractile RFD may be expected in response to resistance training.

It was the aim of the present study to examine changes in maximal contractile RFD, impulse, and efferent neural drive evoked by intense, heavy-resistance strength training.

MATERIALS AND METHODS

Subjects. Fifteen male subjects volunteered to participate in the study (body mass 74.1 ± 5.7 kg, height 179 ± 3 cm, age 23.3 ± 3.7 yr, means ± SD). All subjects gave their informed consent to the procedures of the study. None of the subjects had previously participated in systematic resistance training. The conditions of the study were approved by the local ethics committee.

Training. Details of the training regime have been reported previously (8). In brief, progressive heavy-resistance strength training was performed for 14 wk for a total of 38 sessions. All training sessions were surveyed and supervised by the authors of the study. Obligatory leg training exercises were hack squats, incline leg press, isolated knee extension, hamstring curls, and seated calf raises. Four (*weeks 1–10*) or five (*weeks 11–14*) sets were performed for each exercise. Training loads ranged between 3 repetitions maximum (RM) to 10 RM, except for the first 10 days (4 sessions), when lower loading was used (10–12 RM). Very heavy loadings (4–6 RM) and increased number of sets (ensuring unchanged total workload) were used in the final 4 wk of the study.

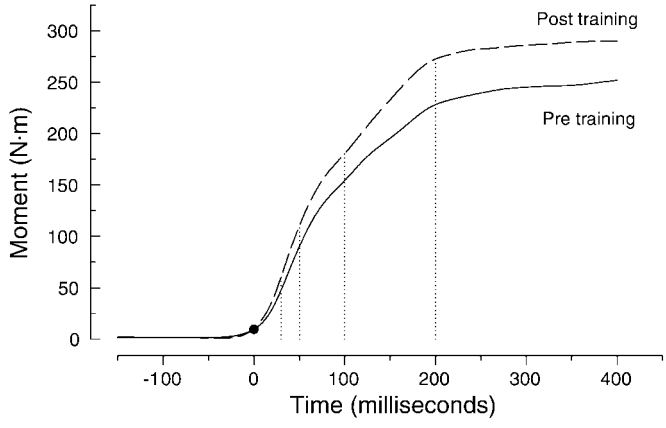

Fig. 2. Average moment-time curves (n = 15) obtained before and after 14 wk of heavy-resistance strength training. Onset of contraction is denoted by solid circle. Dotted vertical lines indicate time intervals of 30, 50, 100, and 200 ms relative to the onset of contraction. Increases in peak isometric moment were observed posttraining in parallel with a steeper slope of the moment-time curve in the early time phase of muscle contraction. The increase in slope was reflected by a significant increase in contractile RFD, which was observed both in the initial (30 and 50 ms) and later (100 and 200 ms) phases of force rise.

Measurement of maximal muscle strength and contractile RFD. As previously described (2), maximal quadriceps muscle strength was measured as maximal isometric knee extension moment exerted in an isokinetic dynamometer (KinCom; Kinetic Communicator, Chattecx, Chattanooga, TN). The reliability and validity of this dynamometer have been described elsewhere (18). Subjects were seated in a rigid chair and firmly strapped at the hip and distal thigh. The rotational axis of the dynamometer was visually aligned to the lateral femoral epicondyle, and the lower leg was attached to the dynamometer lever arm above the medial malleolus, with no static fixation of the ankle joint. The individual positioning for each subject of the seat, backrest, dynamometer head, and lever arm length was similar pre- and posttraining. All measurements were performed in the right leg. Subjects were familiarized with the dynamometer and the procedures of the experiment on separate occasions.

Maximal isometric quadriceps contractions were performed during static knee extension at a knee joint angle of 70° (0° = full knee extension) (2). After 10 min of warmup followed by a number of submaximal and maximal preconditioning trials, each subject performed three to four knee extensions at maximal voluntary effort. Subjects were carefully instructed to contract "as fast and forcefully as possible." On-line visual feedback of the instantaneous dynamometer force was provided to the subjects on a computer screen. Trials with an initial countermovement (identified by a visible drop in the force signal) were always disqualified, and a new trial was performed.

The dynamometer strain-gauge signal and all EMG signals were synchronously sampled at a 1,000-Hz analog-to-digital conversion rate by using an external analog-to-digital converter (dt2801-A, Data Translation, Marlboro, MA) (Fig. 1*A*). During later off-line analysis, the strain-gauge signal was smoothed by a digital fourth-order, zero-lag Butterworth filter, by using a cutoff frequency of 15 Hz (48). Subsequently, the strain-gauge signal was converted to newtons and multiplied by the individual lever arm length to calculate the moment of force ("torque"). All recorded moments were corrected for the effect of gravity on the lower limb according to procedures described previously (6).

