INVITED REVIEW

ABSTRACT: Skeletal muscle architecture is the structural property of whole muscles that dominates their function. This review describes the basic architectural properties of human upper and lower extremity muscles. The designs of various muscle groups in humans and other species are analyzed from the point of view of optimizing function. Muscle fiber arrangement and motor unit arrangement is discussed in terms of the control of movement. Finally, the ability of muscles to change their architecture in response to immobilization, eccentric exercise, and surgical tendon transfer is reviewed. Future integrative physiological studies will provide insights into the mechanisms by which such adaptations occur. It is likely that muscle fibers transduce both stress and strain and respond by modifying sarcomere number in a way more suited to the new biomechanical environment.
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FUNCTIONAL AND CLINICAL SIGNIFICANCE OF SKELETAL MUSCLE ARCHITECTURE

RICHARD L. LIEBER, PhD,[1,2] and JAN FRIDÉN, MD, PhD[3]

[1] Veterans Affairs Medical Center and University of California, Department of Orthopaedics, U.C. San Diego School of Medicine and V.A. Medical Center, 3350 La Jolla Village Drive, San Diego, California 92161, USA
[2] Veterans Affairs Medical Center and University of California, Department of Bioengineering, San Diego, California, USA
[3] Department of Hand Surgery, Göteborg University, Göteborg, Sweden

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Structure–function relationships in skeletal muscle have been described and examined for over a century. Classic studies have elucidated the microscopic and ultrastructural properties of skeletal muscle fibers, yielding great insights into their function. However, less attention has been paid to the studies of the macroscopic properties of skeletal muscle tissues dating back to the 1600s (see discussion and references in Kardel[27]). This macroscopic arrangement of muscle fibers is known as a muscle's architecture.[20] Because muscle architecture is a primary determinant of muscle function, understanding this structure–function relationship is of great practical importance. This understanding not only clarifies the physiological basis of force production and movement but also provides a scientific rationale for surgery that may involve tendon-transfer procedures, provides guidelines for electrode placement during electromyographic measures of muscle activity, explains the mechanical basis of muscle injury during normal movement, and aids in the interpretation of histological specimens obtained from muscle biopsies. The purpose of this review is to describe the theoretical significance of muscle architecture, to describe the basic architectural properties of human upper and lower extremity muscles, and to introduce advanced topics that represent current issues not yet resolved in the literature.

Abbreviations: CT, computed tomography; $dSL/d\omega$, sarcomere length change during joint rotation; ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; EMG, electromyography; L_f, muscle fiber length; L_m, muscle length; L_o, optimal length; MRI, magnetic resonance imaging; MTU, muscle tendon unit; PCSA, physiological cross-sectional area; P_o, maximum tetanic tension; ρ, muscle density; r, moment arm; ROM, range of motion; θ, muscle fiber pennation angle; V_{max}, maximum contraction velocity

Key words: architecture; muscle design; muscle fiber; skeletal muscle; tendon transfer surgery
Correspondence to: R.L. Lieber; e-mail: rlieber@ucsd.edu

BASIC ARCHITECTURAL DEFINITIONS

It is well-known that skeletal muscle is highly organized at the microscopic level, as witnessed by the incredible number and diversity of electron micrographs and schematics of muscle sarcomeres that have been published in review articles and textbooks. However, with few exceptions, the arrangement of muscle fibers within and between muscles has received much less attention. Muscle fibers are often depicted as projecting in bundles (fascicles) from an origin on a proximal tendon plate to an insertion more distally. This simply does not do justice to the wide array of muscle "designs" that are apparent throughout the animal kingdom. The ar-

chitecture of a given muscle is extremely consistent between individuals of the same species, giving rise to the concept that certain constraints that determine muscle architectural properties are present.[5,26,34,36,41,55,58,59,65] Although much attention has been paid to factors such as fiber-type distribution in determining muscle function, there is no question that function is also strongly determined by a muscle's architecture.[7]

Skeletal muscle architecture can be defined as "the arrangement of muscle fibers within a muscle relative to the axis of force generation."[31] Whereas muscle fibers have a relatively consistent fiber diameter between muscles of different sizes and fiber size is directly proportional to fiber force generation, architectural differences between muscles demonstrate much more variability and more strongly affect function. Although it might be proposed that different muscles produce different amounts of force based on differences in fiber size, fiber size between muscles actually varies little. Architectural differences between muscles are the best predictors of force generation. The various types of architectural arrangements are as numerous as the number of muscles themselves. For discussion purposes, however, we describe three general classes of muscle fiber architecture.

