Normalized force, activation, and coactivation in the arm muscles of young and old men

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Klein, C. S., C. L. Rice, and G. D. Marsh. Normalized force, activation, and coactivation in the arm muscles of young and old men. J Appl Physiol 91: 1341–1349, 2001.—The purpose of this study was to determine whether the loss of muscle strength in the elderly could be explained entirely by a decline in the physiological cross-sectional area (PCSA) of muscle. Isometric force, muscle activation (twitch interpolation), and coactivation (surface electromyograph) were measured during maximal voluntary contractions (MVCs) of the elbow flexors (EFs) and extensors (EEs) in 20 young (23 ± 3 yr) and 13 older (81 ± 6 yr) healthy men. PCSA was determined using magnetic resonance imaging, and normalized force (NF) was calculated as the MVC/PCSA ratio. The PCSA was smaller in the old compared with the young men, more so in the EEs (28%) compared with the EFs (19%) (P < 0.001); however, the decline in MVC (~30%) with age was similar in the two muscle groups. Muscle activation was not different between the groups, but coactivation was greater (5%) (P < 0.001) in the old men for both muscles. NF was less (11%) in the EEs (P < 0.01) and tended to be unchanged in the EEs of the old compared with young subjects. The relative maintenance of NF in the EEs compared with the EFs may be related to age-associated changes in the architecture of the triceps brachii muscle. In conclusion, although the decline in PCSA explained the majority of strength loss in the old men, additional factors such as greater coactivation or reduced specific tension also may have contributed to the age-related loss of isometric strength.

elbow flexors; elbow extensors; specific tension; physiological cross-sectional area; sarcopenia

A common observation in older people is the loss of muscle mass and strength (41). What is controversial, however, is whether the loss of strength (force) is entirely explained by the decrease in muscle size or whether other factors, such as muscle activation, coactivation, and specific tension, are also important (9, 11). The typical approach used to study this problem is to determine the normalized force (NF) in young and old subjects. The NF is the ratio of maximal voluntary force [maximal voluntary contraction (MVC)] to some measure of the size of the muscle(s) producing the force. A number of authors reported no age-related changes in NF (11, 26, 40), which implies that the decrease in muscle size explains virtually all of the force loss. In contrast, others have found up to a 40% decline in NF with age (8, 27, 32, 48), which indicates that other factors, in addition to a decrease in muscle size, may contribute to the loss of strength.

The explanation for the conflicting findings of NF and aging may relate, in part, to the methods used to measure muscle strength and muscle size. For example, a decline in NF with age seemed to be more prevalent during dynamic rather than isometric contractions, and the decrease may be greater in women compared with men (16, 32, 40). As well, muscle size may have been overestimated in older people because of the inability of indirect techniques to distinguish muscle from noncontractile tissue (8, 32). Moreover, the anatomic cross-sectional area (ACSA), which is the area of muscle measured from a single cross section, commonly measured in NF studies may underestimate the loss of muscle mass with age (2). A more accurate index of muscle size and force-generating capacity, particularly in muscles with a pennate fiber architecture such as the triceps brachii, may be the physiological cross-sectional area (PCSA) (12, 42). The PCSA, which is the total cross-sectional area of all the muscle fibers in a muscle, can be determined in vivo by combining the measurement of muscle volume from magnetic resonance imaging (MRI) with muscle fiber length estimated from cadavers. The PCSA of the upper and lower limb muscles has been measured in young subjects but not in the muscles of the elderly (12, 25).

Neural drive to the agonist and antagonist muscles can influence force-generating capacity. The level of muscle activation, as assessed by the twitch interpolation technique, was reported to be less in the elbow flexors (EFs) in old compared with young people (9, 49). As well, coactivation level of the knee flexors, as measured by surface electromyograph (EMG), was higher with old age during MVCs of the knee extensors (15, 22). A decrease in activation or an increase in coacti-
tion, or both, could reduce NF in aged people. However, in many studies of NF, the level of activation was not measured (27, 32, 48), so it is uncertain whether the young and old recruited all motor units maximally during the MVC. In addition, no studies have reported cocontraction measures in relation to old age in the EFs and elbow extensors (EEs) during maximal contractions.

