Peripheral impairments cause a progressive age-related loss of strength and velocity-dependent power in the dorsiflexors

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McNeil CJ, Vandervoort AA, Rice CL. Peripheral impairments cause a progressive age-related loss of strength and velocity-dependent power in the dorsiflexors. J Appl Physiol 102: 1962–1968, 2007. First published February 15, 2007; doi:10.1152/japplphysiol.01166.2006.—Muscle power is more functionally relevant than static muscle strength, particularly with aging. However, the effect of age on power derived from isotonic contractions has been studied sparingly, and it has not been studied at all in subjects >75 yr of age. Thus the purpose was to investigate the magnitude and causes of age-related losses in isotonic power among 13 young (26 yr), 13 old (65 yr), and 13 very old (84 yr) men. Six different loads were employed to create velocity-torque and power-torque relationships. Dorsiflexor cross-sectional area was assessed via magnetic resonance imaging for the calculation of specific power. Electromyographic signals of the tibialis anterior and soleus muscles were recorded to assess agonist activation and antagonist coactivation, respectively. Despite similar contractile masses and levels of voluntary drive and antagonist co-activation, power was significantly impaired in the old vs. young (~25%), and in the very old relative to both the young (~60%) and old (~40%). The novel results punctuate two important considerations for studies concerned with the effect of age on the neuromuscular system. First, the decreased ability of muscles from old men to produce power in the presence of reasonably well-preserved strength indicates the utility of studying isotonic contractions. Second, the precipitous decline in many measures between the seventh and ninth decades underscores the benefit of testing more than one group of aged subjects to improve our understanding of rates of change in key variables.

IN ADDITION TO A DECLINE in strength, aging is associated with a loss of voluntary contractile velocity which, when combined with the loss of absolute torque output, results in great reductions in power (5, 15, 20, 24, 29). Whereas strength capacity is most easily assessed from static (isometric) contractions, power is measured from more complex dynamic movements that involve both strength and velocity of contractions. Thus it has been suggested that power is a more inclusive measure of overall age-related changes to the neuromuscular system (32).

By means of a dynamometer, power is determined typically by holding either the load or the velocity constant while the other variable is measured. Because activities of daily living involve the movement of constant loads through a range of motion (ROM) at variable velocities (isotonic), rather than the movement of variable loads at fixed velocities (isokinetic), isotonic measures of power are more functionally relevant. Despite this understanding, there are few studies that explore age-related changes to muscle power during isotonic (velocity-dependent) contractions (5, 15, 20, 24, 29).

Although altered neural drive (e.g., decreased agonist activation or increased antagonist coactivation) may play a role in the effect of aging on some muscle groups and complex movements, the decline in strength with advancing age is attributed primarily to the loss of muscle mass (1, 9, 12). There are numerous studies concerning the influence of age on strength per contractile cross-sectional area (CSA; specific strength), and although the results are equivocal, many recent studies would suggest that specific strength does decrease with age (5, 7, 12, 16, 21, 31). In contrast to specific strength, there has been little investigation of specific power, and the results of the few studies unequivocally indicate an age-related decrease (2, 17, 18, 23, 28). Of these studies, only the recent one by Thom and colleagues (28) isolated a single muscle group (plantar flexors) in the assessment of muscle power and muscle volume. In the other studies (2, 17, 18, 23), a multijoint movement was performed and the total muscle volume included antagonist muscles that were not involved in power generation.

It is logical that losses in power, like strength, will accelerate with age, and thus it is of interest to assess muscle power in elderly subjects at an age when the risks of falls and loss of independence are high. However, with the exception of our recent fatigue study (20), the elderly participants involved in the previous velocity-dependent studies represent the "young" elderly, i.e., <70 yr of age (5, 15, 24, 29). For some muscle groups, this age range may be too young to effectively characterize power loss. For example, numerous studies on the dorsiflexors have reported that strength and contractile kinetics are not significantly different between young individuals and those in their seventh and eighth decades (13, 19, 20, 26).