Muscles composed of fibers that extend parallel to the muscle's force-generating axis are described as having a parallel or longitudinal architecture (Fig. 1A). Although the fibers may project along a line parallel to the force-generating axis, experimental studies of mammalian muscle suggest that muscle fibers do not extend the entire muscle length. In fact, detailed studies of muscle fiber lengths demonstrate that muscle fibers may not even extend the entire length of a fascicle.[41,47] Muscles with fibers that are oriented at a single angle relative to the force-generating axis are described as having unipennate architecture (Fig. 1B). The angle between the fiber and the force-generating axis has been measured at resting length in mammalian muscles of very different designs and varies from about 0° to 30°. It becomes obvious when performing muscle dissections that most muscles fall into the third and most general category, multipennate muscles—muscles constructed of fibers that are oriented at several angles relative to the axis of force generation (Fig. 1C). Although these three designations are oversimplified, they provide a vocabulary with which to describe muscle designs. Because fiber orientation may have nothing to do with classic anatomical axes, determination of muscle architecture is impossible from a single biopsy or even a magnetic reso-

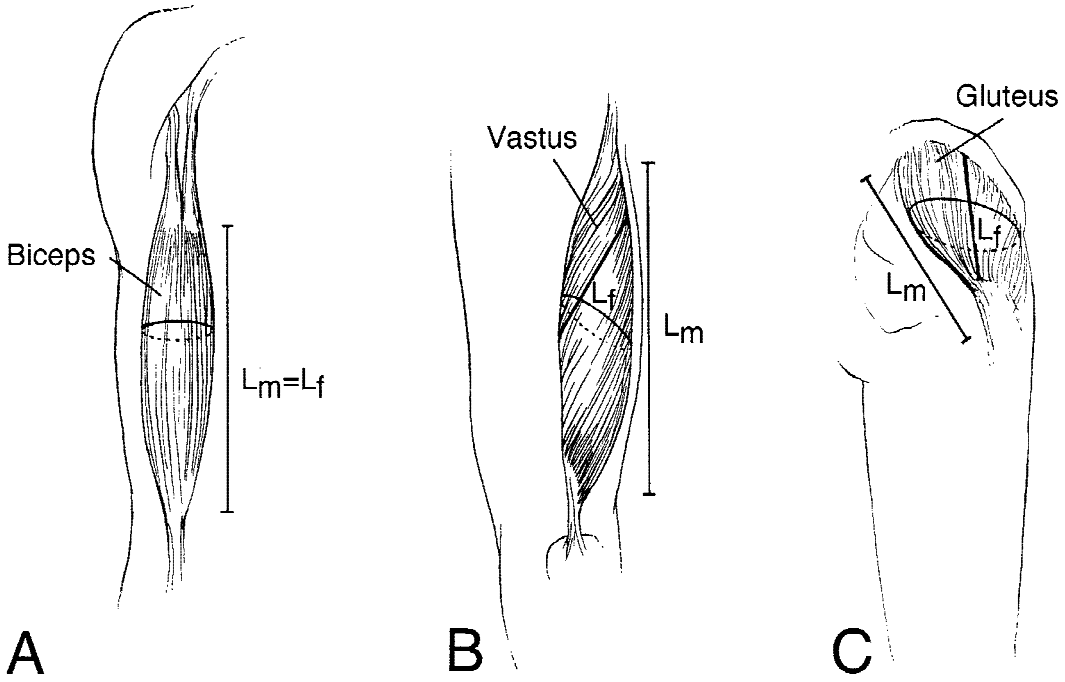

FIGURE 1. Artist's conception of three general types of skeletal muscle architecture. (A) Longitudinal architecture in which muscle fibers run parallel to the muscle's force-generating axis, as in the biceps brachii. (B) Unipennate architecture in which muscle fibers run at a fixed angle relative to the muscle's force-generating axis, as in the vastus lateralis muscle. (C) Multipennate architecture in which muscle fibers run at several angles relative to the muscle's force-generating axis (gluteus medius muscle). Lf, muscle fiber length; L_m, muscle length.

nance imaging (MRI), computerized tomography (CT), or ultrasound image, as these methods cannot account for variations in fiber length and orientation that occur along the muscle length. Thus, experimental methods have been developed to characterize the architectural properties of skeletal muscle.

EXPERIMENTAL DETERMINATION OF SKELETAL MUSCLE ARCHITECTURE

Quantitative studies of muscle architecture were pioneered by Gans and Bock[20] and Gans and De Vries,[21] who developed precise methodology for defining muscle architecture based on microdissection of whole muscles. The usual parameters included in an architectural analysis are muscle length (L_m), fiber length (L_f), pennation angle (i.e., the fiber angle relative to the force-generating axis, θ), and physiological cross-sectional area (PCSA). Typically, muscles are chemically fixed in 10% buffered formalin in order to maintain fiber integrity during dissection. The muscles should be chemically fixed while attached to the skeleton, to roughly preserve their physiological length, or physiological length in the skeleton should at least be noted. After fixation, muscles are dissected from the skeleton, their mass determined, and their pennation angle and muscle length are measured.