In this study we measured PCSA, MVC, NF, activation, and cocontraction in the EFs and EEs of young and old men. The main purpose was to determine whether the NF of the arm muscles changes with age and, if so, whether the EFs and EEs are affected similarly.

**METHODS**

**Subjects.** Twenty young men, aged 20–29 yr (mean age 22.6 ± 2.7 yr), and thirteen old men, aged 76–95 yr (mean age 81 ± 6.5 yr), volunteered for the study. The young subjects were university students, and the old subjects were living independently in the community. A written questionnaire and interview determined their medical history and the extent of their participation in physical activity. Exclusion criteria for participation in this study included orthopedic or neurological pathologies of the upper or lower limbs, diabetes, previous myocardial infarction, uncontrolled hypertension, alcoholism, medications that affect muscle performance, and extreme physical activity. Most of the subjects were sedentary and had not participated in weight training or other formal exercise program. The purpose, methodology, and possible risks involved in the experiments were explained, and informed written consent was obtained from each subject. The ethics review committee of the University of Western Ontario approved all procedures.

**MRI acquisition.** Within 3 wk of recording the MVC, serial T1-weighted images in the axial plane were acquired with MRI using a 20-cm-diameter extremity coil in a 1.5-T whole body magnet and 64-MHz scanner (Siemens Vision System, Siemens Medical, Erlangen, Germany). Subjects were supine with the left elbow extended and adjacent to the trunk and the forearm in a neutral position. Foam padding was placed under the elbow and forearm, so the arm was parallel to the gantry, and the triceps brachii was not compressed. Rather than encircling the arm, the extremity coil was positioned over the dorsal, lateral, and ventral aspect of the arm or forearm and held in place with Velcro straps that crossed the trunk. By not encircling the arm, the origins of the biceps brachii and triceps brachii could be imaged. Reference markers were placed on the medial epicondyle and mid point of the arm.

The arm was imaged in four segments from the top of the humerus to the wrist by moving the extremity coil three times. Using the software, a grid of 20–24 (depending on arm length) slices was centered on a coronal scout image so the slices were perpendicular to the arm (or forearm). Images were acquired using the following parameters: repetition time, 1,000 ms; echo time, 14 ms; number of acquisitions, 1; matrix, 256 × 256; field of view, 200 mm (2 proximal segments, i.e., arm) and 160 mm (2 distal segments i.e., forearm); slice thickness, 7 mm; and interslice gap, 0 mm (imaging time, 4 min/segment). These parameters were selected to delineate muscle borders and differentiate muscle, fat, and connective tissue. To ensure that the four segments were contiguous, the distance between the marker and the most distal slice of a segment was measured (mm) on the scout image using the software. An ink mark, corresponding to this distance, was placed on the arm so that the extremity coil could be moved the appropriate distance.

**Muscles analyzed.** The biceps brachii, brachialis, and brachioradialis muscles were traced in each image and together made up the EFs. The triceps brachii and anconeus were traced and together made up the EEs. The pronator teres and the extensor carpi radialis longus muscles, which together generate 15% of EF torque (38), were not included in the EF volume because they were not clearly visible in the images over their entire length. The brachioradialis, extensor carpi radialis longus, and brevis muscles were traced as a group. The brachioradialis volume was then estimated to be 39% of the combined volume of these three muscles (4). The length of the brachioradialis, however, could be determined because the origin and insertion regions were clearly evident in all images. The coracobrachialis muscle was traced as part of the biceps brachii because these two muscles could not be separated convincingly throughout their entire length. In both young and old cadavers, the coracobrachialis is 23% of the weight of the biceps brachii plus the coracobrachialis (47). To calculate muscle volume from the ACSA determined in each arm, the biceps brachii volume was reduced by 23% in all subjects.