Indeed, we reported that dorsiflexor strength and contractile kinetics were preserved between the third and seventh decades despite motor unit (MU) remodeling and a 40% loss in the number of MUs in the tibialis anterior muscle (19). It was only following further MU remodeling and MU loss (an additional 33% from the seventh decade) that an age-related decrease in strength and a slowing of contractile kinetics were realized early in the ninth decade (19). Although strength and contractile kinetics were maintained, it is uncertain whether muscle power also would be resistant to change during four decades of MU remodeling and loss. Moreover, the rate of decrease in

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velocity and power between the seventh and ninth decades is unknown. Thus it was the purpose of this study to characterize the effects of aging on velocity and isotonic power of the dorsiflexors by comparing young subjects to old and very old subjects in their seventh and ninth decades, respectively.

**MATERIALS AND METHODS**

**Subjects.** Thirteen young men (aged 19–33 yr), 13 old men (aged 60–69 yr), and 13 very old men (aged 80–90 yr) volunteered for this study. Young subjects were recruited from the university environment and considered to be recreationally active. Subjects in the two elderly groups were recruited from a local exercise program designed to maintain cardiovascular fitness, flexibility, and muscular endurance. All subjects were healthy with no evidence of neuromuscular disease. The mean age, height, and body mass of the three groups are reported in Table 1. The study protocol was approved by the local university’s ethics review board and was conducted in accordance with its guidelines for experimentation on human subjects and conformed to the Declaration of Helsinki. Informed, written consent was obtained from each of the 39 participants. Dynamometer data were collected during the second of two visits to the neuromuscular laboratory; the first visit was to familiarize subjects to the experimental procedures. Muscle mass data were collected on a subset of 24 participants (8 per age group) during a single visit to the imaging unit.

**Experimental setup.** Exercise sessions (familiarization and test) were performed on the right leg in a Biodex multijoint dynamometer (System 3, Biodex Medical Systems, Shirley, NY). Subjects sat in a reclined position with angles of ~90 and 160° at the hip and knee, respectively, and their leg was aligned parallel to the ground. Isometric dorsiflexion contractions were performed at an ankle angle of 25° of plantar flexion. Isotonic dorsiflexion contractions began at the plantar flexed position and ended at a neutral ankle position (0°) such that the ROM was 25°. A Velcro strap across the right thigh, an adjustable seat belt across the waist, and shoulder straps minimized hip flexion and upper body movement. Velcro straps across the toes and the dorsum of the foot secured the limb to the dynamometer footplate.

Bipolar surface electromyography (EMG) signals were recorded from the tibialis anterior (TA) and the soleus muscles with self-adhering Ag-AgCl electrodes (1.5 × 1 cm; Kendall-LTP, Chicopee, MA). The skin was abraded and swabbed with alcohol before application of the electrodes. Using a 2-cm interelectrode distance, electrode pairs were placed in a longitudinal arrangement over the belly of the TA, and also on the soleus, distal to the medial head of the gastrocnemius.

**Experimental procedures.** Data collection began with the determination of the maximum twitch torque via supramaximal stimulation delivered over the peroneal nerve, distal to the fibular head. The amplitudes of the twitch and the M wave were monitored as the current intensity was increased incrementally. When both parameters had reached a plateau, the current was increased a further 15% to be certain that stimulation was supramaximal. Subsequently, three isometric maximal voluntary contractions (MVCs) of the dorsiflexors were performed with 2 min of rest separating each attempt. Subjects held each MVC for 3–5 s, during which time they were provided with visual feedback of torque via an oscilloscope, and they received strong verbal encouragement. Voluntary activation was assessed on each MVC attempt by use of the modified interpolated twitch technique (2 pulses at 100 Hz). The torque amplitude of a supramaximal doublet (T2) delivered during the MVC was compared with a resting doublet (T1) delivered after the MVC to quantify voluntary activation [% activation = \(1 - \frac{T2}{T1}\) × 100%].