Contractile RFD and impulse were determined from the trial with maximal isometric moment of force [maximal voluntary contraction (MVC)]. RFD was derived as the average slope of the moment-time curve (Δmoment/Δtime) over time intervals of 0–30, 0–50, 0–100, and 0–200 ms relative to the onset of contraction (Fig. 1*B*, Fig. 2). Similarly, contractile impulse was determined as the area under the moment-time (t) curve ($\int$Moment dt) in time intervals of 0–30, 0–50, 0–100, and 0–200 ms relative to onset of contraction. $\int$Moment dt is identical to the kinetic impulse (or momentum) of the lower limb in case it had been allowed to move. More specifically, during single-joint movements, the kinetic impulse is defined by $I \cdot w$. Thus the magnitude of impulse is directly proportional to the angular velocity (w) that the segment would reach had it been allowed to move. Furthermore, as it is a measure of the cumulated area covered by the moment-time curve, the impulse reflects the entire time history of contraction, including the overall influence of the various time-related RFD parameters (i.e., determined at 30, 50, 100, and 200 ms). Normalized RFD was determined as the slope of the moment-time curve when normalized relative to peak isometric moment, MVC. Normalized RFD (expressed as %MVC/s) was calculated from the onset of contraction to the level of one-sixth, one-half, and two-thirds MVC, respectively (13). Thereby the change in normalized RFD could be evaluated in the very initial phase of contraction (one-sixth MVC, ~30 ms relative to contraction onset), as well as in the intermediate (one-half MVC, ~100 ms) and later phase (two-thirds MVC, ~150 ms) (see RESULTS). Onset of muscle contraction was defined as the time point at which the moment curve exceeded baseline moment by >7.5 N·m (absolute RFD) or by 2.5% of the difference between baseline moment and MVC (normalized RFD).

EMG. After careful preparation of the skin (shaving, abrasion, and cleaning with alcohol), pairs of surface electrodes (Medicotest Q-10-A, 20-mm interelectrode distance) were placed at the VL, vastus medialis (VM), and rectus femoris (RF). All electrode positions were carefully measured in each subject to ensure identical pre- and posttraining recording sites (3). The EMG electrodes were connected directly to small custom-built preamplifiers (input impedance 80 MΩ) taped to the skin (43). EMG signals were led through shielded wires to custom-built differential instrumentation amplifiers with a frequency response of 10–10,000 Hz and common mode rejection ratio exceeding 100 dB.

All EMG signals were recorded at a 1,000-Hz sampling rate as described above for the dynamometer strain-gauge signal (Fig. 1*A*). During the later process of analysis, the EMG signals were digitally high-pass filtered by using a fourth-order, zero-lag Butterworth filter with a 5-Hz cutoff frequency (48), followed by a moving root-mean-square filter with a time constant of 50 ms (10). Raw and filtered EMG signals are shown in Fig. 1, *A* and *B*, respectively.

The following parameters were identified in each trial: *1*) peak EMG amplitude within the entire contraction phase; *2*) integrated EMG (iEMG) in time intervals of 0–30, 0–50, 0–100, and 0–200 ms relative to onset of EMG integration (defined below); *3*) average EMG [mean average voltage (MAV) = iEMG/integration time] in time intervals of 0–30, 0–50, 0–100, and 0–200 ms relative to onset of EMG integration; and *4*) the rate of EMG rise (RER), determined as the slope (ΔEMG/Δtime) of the filtered EMG signal (41), calculated in time intervals of 0–30, 0–50, and 0–75 ms relative to onset of EMG integration. Onset of EMG integration was initiated 70 ms before the individual onset of contraction to account for the presence of electromechanical delay. For the determination of RER, a time interval of 75 ms instead of 100 ms was used for the longer interval, because a decrease in EMG signal amplitude typically occurred after ~80–100 ms of neural activity (i.e., Fig. 1*B*).