**Morphological measures.** The same investigator analyzed the images with a software program (Analyze 7.5, Mayo Clinic) and was not blinded to subject identity because it was obvious that images with the smaller muscle size and larger amount of fat were of older subjects. However, the data corresponding to images of subjects previously analyzed were not referred to during subsequent analysis and data were not compiled into the young and old groups until all images were analyzed. The cross-sectional area of muscle, bone, and noncontractile tissue were determined in each image for the entire arm. The regions were either manually traced and/or autotraced. During an autotrace, which is based on different ranges of pixel intensity for different tissues, the operator pointed to the region and then moved a bar to progressively highlight the tissue to its border. The cross-sectional area of noncontractile tissue was always determined with the autotrace feature. The different muscles, bone, and noncontractile tissue that were traced were automatically filled with color-coded maps that could be edited and saved (Fig. 1).

Muscle volume was calculated as the summation of cross-sectional area times slice thickness (7 mm) for all slices along the length of the muscle. Muscle length was the distance between the most proximal and the most distal images in which the muscle was visible. The PCSA was calculated by dividing muscle volume by the estimated fiber (fascicle) length. Muscle fiber length was estimated by multiplying muscle length (from MRI) by the fiber length-to-muscle length ratio determined in cadavers (38). The ratios used were 0.60, 0.49, 0.68, and 0.33 for the biceps brachii, brachialis, brachioradialis, and triceps brachii, respectively (38). It was assumed that the fiber length-to-muscle length ratio was the same in the young and old men because no studies have determined whether fiber length changes with age. No data are available for the fiber length-to-muscle length ratio in the anconeus, so it was assumed to be the same as the biceps brachii because the fibers run parallel in both muscles (3). The PCSA of the triceps brachii was multiplied by the cosine of the fiber pennation angle, assumed to be 15° for both groups, whereas the EFs were assumed to have no pennation (24, 38). The largest ACSA of the biceps brachii + brachialis and triceps brachii is also reported for comparison to previous work. The noncontractile tissue, which was subtracted from the muscle cross-sectional area, consisted of visible pockets of intramuscular fat, connective tissue, and tendon within the muscle’s border. Noncontractile tissue for each
a wooden box, with the knee and hip joints flexed to 90°, and the shoulders were secured by straps firmly against a padded metal brace. The box and brace prevented the torso from sliding during the MVCs. Two additional straps were used to stabilize the upper limb during an MVC of the EEs. One was positioned across the arm ~2 cm proximal to the elbow crease, and the other was placed across the forearm ~5 cm distal to the elbow crease. Once the subject was secured, the position of the box and the length of all straps were recorded for positioning on subsequent visits.

The elbow joint was flexed 100° (180° = full extension), and the forearm was fully supinated to record optimum MVCs of the EFs and EEs (44). The fingers and wrist were prevented from flexing during an EF MVC by a plastic strap that was straped to the back of the wrist and hand. The splint was not necessary during an EE MVC because the subjects kept the fingers extended and in line with the forearm. The ventral (or dorsal, for EEs) aspect of the wrist was secured with a strap to a padded curved bar (11 x 5.2 cm), that had a strain gauge attached (model SST-700-100A, ASTechnology, Haliburton, ON, Canada). The output from the strain gauge was amplified (Neurolog, models NL 107 and NL 126, Digitimer, Welwyn Garden City, Hertfordshire, UK), and converted to digital format by a 12-bit converter (CED model 1401 Plus, Cambridge Electronic Design, Cambridge, UK) at a sampling rate of 500 Hz. Data were stored concurrently on videotape and computer disk and displayed in front of the subject on an oscilloscope. The strain gauge was calibrated periodically by hanging known weights from the brace. The calculated linear relationship (r = 0.99) between the known weights and voltage outputs did not vary throughout the study.

MVC and NF. The subjects did not participate in any activity that required vigorous use of their arms for 48 h before the measurements. The MVCs were determined during each of three visits to the laboratory over 2–4 wk. Three to four MVCs, each held for 4 s with a 3-min rest, were recorded for each of the EFs and EEs during all visits. Strong encouragement was provided during the contractions and visual feedback was provided. The order in which MVCs of the EFs and EEs were recorded was balanced across the three visits and across groups. The force and EMG signals were analyzed off-line using a customized software package (Spike 2, Cambridge Electronic Design). The highest MVC of the three visits was used to calculate muscle activation,
coactivation, and NF. The NF of the EFs or EEs was calculated as the MVC divided by the PCSA of the respective muscle groups.