After determination of the isometric MVC torque, the dynamometer was switched from the isometric to the isotonic mode. In this mode, a resistance load can be programmed that the dynamometer attempts to hold constant while velocity is measured throughout a ROM. Current dynamometers are unable to maintain a constant load, so movements made in the isometric mode are not strictly isotonic (15). However, for the purposes of this study, the term isotonic will be used throughout the remainder of this paper to describe this movement type. It is worth noting that this discrepancy is not relevant to the aim or results of this study because the movements are still velocity dependent and therefore more applicable to physical function and aging than isometric or isokinetic movements (as noted in the introduction).

To investigate the relationship between load and velocity and load and power, subjects performed maximal (as fast as possible) isotonic contractions at a variety of submaximal loads. These loads were normalized to each subject’s MVC (10, 20, 30, 40, and 50% MVC) to account for differences in isometric strength between individuals. Subjects also performed contractions at a minimum absolute load that represented the resistance of the dynamometer footplate and the lowest possible additional load (1 N•m). The order in which the loads were performed was determined for each subject via a randomized draw. Two contractions were completed at each load with 5 s of rest between each contraction and 1 min of rest between each load. Subjects received visual feedback of velocity via an oscilloscope and were strongly encouraged to contract as explosively as possible on each attempt. An index of voluntary activation was obtained during the isotonic contractions by normalizing agonist (TA) EMG to the M wave.

**Measurement of muscle cross-sectional area.** Anatomical CSAs of the dorsiflexors were acquired via axial serial plane scans in a 3.0-T superconducting magnet (IMRIS, Winnipeg, Canada). T1-weighted images were acquired using the following parameters: repetition time, 850 ms; echo time, 21 ms; matrix, 512 × 512; field of view, 250 mm; slice thickness, 7 mm; and slice separation, 3 mm. Subjects were supine and inserted into the bore of the magnet to waist level. To position the right leg in the middle of the coil, and thereby maximize the image quality, the right foot was crossed over top of the left foot. A series of 10 slices were obtained and centered on a plastic capsule filled with vegetable oil taped to the right leg over the muscle belly of the TA. The slice with the largest CSA was selected for further analysis. A representative scan for each age group is displayed in Fig. 1.

**Data reduction and statistics.** Torque, velocity, and ankle position data were recorded by the Biodex at a sampling rate of 100 Hz. Using a 12-bit analog-to-digital converter (model 1401 Plus, Cambridge Electronic Design, Cambridge, UK), EMG, torque, and position data were sampled online (at 2,500, 100, and 100 Hz, respectively) with Spike 2 software (version 4.13, Cambridge Electronic Design). Peak values for isometric torque (N•m) and the isotonic velocities (°/s) of the six submaximal loads were recorded from the Biodex display during the experiment. During offline analysis, isotonic power was calculated for the submaximal loads as the product of torque (N•m) and velocity (rad/s). Spike2 software was used to determine the maximum rate of torque of development (RTD) during the isometric MVC, evoked twitch torque, time to peak torque (TPT), and half relaxation time (HRT), M-wave amplitude, agonist (TA) and antagonist (soleus) EMG, and ROM during the isotonic contractions. The RTD was obtained from the first derivative of the torque signal. Root-mean-square values of the EMG signals were calculated for a
0.5-s interval about the peak torque during isometric contractions and for the interval between the initiation of movement and the achievement of the end point of the ROM during isotonic contractions. Levels of antagonist coactivation were calculated as the ratio between soleus and TA root-mean-square EMG (11, 25). Analyze software (version 7.0, Mayo Clinic, Rochester, MN) was used to determine contractile, noncontractile, and total CSAs of the dorsiflexors. A single investigator performed all analyses and employed a combination of auto-trace functions and manual-trace adjustments as necessary. Noncontractile CSA was divided by total CSA to determine the relative percentage of noncontractile tissue in the dorsiflexors.

Using SPSS software (version 11), a univariate ANOVA was used to assess isometric MVC torque, isometric antagonist coactivation, isometric RTD, twitch torque, TPT, and HRT, M-wave amplitude, contractile CSA, noncontractile CSA, total CSA, and the relative percentage of noncontractile tissue. A two-way repeated-measures ANOVA, with age as one factor for comparison and load as the other, was used to compare changes in velocity, power, agonist activation, and antagonist coactivation at the submaximal loads. All data are reported in the text as group means, and the level of significance was *P < 0.05. When a significant main effect or an interaction was found, Tukey’s honestly significant difference post hoc tests were performed to indicate where significant differences existed. In the tables and figures, a significant change from young is denoted by an asterisk (*) and a significant change from both young and old is denoted by a dagger (†).