EMG signals were also obtained in the lateral (biceps femoris caput longus) and medial (semitendinosus) hamstring muscles to monitor the degree of antagonist muscle coactivation. The EMG signals were sampled and processed as described above for VL, VM, and RF. Electrode positions were carefully measured to ensure identical recording sites pre- and posttraining. Peak EMG amplitude was determined within each 2-s contraction phase, and iEMG and MAV were obtained in time intervals of 0–30, 0–50, 0–100, and 0–200 ms relative to the onset of EMG integration. In addition, antagonist coactivation was evaluated by expressing all hamstring EMG parameters relative to that measured in separate trials of maximal agonist hamstring contraction (70° knee joint angle; experimental procedures identical to those described above).

Statistics. Data are given as group mean values ± SE. All pre- to posttraining changes were evaluated with Wilcoxon signed-rank test for paired samples (two-tailed, 0.05 level of significance).

RESULTS

Figure 1 shows the moment of force and EMG recorded for the quadriceps femoris muscle during a representative trial of maximal voluntary effort. Figure 1*A* displays the moment signal and raw EMG signals recorded during the entire contraction phase, and Fig. 1*B* shows the moment signal and filtered EMG signals in the time interval of −200–600 ms relative to the onset of contraction.

Maximum isometric quadriceps contraction strength (MVC) increased with training, from 291.1 ± 9.8 to 339.0 ± 10.2 N·m ($P < 0.001$). In addition, a steeper slope was observed for the moment-time curve after training (Fig. 2). Thus contractile RFD increased from 1,601 ± 117, 1,802 ± 121, 1,543 ± 83, and 1,141 ± 45 $N \cdot m \cdot s^{-1}$ at 30, 50, 100, and 200 ms, respectively, to 2,020 ± 119, 2,201 ± 106, 1,806 ± 69, and 1,363 ± 44 $N \cdot m \cdot s^{-1}$ posttraining, respectively ($P < 0.01$–0.05) (Fig. 3). Contractile impulse increased from 0.73 ± 0.04, 2.12 ± 0.14, 8.48 ± 0.50, and 27.97 ± 1.37 N·m·s when obtained at time intervals of 0–30, 0–50, 0–100, and 0–200 ms, respectively, to 0.91 ± 0.05, 2.64 ± 0.14, 10.18 ± 0.45, and 33.31 ± 1.25 N·m·s, respectively ($P < 0.01$–0.05) (Fig. 4).

To examine the change in normalized RFD, all obtained moment-time curves were also analyzed when divided by the peak moment exerted within the 2-s contraction phase (Fig. 5). A 15% increase in normalized RFD was observed in the initial phase of force rise (zero to one-sixth MVC), from 536.2 ± 33.5% MVC/s before training to 616.2 ± 22.5% MVC/s after training ($P < 0.05$) (Fig. 6). The time from onset of contraction to one-sixth MVC decreased from 34.0 ± 2.8 to 28.5 ± 1.1 ms after training ($P < 0.05$). Normalized RFD remained unchanged at higher force levels: 573.4 ± 46.2 and 578.4 ± 28.1% MVC/s (at zero to one-half MVC) and 475.4 ± 38.5 and 471.3 ± 15.8% MVC/s (at zero to two-thirds MVC) pre- and posttraining, respectively (Fig. 6). Similarly, no changes were observed in the time to reach one-half MVC (97.5 ± 9.7 vs. 90.1 ± 4.6 ms) or two-thirds MVC (155.5 ± 14.7 vs. 144.4 ± 5.4 ms).

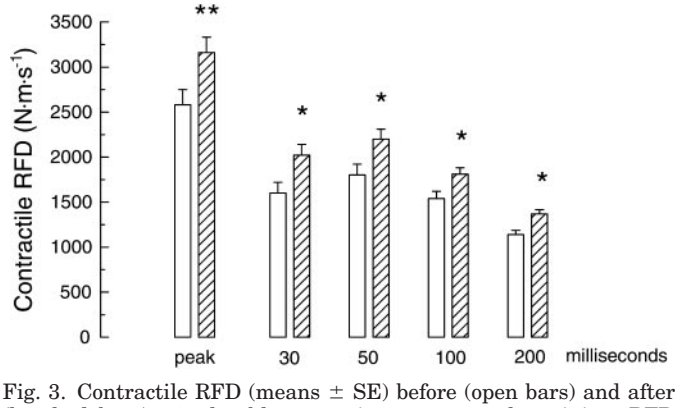

Fig. 3. Contractile RFD (means ± SE) before (open bars) and after (hatched bars) 14 wk of heavy-resistance strength training. RFD (Δmoment/Δtime) was calculated in time intervals of 0–30, 50, 100, and 200 ms (Δtime) from the onset of contraction. In addition, peak RFD was determined within the early contraction phase (0–200 ms). Pre- to posttraining differences: $^*P < 0.05$ and $^{**}P < 0.01$.