**Activation level.** A modified twitch interpolation technique was used to assess the level of muscle activation during the MVC (1). The stimulating electrodes were made of aluminum foil, measured 3 × 4 cm to 7 × 4 cm, and were wrapped in gauze and soaked in saline and conducting gel. The cathode was placed over the motor point of the biceps brachii or triceps brachii and the anode over the distal third of the muscle. Using a constant-current stimulator (model DSH7, Digitimer), two doublets (2 pulses at 100 Hz, 50-μs pulse duration) were applied both during and after the MVC. The doublets were triggered manually at the peak of the MVC. The stimulus current intensity for the doublets was 50–60% of the level used to obtain the maximal twitch force. Pilot studies and previous reports (6) found that in the arm muscles, submaximal stimulation resulted in larger interpolated twitches than maximal stimulation. Activation level was calculated as: percent activation = (2 × (T2/T1)) × 100%, where T2 is the force of the interpolated response of the doublet at peak force, and T1 is the mean force of the two post-MVC doublets.

**Coactivation level.** Coactivation during the MVC was determined by recording the EMG activity over the antagonist muscle(s). After the skin was abraded with sandpaper and cleansed with ethyl alcohol, bipolar silver-silver chloride strip electrodes (1 × 3 cm, Marquette Electronics, Milwaukee, WI) were positioned 15 mm apart over the midbiceps brachii, midtriceps brachii, and proximal third of the brachioradialis. The raw EMG signals were amplified (×500) and filtered (between 10 Hz and 10 kHz) using a Neurolog NL284 (Digitimer) preamplifier, amplifier, and filter, and sampled at 2.5 kHz. A 0.5-s period of the EMG was analog corresponding to the point of peak force, which usually occurred within the first 2 s of the MVC. The EMG signals were full-wave rectified and integrated (IEMG) over a 500-ms time constant. Coactivation level was expressed as a percentage of the muscle’s maximal IEMG recorded during the MVC.

The extent of possible cross talk between the biceps brachii and triceps brachii was determined by examining whether M waves were recorded concurrently over both muscles during supramaximal stimulation of the musculocutaneous or radial nerve. A custom-designed bar electrode was used for stimulation and placed over the musculocutaneous nerve just medial to the anterior deltoid at the axilla. For stimulation of the radial nerve, the electrode was placed over medial head of the triceps brachii at the posterior axilla. Because the nerves were not easily accessible in the supine position, the M waves were recorded with the subject seated and the elbow joint flexed 100°. Bipolar electrodes for recording the voluntary EMG were used but were placed on the distal third of the biceps brachii and triceps brachii to record M waves that were uncontaminated by stimulus artifact.

**Statistical analysis.** A two-by-two-factor of ANOVA was used to assess the influence of age (young and old) and muscle (EFs, EEs) on PCSA, ACSA, MVC, and NF. A two-by-three-factor ANOVA determined the effect of age and muscle (biceps brachii, brachioradialis, and triceps brachii) on coactivation level, and post hoc comparisons of the three muscles were done with the Tukey’s test. Muscle activation level was assessed using the Mann-Whitney test because the data were not normally distributed. Pearson’s product-moment correlation coefficients were applied to determine the relationships between MVC and PCSA and between NF, activation, and coactivation. The CV was calculated to determine the reliability of measurements. Analyses were performed using SPSS software (version 10 for Windows). Differences were considered significant when \( P < 0.05 \), and all data are presented as means ± SD.