RESULTS

Isometric measures. The dorsiflexors of very old men were significantly weaker than those of both young and old men (33 and 25% deficits, respectively), with no significant difference (11%) between the young and old men (Table 2). Voluntary activation of the dorsiflexors, as assessed by the interpolated twitch technique, was complete in each of the three age groups. Coactivation of the soleus muscle during dorsiflexion MVCs was similar among the age groups (14, 12, and 10% for the young, old, and very old men, respectively). The maximum RTD during the MVC slowed with age (Table 2). Old men produced a rate ~17% slower than young men, whereas very old men produced rates ~47 and ~36% slower than young and old men, respectively.

M-wave amplitude and maximal evoked twitch torque were similar among the three groups (Table 2). Twitch TPT was significantly prolonged in both old and very old men compared with young men (23 and 30%, respectively; Table 2). In contrast, HRT of the twitch was similar between young and old but significantly prolonged in very old men compared with both young and old men (35% and 27%, respectively; Table 2).

Dynamic measures. Young men achieved the full 25° ROM at all loads with the exception of 50% MVC (mean of 24°; range 22–25°). Old men demonstrated a similar small loss of ROM at 50% MVC (mean of 24°; range 15–25°) but also at 40% MVC (mean of 24°; range 20–25°). ROM was incomplete at 30% MVC (mean of 24°; range 15–25°), 40% MVC (mean of 22°; range 10–25°), and 50% MVC (mean of 22°; range 8–25°) in the very old men. Although there was a progressively earlier and greater failure of ROM with age, these differences were not statistically significant. Soleus coactivation did not differ among the age groups during maximal isometric efforts, nor was there a systematic increase or decrease in antagonist coactivation with increasing load (ranges between 11 and 13%, 10 and 12%, and 8 and 10% for the young, old, and very old men, respectively). Similarly, voluntary activation, assessed by the ratio of TA EMG normalized to the M wave, was unaffected by age or load (ranges between 0.13 and 0.14 for young and old men and 0.16 and 0.18 for very old men).

Absolute velocity developed by the dorsiflexors was significantly reduced with age. Old men were significantly slower than young men at all loads but 30% MVC (Fig. 2). The reduction in velocity, relative to the young men, ranged from 11% at 1 N-m to 24% at 50% MVC. Very old men were significantly slower than young men at all submaximal loads, and they were slower than old men at all loads except 50% MVC (Fig. 2). Deficits in velocity for the very old men ranged from minima of 27 and 17% (10% MVC) to maxima of 41 and 29% (40% MVC) compared with young and old men, respectively. The relative loss of velocity with increasing load was similar between groups such that there were no age-related differences in a velocity-load relationship normalized to the lightest load, i.e., 1 N-m (data not illustrated).

Table 2. Voluntary and evoked neuromuscular properties of the dorsiflexors

<table>
<thead>
<tr>
<th></th>
<th>Young (n = 13)</th>
<th>Old (n = 13)</th>
<th>Very Old (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVC torque, N-m</td>
<td>49.1 (6.6)</td>
<td>43.8 (6.3)</td>
<td>33.1 (5.6)†</td>
</tr>
<tr>
<td>MVC RTD, N-m/s-1</td>
<td>297.3 (55.2)</td>
<td>247.4 (53.7)*</td>
<td>158.1 (35.2)†</td>
</tr>
<tr>
<td>Twitch torque, N-m</td>
<td>5.0 (1.2)</td>
<td>4.9 (0.8)</td>
<td>4.5 (1.2)</td>
</tr>
<tr>
<td>Twitch TPT, ms</td>
<td>92.3 (19.6)</td>
<td>113.6 (15.7)*</td>
<td>119.7 (15.9)*</td>
</tr>
<tr>
<td>Twitch HRT, ms</td>
<td>107.1 (18.0)</td>
<td>113.7 (14.1)</td>
<td>143.9 (26.7)†</td>
</tr>
<tr>
<td>M-wave amplitude, mV</td>
<td>3.3 (1.0)</td>
<td>2.7 (1.0)</td>
<td>2.4 (1.0)</td>
</tr>
</tbody>
</table>