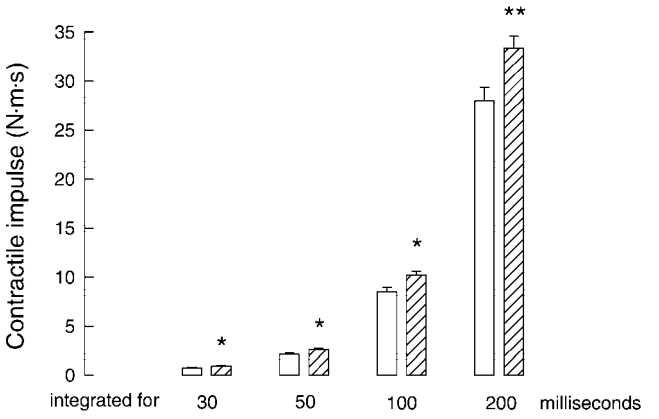

Fig. 4. Contractile impulse (means ± SE) before (open bars) and after (hatched bars) 14 wk of heavy-resistance strength training. Contractile impulse, defined as the area covered by the moment-time curve ($\int$Moment dt), was calculated in the time intervals of 0–30, 50, 100, and 200 ms from the onset of contraction. Pre- to posttraining differences: $^*P < 0.05$ and $^{**}P < 0.01$.

In the initial phase of muscle contraction, mean average EMG (MAV) increased 77–143% in VL (0–30, 0–50, and 0–100 ms), 25% in VM (0–100 ms), and 22–44% in RF (0–50 and 0–100 ms) (Fig. 7*A*). In addition, RER increased 54–106% in VL (0–30, 0–50, and 0–75 ms), 41–68% in VM, and 71–97% in RF (0–30 and 0–50 ms) (Fig. 7*B*). No statistical changes in maximum EMG amplitude were observed with training: VL, 635.0 ± 66.1 vs. 740.6 ± 102.5 μV; VM, 854.0 ± 65.1 vs. 955.2 ± 91.3 μV; and RF, 511.5 ± 46.5 vs 590.9 ± 71.0 μV pre- and posttraining, respectively.

The magnitude of antagonist hamstring EMG remained unchanged with training when examined as peak EMG amplitude and also when iEMG and MAV

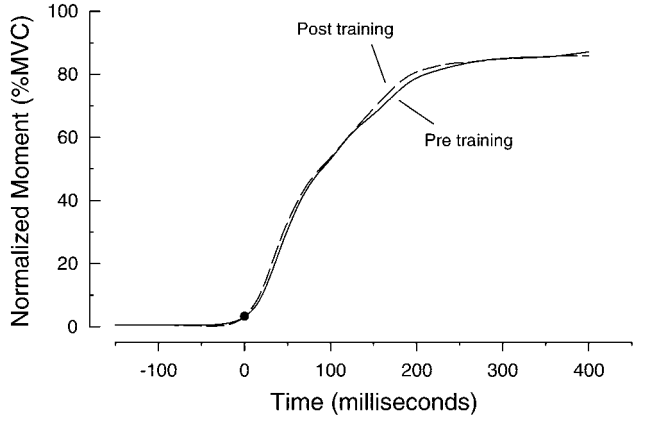

Fig. 5. Normalized moment-time curve, averaged for all subjects before (solid line) and after (dashed line) the period of heavy-resistance strength training. Onset of contraction is denoted by the solid circle. Steeper slopes of the normalized moment-time curves were observed in the initial phase of force rise after training, as reflected by an increase in normalized RFD at 0–⅙ maximal voluntary contraction (MVC). In contrast, normalized RFD remained unchanged when determined at higher force levels (0–½ MVC, 0–⅔ MVC) (see Fig. 6).