**RESULTS**

**Anthropometric and morphological measures.** The subjects’ weight, height, and length of the humerus were not different (\( P > 0.05 \)) between the young and old men (Table 1). Muscle volume, excluding noncontractile tissue, was less in the old compared with the young men in the EFs (297 ± 56 vs. 375 ± 54 cm\(^3\)) and EEs (323 ± 62 vs. 444 ± 61 cm\(^3\)) (\( P < 0.001 \)). The results for morphological measures, MVC, NF, and activation are presented in Table 2. The PCSA was 19 and 28% smaller with age in the EFs and EEs respectively (\( P < 0.001 \)), and the decrease was significantly greater in the EEs (age × muscle interaction, \( P = 0.001 \)). The percent decrease in PCSA was the same among muscles that constitute the EFs and EEs. Because age had no effect on muscle length in the EFs or EEs, the lower PCSA in the old men was due to a smaller CSA over the length of the muscle. The ACSA of the biceps brachii + brachialis and triceps brachii were also lower in the old compared with the young men, and the decrease was less (2%) than the decline in PCSA. The relatively larger age-related decrease in PCSA compared with ACSA was significant in the triceps brachii (age × measurement interaction, \( P = 0.01 \)) but not in the biceps brachii + brachialis (\( P = 0.43 \)). The percentage of muscle volume that was noncontractile tissue was greater (\( P < 0.001 \)) with age (Table 2). The PCSA and noncontractile tissue were smaller (\( P < 0.001 \)) in the EFs compared with the EEs in the young and old. In both the young and old groups, the brachialis, biceps brachii, and brachioradialis were 52, 36, and 12%, respectively, of EF PCSA, and the anconeus was 5% of EE PCSA.

**MVC.** The highest MVC on each of the three visits was highly reproducible; the CV was 3 ± 2 and 6 ± 4% in the young and 3 ± 1 and 5 ± 3% in the old men for the EFs and EEs, respectively. The MVC was less (\( P < 0.001 \)) in the EFs (−27%) and EEs (−33%) in the old compared with the young, but the magnitude of the decrease was not different between the muscles (age × muscle interaction, \( P = 0.84 \)) (Table 2). The MVC of the EFs were significantly (\( P < 0.001 \)) greater than the

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<thead>
<tr>
<th>Table 1. Subject characteristics</th>
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<tr>
<td>Age, yr</td>
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<td>Age (yr)</td>
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<tr>
<td>Weight, kg</td>
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<tr>
<td>Height, cm</td>
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<td>Humerus length, cm</td>
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Values are means ± SD, with the range of each value in parentheses; \( n \), is no. of subjects. Except for age, there were no significant group differences in any measurements. Length of the humerus was determined using magnetic resonance imaging.
Table 2. Contractile and morphological measures in the elbow flexors and extensors

<table>
<thead>
<tr>
<th></th>
<th>Elbow Flexors</th>
<th>Elbow Extensors</th>
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<tr>
<td></td>
<td>Young</td>
<td>Old</td>
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<tr>
<td>MVC, N</td>
<td>333 ± 40</td>
<td>241 ± 50‡</td>
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<tr>
<td>PCSA, cm²</td>
<td>28.2 ± 3.5</td>
<td>22.9 ± 4.4‡</td>
</tr>
<tr>
<td>ACSA, cm²</td>
<td>20.2 ± 3.0</td>
<td>16.9 ± 3.2‡</td>
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<tr>
<td>NCT, %</td>
<td>1.0 ± 0.6</td>
<td>4.6 ± 2.4‡</td>
</tr>
<tr>
<td>MVC/PCSA, N/cm²</td>
<td>11.8 ± 1.0</td>
<td>10.5 ± 1.1†</td>
</tr>
<tr>
<td>MVC/ACSA, N/cm²</td>
<td>16.5 ± 1.3</td>
<td>14.3 ± 1.6†</td>
</tr>
<tr>
<td>Activation, %</td>
<td>98.4 ± 1.8</td>
<td>96.9 ± 2.5</td>
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Values are means ± SD for 20 young and 13 old men. The MVC is the highest force produced during a maximal voluntary isometric contraction. Physiological cross-sectional area (PCSA) of the elbow flexors is the biceps brachii + brachialis + brachioradialis. The PCSA of the elbow extensors is the triceps brachii + anconeus. Anatomic cross-sectional area (ACSA) is the largest cross-sectional area of the biceps brachii + brachialis or triceps brachii. Noncontractile tissue (NCT) is expressed as a percentage of the elbow flexor or elbow extensor muscle volume. Normalized force (NF) was determined using the twitch interpolation technique. The age-related reduction in PCSA and ACSA was greater in the elbow flexors than the elbow extensors (significant age × muscle interaction, \( P < 0.001 \)). The NF was significantly lower in the elbow flexors with age. The NF of the elbow extensors tended to be less affected by age (age × muscle interaction, \( P = 0.06 \)). The NF of the elbow flexors significantly decreased with age (main effect for age, \( P < 0.001 \)) with a greater decrease in NF with age in the EFs (11%) compared with the EEs (7%), and the difference between the muscles approached significance (age × muscle interaction, \( P = 0.06 \)) (Table 2, Figs. 3 and 4). The MVC/ACSA also was reduced more in the EFs compared with EEs with age. There were no significant correlations between activation or coactivation and NF in either group or when the data from both groups were combined. The NF of the EFs were greater in both groups compared with the young group (main effect for age, \( P = 0.001 \)). The decrease in NF with age was more prevalent in the EFs (11%) compared with the EEs (7%), and the difference between the muscles approached significance (age × muscle interaction, \( P = 0.06 \)) (Table 2, Figs. 3 and 4).