Values are means (SD); n, no. of subjects. Maximum voluntary contraction (MVC) torque was significantly lower in very old than young and old men (†P < 0.05). Maximum voluntary contraction rate of torque development (MVC RTD) was slower in old than young men (P < 0.05) and slower in very old men compared with both young and old men (P < 0.05). Twitch time-to-peak torque (TPT) was prolonged in both old and very old compared with young men (P < 0.05). Twitch half relaxation time (HRT) was slower in very old than both young and old men (P < 0.05).
MVC († men generated less power than both young and old men at 20, 30, 40, and 50% MVC than both young and old men at all other loads († P < 0.05). The deficits in power for the very old men ranged from with the young and old men at 20, 30, 40, and 50% MVC (Fig. 3). The reduction in power between men generating significantly less power than young men at 30, 40, and 50% MVC (Fig. 3). The reduction in very old men compared with young and old men (Fig. 4). In contrast, power

Despite a similar MVC torque, the loss of velocity led to old men generating significantly less power than young men at 30, 40, and 50% MVC (Fig. 3). The reduction in power between young and old men ranged from 11% at 1 N·m to 31% at 50% MVC. Power was significantly less for the very old compared with the young and old men at 20, 30, 40, and 50% MVC (Fig. 3). The deficits in power for the very old men ranged from minima of 30 and 22% (1 N·m) to maxima of 60 and 47% (40% MVC) compared with young and old men, respectively.

Muscle CSA measures. Contractile CSA and total CSA of the anterior compartment were similar among the three age groups (Table 3). Noncontractile tissue, expressed as absolute CSA or as the percentage of the total CSA, increased significantly and progressively with age (Table 3). Strength normalized to the contractile CSA of the anterior compartment (specific strength) was similar between young and old men (15% reduction in old men), and between old and very old men (11% reduction in very old men), but significantly less (25%) in the very old compared with young men (Fig. 4). In contrast, power normalized to contractile CSA (specific power) was significantly less (25%) in old than young men, and it was lower in the very old men than both the young (51%) and old (35%) men (Fig. 4).

DISCUSSION

Despite reasonably well-preserved contractile muscle mass, isotonic power of the dorsiflexors was markedly impaired with advancing age. Substantial decreases in velocity led to reductions in power that were two to three times the magnitude of losses in strength. A decreased ability to generate power in the seventh decade, in the presence of isometric strength levels similar to young adults, illustrates the value of isotonic vs. isometric testing with aging. Moreover, the accelerated declines of strength, velocity, and power between the seventh and ninth decades demonstrate the importance of testing more than one narrow age group of elderly subjects in these types of cross-sectional studies, which are obligatory when one is interested in changes that may span 50 yr or more.

Strength. Similar to our previous findings (19, 20), maximum dorsiflexor strength was preserved in the seventh decade but reduced in the ninth decade. Our results, used in conjunction with studies that reported maintained dorsiflexor strength in the eighth decade (13, 26), would suggest that the loss of strength occurs either at the end of the eighth or at the beginning of the ninth decade. Age-related loss of strength is typically ascribed to some combination of a decrease in agonist activation, an increase in antagonist coactivation, a decrease in

Table 3. Anterior compartment cross-sectional areas and percent noncontractile area