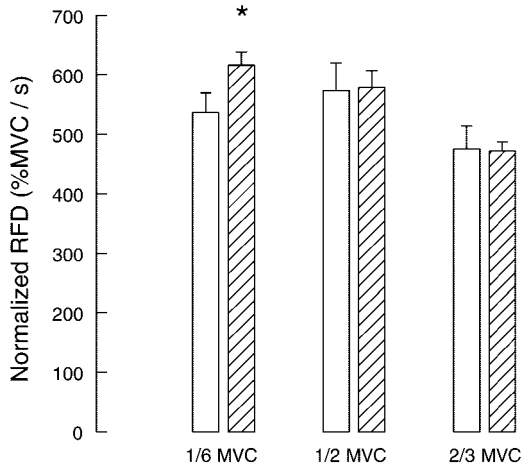

Fig. 6. Normalized RFD (means ± SE) before (open bars) and after (hatched bars) 14 wk of heavy-resistance strength training. Normalized RFD was determined at normalized force intervals of 0–⅙ MVC, 0–½ MVC, and 0–⅔ MVC. Pre- to posttraining differences: $*P < 0.05$.

were determined in time intervals of 30, 50, and 100 ms relative to the onset of EMG integration (P = 0.46–0.82). Similarly, no change was observed when these antagonist hamstring EMG values were normalized relative to that measured in separate trials of maximal agonist hamstring contraction (P = 0.31–0.39).

DISCUSSION

The present study demonstrated significant increases in contractile RFD and impulse, as well as in the efferent neural drive to the quadriceps femoris muscle after 14 wk of intensive heavy-resistance strength training. As discussed below, the training-induced gains in contractile RFD and impulse were attributed to an enhanced neural drive in the early phase of muscle contraction (0–200 ms). As a novel finding, concurrent increases in RFD and EMG signal amplitude also were observed in the most initial phase of muscle contraction (0–50 ms). Furthermore, marked increases in the RER were observed for VL, VM, and RF after training.

Changes in contractile RFD and impulse. Contractile RFD increased 17–26% after the period of resistance training, both when determined in the very early (0–50 ms, 23–26%) and the later (100–200 ms, 17–20%) phases of rising muscle force (Fig. 3). Similarly, RFD has previously been reported to increase in response to resistance training (21–26, 41, 45–47). In the present study, normalized RFD increased 15% at zero to one-sixth MVC, whereas no change was observed at higher force levels. RFD obtained at zero to one-sixth MVC corresponds to the very initial phase of muscle contraction, as it involves a time interval of ~30 ms relative to the onset of contraction (34.0 ms pretraining, 28.5 ms posttraining). This change in relative RFD properties indicates that qualitative changes may have occurred with training, i.e., potentially involving alterations in motoneuron recruitment and firing frequency, increased incidence of discharge doublets, and changes in MHC isoform composition and sarcoplasmic reticulum Ca^{2+} kinetics (factors discussed in detail below). Other studies have observed similar increases in normalized RFD after explosive-type resistance training, typically measured as the time to reach 30% MVC with no changes occurring at force levels of 60 and 90% MVC (24, 25). In contrast, normalized RFD has also been found to remain unchanged (24, 35) or even decrease (21) in response to resistance training.

Although only rarely reported in the literature (9), contractile impulse is perhaps the single most important strength parameter because it incorporates the aspect of contraction time, which is neglected using most other strength parameters. By providing a measure of the accumulated area covered by the moment-time curve, the impulse reflects the specific time history of contraction, which includes the overall influence of all of the examined RFD parameters. In the present study, contractile impulse increased in response to resistance training, when determined in both the initial (0–50 ms) and later (100–200 ms) phases of muscle