EEs in both the young and old. There was a significant correlation between PCSA and MVC \(( r = 0.74 )\) in the EFs and EEs in the young and old men (Figs. 3 and 4). When data of both groups were combined the correlation coefficients between PCSA and MVC were \( r = 0.86 \) for the EFs and \( r = 0.87 \) for the EEs \(( P < 0.01 \)). These correlations were larger than the correlations between ACSA and MVC in both the EFs \(( r = 0.83 \) and EEs \(( r = 0.80 )\) for the groups combined.

**Activation and coactivation level.** The level of activation during the MVCs was incomplete \(( < 100\% )\) in the biceps brachii (range 92–100%) and triceps brachii (94–100%) in both groups. The deficit in activation was similar in both groups for the triceps brachii \(( P = 0.13 )\) but tended to be greater \(( P = 0.07 )\) in the biceps brachii of the old compared with the young (Table 2). The level of coactivation in the antagonist muscles during MVCs of the EFs and EEs was \(~ 5\% \) greater \(( P < 0.001 )\) with age (Fig. 5). There was no evidence of cross talk because M waves were not evident in the biceps brachii or triceps brachii during supramaximal stimulation of the radial and musculocutaneous nerves, respectively.

**NF.** The NF was less in the old compared with the young group (main effect for age, \( P = 0.001 \)). The decrease in NF with age was more prevalent in the EFs (11%) compared with the EEs (7%), and the difference between the muscles approached significance (age × muscle interaction, \( P = 0.06 \)) (Table 2, Figs. 3 and 4). The MVC/ACSA also was reduced more in the EFs compared with EEs with age. There were no significant correlations between activation or coactivation and NF in either group or when the data from both groups were combined. The NF of the EFs were greater in both groups compared with the young group (main effect for age, \( P = 0.001 \)). The decrease in NF with age was more prevalent in the EFs (11%) compared with the EEs (7%), and the difference between the muscles approached significance (age × muscle interaction, \( P = 0.06 \)) (Table 2, Figs. 3 and 4). The MVC/ACSA also was reduced more in the EFs compared with EEs with age. There were no significant correlations between activation or coactivation and NF in either group or when the data from both groups were combined. The NF of the EFs were greater in both groups compared with the young group (main effect for age, \( P = 0.001 \)).
with PCSA, the age-related change in NF was the same within the two groups and muscle length was unchanged, the estimated fiber length was not different with age. We are unaware of any studies that have examined whether fiber length or peniation angle is altered with age in humans. A decrease in fiber length in old compared with young rats, however, was reported and attributed to a decline in the number of sarcomeres in-series (18).

As well, the peniation angle is smaller after disuse atrophy (39) and larger in hypertrophied muscles of young subjects (24). Thus, if fiber length and peniation angle are less with age in humans, the PCSA would be underestimated and NF overestimated in our old compared with young men.