<table>
<thead>
<tr>
<th></th>
<th>Young (n = 8)</th>
<th>Old (n = 8)</th>
<th>Very Old (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contractile, cm²</td>
<td>14.3 (2.0)</td>
<td>14.6 (2.7)</td>
<td>12.9 (1.5)</td>
</tr>
<tr>
<td>Noncontractile, cm²</td>
<td>0.9 (0.3)</td>
<td>1.5 (0.4)</td>
<td>2.1 (0.5)</td>
</tr>
<tr>
<td>Total, cm²</td>
<td>15.2 (2.0)</td>
<td>16.1 (2.8)</td>
<td>15.9 (1.7)</td>
</tr>
<tr>
<td>% Noncontractile area</td>
<td>5.7 (2.0)</td>
<td>9.3 (2.7)</td>
<td>14.3 (3.2)</td>
</tr>
</tbody>
</table>

Values are means (SD); n, no. of subjects. Noncontractile tissue, in absolute units and expressed as a percentage of the total cross-sectional area, was larger in old than young men (*P < 0.05), and larger in very old men compared with both young and old men (†P < 0.05).
muscle mass, and a decrease in strength per contractile CSA (specific strength). Our results suggest that reduced specific strength is primarily responsible for age-related loss of strength in the dorsiflexors. In keeping with recent findings in the dorsiflexors, agonist activation, using interpolation techniques, was maximal (11, 13, 19, 20, 26, 31) and antagonist coactivation, assessed by EMG, was equivalent (11, 26) in each age group. However, in contrast to two earlier studies (9, 10), dorsiflexor contractile CSA was not significantly reduced with age. The age-related maintenance of muscle mass is most likely due to the fact that the elderly participants in our study were physically active, whereas those in the two studies by Kent-Braun and colleagues (9, 10) were sedentary. Because contractile CSA was similar among our age groups yet MVC torque was reduced in the very old, specific strength was maintained in the old men but significantly less in the very old than young men. Similarly, Kent-Braun and Ng (9) reported that dorsiflexor specific strength was maintained in elderly adults with a mean age of 72 yr. Our results, together with those of Kent-Braun and Ng (9), suggest that significant deficits in specific strength do not present themselves until toward the end of the eighth or the beginning of the ninth decade.

In addition to peak torque, the maximum RTD was assessed during the isometric MVC. Unlike strength, significant impairments to RTD were present in old compared with young men, and they were present in very old compared with both young and old men. The values for young and old matched closely with the previously published results for this muscle group in men of similar ages (means of 23 and 70 yr, respectively; Ref 27). Our results from men nearing the middle of the ninth decade extend these observations and provide further evidence of a more precipitous decline in dorsiflexor function beyond the age of 80 yr.

Velocity. Similar to RTD, angular velocity measured from the isotonic contractions was significantly reduced between young and old men in addition to the impairment present in very old relative to young and old men. Although absolute velocities were significantly slower with age, each group presented a similar pattern of decline in velocity for each increase in torque (Fig. 2). Only the modest loss of velocity between 1 N-m and 10% MVC in the very old men was visibly different among the groups when comparing the pattern of decline. This difference was the product of the lower strength present in the very old men; i.e., the minimum load of 1 N-m represented a greater percentage of MVC than in the young and old men, so there was a smaller increase in torque between the 1-N-m load and 10% MVC.

Testing of the central motor control influences on the age-related loss in velocity during maximum isometric efforts presents a methodological challenge. During isotonic contractions, torque is fixed by the dynamometer rather than determined by subject effort, and therefore voluntary activation cannot be assessed by existing interpolation methods. To obtain some index of activation, we normalized TA EMG during the isotonic contractions to the M wave, and we found that this ratio of neural efficiency was unaffected by age. The preservation of voluntary activation under isometric conditions supports the isometric findings of this and other studies (11, 13, 19, 20, 26, 31), and it supports the isokinetic findings of Klass and colleagues (11) at a variety of concentric and eccentric velocities. We also assessed antagonist coactivation during isotonic contractions, and we found that, similar to the isometric efforts, levels of soleus coactivation were equivalent among age groups; this is a finding supportive of other recent studies involving dynamic exercise (11, 29). Thus, although the development of more direct or sensitive measures of neural drive during isotonic contractions may yield different results, the current findings suggest that age-related impairments to velocity reside at the periphery and not the central nervous system.