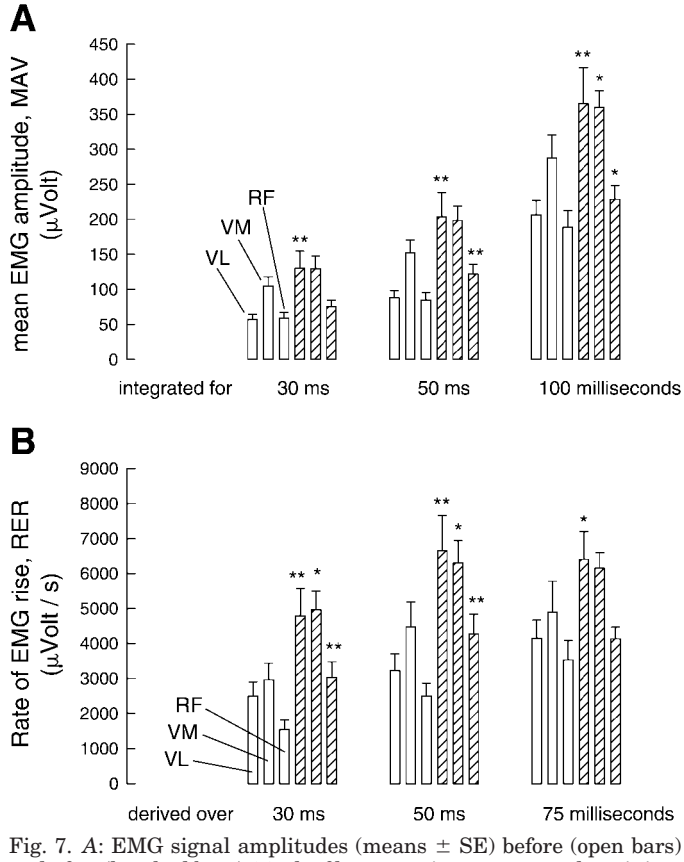

Fig. 7. *A*: EMG signal amplitudes (means ± SE) before (open bars) and after (hatched bars) 14 wk of heavy-resistance strength training. Neural efferent drive was calculated as the mean integrated EMG divided by the respective integration time in time intervals of 0–30, 50, and 100 ms relative to the onset of EMG integration. MAV, mean average voltage. Pre- to posttraining differences: $*P < 0.05$ and $**P < 0.01$. *B*: rate of EMG rise (RER) (means ± SE) before and after 14 wk of heavy-resistance strength training. RER (ΔEMG/Δtime) was determined in time intervals (Δtime) of 0–30, 50, and 75 ms relative to the onset of EMG integration. Pre- to posttraining differences: $*P < 0.01$ and $**P < 0.001$.

contraction (Fig. 4). These increases were found to occur simultaneously with a marked increase in mean average EMG amplitude in the initial phase of contraction (Fig. 7*A*). Similarly, increases in contractile RFD (Fig. 3) were accompanied by marked increases in the RER (Fig. 7*B*) (changes in EMG are discussed in detail below). In contrast, Baker and coworkers (9) were unable to demonstrate any increase in contractile impulse obtained in unilateral isometric leg extension and unilateral isometric bench press after 12 wk of heavy-resistance strength training. However, in their study, all training was performed as bilateral resistance exercises, whereas changes in RFD and impulse were evaluated in unilateral leg extension tests (9). In fact, RFD has been reported to increase after bilateral resistance and jump training when measured in bilateral tests (21, 25), whereas no change was observed during unilateral test conditions (24).

Changes in efferent neural drive. Increases in efferent motoneuron output ("efferent neural drive"), as evidenced by changes in EMG signal amplitude, have been reported after heavy-resistance strength training (3, 21–26, 30, 36). Because a "parallelism" between rates of EMG and force development has been proposed to exist (31), training-induced increases in contractile RFD would be expected to be accompanied by a rise in efferent neural drive. This notion was supported by the marked increases in RFD and EMG presently recorded in the early phase of muscle contraction, which confirms previous observations (21–26, 41, 47). Importantly, in the present study, concurrent increases in RFD and EMG interference amplitude were demonstrated to occur in the most initial phase of muscle contraction (0–50 ms), which has not been reported previously. Furthermore, differentiated changes in the RER were examined in VL, VM, and RF for the first time (Fig. 7*A*). In the studies by Häkkinen and coworkers (21–26), EMG signals were integrated in fixed 100-ms time periods by using a 50-ms overlap with a resulting time resolution of 50 ms. In contrast, the moving root-mean-square filter used in the present study allowed the analysis of all EMG signals with a 1-ms time resolution. Consequently, for the first time, it was possible to identify changes in efferent neural drive in the very early phase of rising muscle force (0–50 ms). This allowed us to evaluate the effect of increased neural drive on contractile RFD at the onset of contraction, thus reflecting changes in motoneuron discharge rate and/or recruitment.

An increase in motoneuron firing frequency would be expected to yield a disproportionately greater increase in EMG interference amplitude than in contractile force when approaching the level of maximal force generation (16). Thus, in the present study, the disproportionately greater increases in iEMG and RER compared with RFD and maximal muscle strength may indicate that increased motoneuron firing frequency at the onset of contraction was mainly responsible for the training-induced rise in contractile RFD. This notion is further supported by the qualitative change in RFD observed in the initial phase of contraction, in terms of an increase in normalized RFD at zero to one-sixth MVC (Fig. 6). Interestingly, when recorded during maximal isometric contraction, evoked soleus H-reflex and V-wave responses were found to increase after the period of training ($n = 12$) (4). The elevated V-wave response directly reflects an increase in spinal motoneuronal output, which may comprise a significant increase in motoneuron firing frequency (4).