**MVC, activation, and coactivation.** The MVC was less (~30%) in the EEs and EEs in the old compared with the young men. Others have reported similar (~9, 10) or smaller (~20%) (36) losses of EF strength with age, but no previous data on the isometric strength of the EEs are available. A primary question of the present study was whether there is an age-related change in the neural drive to the agonist and antagonist muscles and, if so, what effect this would have on NF. Neural drive to the agonists was estimated by measuring the activation level of the biceps brachii and triceps brachii using the twitch interpolation technique. Similar to previous work (9, 49), our results indicate that activation was incomplete (~100%) and tended to be less in the EFs with age, although this age difference was not significant. A deficit in activation in the old men could imply that the neural drive (i.e., motor unit recruitment and/or firing rate) is less during strong contractions and this could contribute to the lower NF of the EFs. However, there was no correlation between activation (range of 94–100%) and NF in either the young or old men. Although the age-related difference in activation was small and not significant, larger differences may occur during dynamic compared with isometric contractions. For example, the IEMG of the vastus lateralis tended to be reduced more with age during the standing long jump than during isometric maneuvers (15). Although Herbert and Gandevia (17) have questioned whether twitch interpolation is a sensitive measure of motoneuron excitation, a larger impairment in activation with age may become apparent if stimulation is applied during dynamic movements.

Neural drive to the antagonist muscles during the MVC was estimated by measuring the level of coactivation in the biceps brachii, brachioradialis, or triceps brachii. Coactivation was higher (~5%) in all muscles
of the old compared with the young men. Age-related increases in coactivation were previously reported in the biceps femoris during downward stepping (19) and during maximal dynamic and isometric contractions (15, 22), although coactivation was not different with age during submaximal contractions of the EFs (13). Because the MVC represents the net force about the joint (i.e., agonist minus antagonist forces), the greater coactivation in the old could contribute to the lower NF in the EFs. There was, however, no correlation between coactivation and NF in either group. For example, NF of the EFs was not lower than the group average in the two old subjects with the highest coactivation in the triceps brachii (32 and 35%). Thomas and colleagues (45) also reported no significant relationship between EE MVC and biceps brachii coactivation levels of 15–50% in controls and subjects with spinal cord injuries. It may be difficult, however, to derive a significant correlation between coactivation and NF because these measures are affected by many factors. Although greater coactivation may reduce NF, it may also prove beneficial for the elderly by increasing joint stiffness and postural stability during movement (19).

NF. The NF was 11% lower in the EFs and tended to be less affected in the EEs with ageing. As mentioned previously, the lower NF in the EFs could stem from an increase in coactivation. Other factors, however, such as a decline in specific tension of the muscle fibers could also contribute to a decrease of NF (11). It is unclear why NF in the EEs is less affected than the EFs with old age. One possible explanation is a reduction in fiber pennation angle in the triceps brachii, secondary to muscle atrophy, compensates for any age-related changes in coactivation and specific tension, by transmitting relatively more tension to the tendon during a contraction (31, 39). In young athletic subjects, the pennation angle of the triceps brachii is positively correlated with muscle size (24) and negatively correlated with NF of the EEs (21). It is interesting that Frontera and co-workers (11) reported a decrease (30%) in specific tension of single fibers but no change in NF in the quadriceps with age. This paradoxical finding may be a reflection of age-related changes in muscle architecture and connective tissue (43) that sustains NF by enhancing force transmission in vivo (20).

In previous studies, investigators found either no change in NF with age (11, 26, 40) or a decrease of up to 40% (8, 23, 27, 32, 46, 48). Similar to our findings, Klittgaard et al. (27) reported a 14% decrease in isometric NF (MVC/ACSA) in the EFs of old (68 yr) compared with young men when computed tomography was used to measure ACSA. As well, in these same subjects, the age-related decrease (27%) in NF of the knee extensor muscles was larger than in the EFs (27). Young and co-workers (48) also reported a 27% decline in isometric NF of the knee extensors with age using ultrasound imaging. However, other investigators reported no change in isometric NF with age in the knee extensors using computed tomography (11, 40) or in the ankle dorsiflexors using MRI (26). An age-related decline in NF during dynamic (isokinetic) contractions also has been reported (23, 32, 40). For example, Overend et al. (40) found that isokinetic NF, but not isometric NF, of the knee extensors and flexors was 10–20% lower in men aged 70 yr compared with 20-yr-old men. Jubrias and co-workers (23) used MRI and reported a 21% decrease in NF of the knee extensors at contraction speeds of 120°/s and greater, but not at 60°/s, in 42 men and women between the ages of 65 and 80 yr. In a large sample of men aged 19–91 yr, Lynch and co-workers (32) reported a decrease in isokinetic (30–45°/s) NF of 28 and 40% in the arm (EFs plus EEs) and thigh (knee flexors plus knee extensors) muscles, respectively, using dual-energy X-ray absorptiometry (DXA). The prevalence of a decline in isokinetic NF with age evident in previous studies, particularly at faster contraction speeds, may be due to a number of factors, including a decrease in fast-twitch fiber area (30), a decline in muscle activation, and increased coactivation (15, 22).