Although derived from isometric contractions, slowed evoked contractile properties are common indexes of changes intrinsic to the muscle. Similar to many other reports on the dorsiflexors (11, 13, 19, 20, 26, 31), twitch TPT, and HRT were prolonged with age in the present study. Potential alterations at the periphery responsible for the decrease in contraction velocity include increased percentages of type I muscle fibers (28, 29), increased expression of slow myosin heavy chain (MHC) isoforms (28, 29), increased internal drag produced by increased connective tissue in the muscle and structural changes to the myosin filaments (29), decreases in fiber fascicle length due to a reduction in the number of sarcomeres in series (22), and alterations to the excitation-contraction complex (7, 11). Although there is some experimental evidence to support age-related increases in the percentage of type I muscle fibers (6, 14) and the relative proportion of slow MHC isoforms (3, 23), the impact of these factors would be relatively minor in the dorsiflexors considering the predominance (~80%) of type I fibers in the young (4, 6).

Power. Similar to velocity, there was a significant and progressive loss of power with advancing age. However, because of the combined losses of both strength and velocity, the deficits in power were in excess of either factor as assessed individually, particularly in the very old. The inclusion of two groups of elderly men in this study provided two important findings with respect to muscle power. First, the load at which peak power was achieved also decreased progressively with age. That is, the young men did not achieve peak power until the 50% MVC load, whereas old and very old men did so at 40 and 30% MVC, respectively. Second, the relative contributions of the losses in strength and velocity were also influenced by age. Because absolute strength level was relatively well maintained between the third and seventh decades of life, it was decreased velocity that was more responsible for the reduced power generation present in the old men. In contrast, in a comparison between young and very old, the loss of power was due to nearly equal deficits in strength (33%) and velocity (27–41% at the various loads).

In the manner that power losses greatly exceeded strength losses, specific power was decreased to a greater extent (nearly twofold) than specific strength in both old and very old men. In the other study to determine specific power using only the agonist muscle volume, Thom and colleagues (28) reported a 55% decrease in plantar flexor specific power in men between 27 and 74 yr of age. This figure is in excess of the 25 and 51% deficits present in our old and very old men, respectively. The greater deficit in specific power reported in the study by Thom and colleagues is most likely due to the fact that 1) the plantar flexors undergo age-related alterations that are greater in magnitude and more rapid in onset than the dorsiflexors (26, 33); and 2) they utilized muscle volume in the calculation of specific power, a more precise and appropriate measure than...
the simpler anatomic CSA used in the present study (28). A third potential factor that may have had a small influence is the difference in the mode of contraction, i.e., isokinetic vs. isotonic.

The progressive and extensive reduction in power reported in this study highlights the substantial risk of limited functional mobility in very old individuals. The dorsiflexors are an important muscle group in the gait cycle, and a decrease in their performance has been associated with an increased risk for falls (8, 30). Although a 60% decrease in muscle power by the ninth decade of life is substantial, it should be noted that those individuals involved in this study were all participants in physical activity programs. In a sample of subjects more representative of a typical man in, or beyond, his ninth decade, the loss of power likely would exceed 60%. This point may be illustrated by the results obtained from the oldest subject (91 yr) tested in this study (note: data not included in group means). Despite his participation in an exercise program three times per week, his dorsiflexor strength was reduced to 15 N·m, and, more importantly, he was unable to generate sufficient power to move the lightest load employed (1 N·m) beyond two-thirds of the desired 25° ROM.

In conclusion, although isometric muscle strength was relatively well preserved with age in the dorsiflexors, the ability to generate power was markedly impaired during maximal isometric contractions. Whereas the initial decrease in power was gradual (~25% between the third and seventh decades), the impairment doubled in the next two decades such that men in their ninth decade of life produced ~60% less power than young men. A decrease in velocity of movement achieved was the predominant factor in the loss of power between the young and old, but strength and velocity were equally responsible for the power deficit present in the very old. Age-related reductions in specific strength and specific power indicated that muscle mass alone could not account for the losses of strength and power. Evidence suggests that age-related impairments in dorsiflexor function develop at the muscle rather than in the noncontractile components in young and older women and men. J Appl Physiol 88: 662–668, 2000.