When electrically evoked tetanic contractions were used to examine the change in intrinsic muscle properties, peak RFD was augmented to a greater extent by fast ballistic training than isometric training (31 vs. 18%) in the human adductor pollicis muscle (17), which suggests that maximal ballistic training could also have an important effect on RFD. Comparing dynamic and isometric ballistic training (i.e., involving muscle contractions with high RFD), similar increases in RFD were produced with these two distinctly different types of training, during both evoked and voluntary contraction conditions (12). Thus the involvement of an intended ballistic effort appears to be more important for inducing increases in RFD than the type of contraction actually performed (12). It is possible that the primary stimulus for training-induced increases in RFD resides in the high-frequency motor unit-firing pattern associated with an intended ballistic effort (12).

Influence of motoneuron firing frequency on RFD. The present increase in efferent neural drive (increased MAV, RER) in response to resistance training may primarily reflect an increase in motoneuron firing frequency, although recruitment of previously nonactivated motoneurons could have contributed as well. Although the direct influence of motoneuron firing frequency was not directly addressed in the present study, a few considerations should be made regarding its potential influence on the RFD and contractile impulse. Motoneuron discharge rate influences not only the magnitude of contractile tension, but also the RFD, as observed both in isolated single-muscle fibers (32, 33), whole muscle in situ (14), and human muscle in vivo (20, 34, 37). When individual motor units were stimulated in the neonatal rat (38), it was noticed that RFD continued to increase at innervation rates higher than the rate producing maximum tetanic tension (37). Similar findings have been reported, both for whole muscle animal preparations (14) and human musculature in vivo, where an innervation rate of 100 Hz produced greater RFD, but not greater peak isometric force, compared with an innervation rate of 50 Hz (20). Thus the ability to produce supramaximal firing frequencies likely serves to increase maximal RFD, rather than increasing maximal contraction force per se (11, 40). In support of this notion, so-called discharge doublets (interspike interval <5–10 ms) can be observed in the firing pattern of single motoneurons at the onset of maximal rapid muscle contraction (47). The occurrence of such discharge doublets may result in a marked increase in contractile force and RFD (14–15, 34). Interestingly, the incidence of discharge doublets in the firing pattern of individual motor units increased sixfold (from 5.2 to 32.7%) in parallel with an increased

contractile RFD in response to a regime of ballistic-type resistance training (47).

Effects of changes in muscle morphology. Contractile RFD is influenced not only by the level of neural activation but also by muscle size and fiber-type (MHC isoform) composition (27). Moreover, during dynamic contraction conditions, RFD is also influenced by the specific force-velocity and length-tension properties of the respective muscles involved in the movement. Maximal cross-bridge cycle transition rate appears to be the major limiting factor for the maximal intrinsic RFD of mammalian muscle fiber (19). Thus a predominance of type II MHC isoforms will result in a high RFD (28), due to their elevated rate of cross-bridge cycling (32). In consequence, the maximal muscle force that can be reached in situations of limited contraction time (<250 ms) is positively related to the proportion of type II MHC (1, 29).

The present data cannot exclude the possible contribution from morphological adaptation to the training-induced increases in RFD and contractile impulse. In fact, preferential type II muscle fiber hypertrophy was observed after the period of training (19% increase in type II fiber cross-sectional area) when a subgroup of the present subjects was examined, although no changes were found in overall fiber-type composition (8). In addition, muscle fiber pennation angle also increased, which allowed for a marked increase in physiological muscle-fiber area, with a resulting rise in maximal contractile force (2). Most likely, the above factors have made additional contributions to the increase in contractile RFD and impulse observed in the present study, although their relative importance remains unknown.

The increase in normalized RFD (at one-sixth MVC) observed in the present study suggests that qualitative RFD changes might have occurred with training. In addition to the neural mechanisms discussed above, a possible explanation for this change could be a shift in type I vs. type II MHC isoform composition with training. However, no signs of such a change were observed for the present subjects (8). Theoretically, the increase in normalized RFD could in part be caused by enhanced intracellular Ca^{2+} release with training. Although sarcoplasmic reticulum Ca^{2+} kinetics recently were reported to increase in response to sprint cycle ergometer training (39), it is not known what impact resistance training would have on this parameter.