The extent of the age-related decline in NF demonstrated in previous work may be related to the method used to measure muscle size as well as the general health, motivation, and physical activity level of the subjects. In the present study the subjects were equally motivated to give their best effort during the MVCs. This conclusion is based on the small day-to-day variability of the MVC and activation level. As well, the subjects were healthy and not highly trained and the nondominant arm was measured in all cases. Thus the group differences in PCSA, strength, and NF are more likely related to the effects of aging per se rather than to differences in physical activity level, although we have no direct measures of activity level. More importantly, the large age-related deficits (27–40%) in NF reported in some studies (8, 32, 46) could be exaggerated because the techniques that were used may not have adequately distinguished muscle and noncontractile tissue. Because the noncontractile tissue is increased in the elderly (43), ultrasound (46), DXA (29, 32), and less direct techniques (8) may overestimate muscle size and thereby underestimate NF. For example, Lynch et al. (32) reported a 20% decrease in arm and thigh muscle mass between young and old (>80 yr) men with DXA. In the present study, we found a 20–28% decrease in EF and EE PCSA and Lexell et al. (30) reported a 45% decrease in the ACSA of the vastus lateralis muscle in cadavers across the same age range. A 45% decrease in ACSA would be enough to account for all of the strength loss commonly reported in the knee extensors (41). The age-related deficit (15–25%) in specific tension of whole animal muscle may also be exaggerated because noncontractile tissue was not accounted for in the ACSA measurements (7, 28). Ansved and Larsson (5), however, measured specific tension with (ACSA minus total fiber area) and without (muscle wet weight) correction for noncontractile tissue in young and old rats. Specific tension was reduced by 20% in the old rats when wet weight was used but was unaffected when noncontractile tissue was accounted
for. Thus it may be that NF and whole muscle specific tension during isometric contractions are not greatly impaired when adequate techniques are used to measure muscle size.

The EEs were ~10–20% weaker than the EFs in both groups despite having almost twice the PCSA. This resulted in a lower NF in the EEs compared with the EFs by almost 50%. Part of the difference in NF between the muscle groups is because the pronator teres and the extensor carpi radialis longus muscles, which can function as elbow flexors, were not included in the EF volume. Consequently, NF of the EFs was likely overestimated by ~15% in the present study (38). A more important factor contributing to the lower NF in the EEs compared with the EFs is the smaller moment arm length of the triceps brachii compared with the EEs (38). We have since confirmed using MRI that the moment arm length of the triceps brachii is approximately one-half that of the EFs in young and old men and that the specific tension of the two muscle groups is similar (25).

In conclusion, isometric strength and PCSA of the EFs and EEs were reduced in old compared with young men. The percent loss in strength and PCSA with old age was similar in the EEs resulting in an unchanged NF for this muscle group. In contrast, since strength was reduced more than PCSA, the NF of the EFs was reduced more than PCSA, the NF of the EFs was reduced in old compared with young men. The percent loss in strength and PCSA with old age was similar in the EEs resulting in an unchanged NF for this muscle group. In contrast, since strength was reduced more than PCSA, the NF of the EFs was reduced in old compared with young men. Possible mechanisms that may contribute to the lower NF of the EFs include an increase in coactivation, or a decline in specific tension of the muscle fibers.

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