Implications for functional muscle performance. The specific time to reach a given level of contractile impulse is shown in Fig. 8. Curve points were derived by integration of the group-averaged moment-time curves (Fig. 2) from the time of contraction onset. As demonstrated in Fig. 8, a shorter time was needed to reach a given contractile impulse after training. For example, it took 109 ms to reach an impulse of 10 N·m·s before training, whereas only 98 ms were required after the period of training. Notably, 10 N·m·s corresponds closely to the kinetic impulse achieved during maximally fast unloaded knee extension with a peak angular velocity of 900°/s. Thus, with a moment of inertia (I) of the lower leg of 0.5–0.75 kg/m^2 (5) and a peak velocity of 900°/s (15 rad/s = w) reached after 45–60° range of joint excursion to yield a contraction time of 100–130 ms, an impulse of 8–11 N·m·s (I·w) can be calculated. This estimate is in good agreement with the contractile impulse of 10 N·m·s that was reached after 109 ms of contraction before training (Fig. 8). On average, subjects reached an impulse of 10 N·m·s after 98 ms of contraction posttraining to allow an additional 11 ms (109 to 98 ms) for further limb acceleration compared with the pretraining situation. Based on the data shown in Fig. 8, an extended contraction time of 98 + 11 = 109 ms would allow subjects to reach an impulse of 12.0 N·m·s posttraining. Consequently, a 20% (12.0 to 10.0 N·m·s) increase in maximal unloaded movement speed would be expected posttraining. In support of this notion, peak angular velocity recorded during maximal unloaded knee extension was observed to increase after the period of resistance training (from 949.9 ± 26.0 to 1,305.5 ± 80.5 °/s, $P <$ 0.005; unpublished results). Heavy-resistance strength training has previously been reported to induce increases in peak velocity during maximal unloaded limb movement (42) while also increasing the muscle moment and power exerted at peak velocity (7).

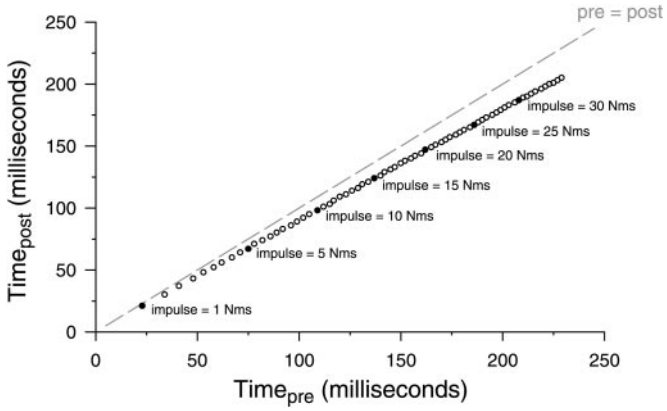

Fig. 8. Time needed to reach a given level of contractile impulse ($\int$Moment dt) before (Time$_{pre}$) and after (Time$_{post}$) the period of resistance training. Curve points were derived by integration of the group-averaged moment-time curves (Fig. 2). Abscissa and ordinate axes denote time of integration (relative to the onset of contraction), and impulse values are marked in 5 N·m·s increments. The magnitude of contractile impulse is directly proportional to the velocity of the lower limb segment, had it been allowed to move (see MATERIALS AND METHODS for details). Note that all curve points are located below the line of identity (dashed line), as it took significantly shorter time to reach a given value of contractile impulse after the period of training. For example, it took 109 ms to reach an impulse of 10 N·m·s before training, whereas only 98 ms were required after the period of training. Notably, 10 N·m·s corresponds closely to the kinetic impulse achieved during maximally fast unloaded knee extension with a peak angular velocity of 900°/s (see DISCUSSION).

Seen from a functional perspective, the increase in contractile RFD and impulse is probably the most important strength adaptation elicited by resistance training. Very fast movements may be characterized by muscle contraction times of 50–200 ms (karate, sprint running, rapid jumps), which are considerably less than the time it takes to reach maximal force (compare

with Fig. 1). Consequently, an elevated RFD induced by resistance training allows increase of the maximal force and velocity that can be achieved during rapid movements. It is important to notice that training-induced changes in contractile RFD will have important functional consequences not only in athletes but also in nonathletic subjects. For example, in the elderly individual, a high muscular RFD seems to be of vital importance for the ability to rapidly regain balance during sudden postural perturbations, thereby potentially reducing the risk of falls.

Conclusions. The present study demonstrated concurrent increases in the contractile RFD, impulse, and efferent neuromuscular drive of human skeletal muscle after intense, heavy-resistance strength training. This training-induced gain in explosive muscle strength could be explained by increases in efferent neural drive, as evidenced by marked elevations in EMG signal amplitude and RER in the early phase of muscle contraction.

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