The majority of reported studies fix muscles by direct immersion into fixative, relying on diffusion of the fixative throughout the tissue thickness to facilitate subsequent dissection. However, for very thick muscles, the quality of the resulting fixation may not be adequate for architectural measurements to be made. Some investigators advocate allowing the muscle to go into rigor prior to fixation to preserve sarcomere length homogeneity. Recently, we implemented a high-pressure infusion method to chemically fix large human muscles of the shoulder and chest.[17] Muscles from fresh cadaveric specimens were fixed by high-pressure perfusion of 10% buffered formaldehyde via the carotid artery. To achieve adequate pressure, ~50 L containers filled with fixative were placed at a height of approximately 3 m above the cadaver. The infusion tubing had a diameter of approximately 5 mm. The carotid artery was cannulated and fixative introduced under pressure over a period of about 3 days. This method produced the best fixation of muscle tissue from cadaveric specimens that we have observed in approximately 10 years of this type of experimentation.

After fixation, θ is measured by determining the average angle of the fibers on the superficial muscle surface relative to the muscle's axis of force generation. Of course, this is only an estimate, because angles may vary from superficial to deep and even from proximal to distal (Fig. 1). Although more sophisticated methods could be developed for measurement of pennation angle, it is doubtful they would provide a great deal more insight into muscle function, because variations in pennation angle may not strongly affect function[20] (see below).

Muscle length L_m is defined as "the distance from the origin of the most proximal muscle fibers to the insertion of the most distal fibers."[31] This is not the same as L_f because of the variable degree of "stagger" seen in muscle fibers as they arise from and insert onto tendon plates. To date, muscle fiber length can be determined only by microdissection of individual fibers from fixed tissues or by laborious identification of fibers by glycogen depletion on serial sections along the length of the muscle.[47] Unless investigators are explicit, when they refer to muscle fiber length, they are probably referring to muscle fiber bundle length, because it is extremely difficult to isolate intact fibers, which run from origin to insertion, especially in mammalian tissue.[41,47] Thus, when microdissection is performed, bundles consisting of 5–50 fibers are typically used to estimate L_f. Because of the many different methods available for L_f determination, a distinction between "anatomical" fiber length and "functional" fiber length may be made. The anatomical fiber length determined by microdissection may not accurately represent the function of the fibers within the muscle, because fibers may be broken and their activation pattern unknown. In contrast, when determining L_f by glycogen depletion of single motor units,[47] it is clear that the entire fiber length is activated and can be identified histologically.

The final experimental step required to perform architectural analysis of a whole muscle is to measure sarcomere length within the isolated fibers. This is necessary to compensate for differences in muscle length that occur during fixation. In other words, to conclude that a muscle has "long fibers," one must ensure that it truly has long fibers and not that it was fixed in a highly stretched position corresponding to a long sarcomere length. Similarly, muscles with "short fibers" must be further investigated to ensure that they were not simply fixed at a short sarcomere length. In order to permit such conclusions, architectural measurements should be normalized to a constant sarcomere length, which eliminates fiber length variability due to variation in fixation length. This provides a reference value that can even be related back to the physiological length if the relationship between muscle length and joint

position is noted. Then, based on measured architectural parameters and joint properties, the relationship between sarcomere length and joint angle can be calculated. Because sarcomere length also strongly influences muscle force generation, an understanding of the relationship between sarcomere length change and movement has been used in many studies to provide added understanding of muscle design.[8,37,38,52,54]

After measurement of the architectural parameters described above, the PCSA is calculated. The PCSA of a muscle is the only architectural parameter that is directly proportional to the maximum tetanic tension generated by the muscle. This value is almost never the actual cross-sectional area of the muscle as measured in any of the traditional anatomical planes, as would be obtained, for example, using a noninvasive imaging method such as MRI, CT, or ultrasound. Theoretically, PCSA represents the sum of the cross-sectional areas of all the muscle fibers within the muscle. It is calculated using eq. 1, which was verified experimentally by Powell et al.,[50]

$$\text{PCSA } (\text{mm}^2) = \frac{\text{muscle mass (g)} \cdot \cos\theta}{\rho\ (\text{g/mm}^3) \cdot \text{fiber length (mm)}} \quad (1)$$

where ρ represents muscle density (1.056 g/cm^3 for mammalian muscle) and θ represents surface pennation angle.

Equation 1 represents muscle volume (mass/density) divided by fiber length and has units of area (in this case, cm^2). Because fibers may be oriented at an angle relative to the axis of force generation, the cosine term is often included, considering that not all the fiber tensile force is transmitted to the tendons. This idea can be envisioned using a model of a muscle fiber pulling with x units of force at an angle θ relative to the muscle axis of force generation. In this configuration, only a component of muscle fiber force will actually be transmitted along the muscle axis, which will be $x \cdot \cos\theta$. In other words, in this scenario, pennation is seen to result in a loss of muscle force relative to a muscle with the same mass and fiber length but with zero pennation angle.

EXPERIMENTAL VERIFICATION OF MUSCLE ARCHITECTURE EQUATIONS

Cross-Sectional Area. The accuracy of Eq. 1 was highlighted by experimental comparison between the calculated maximum muscle tetanic tension (based on PCSA) and measured maximum tetanic tension (using traditional physiological testing techniques) of guinea pig skeletal muscle.[50] It was found that the estimations and predictions agreed within experimental error and that the only exception was the soleus muscle (Fig. 2). For all "fast" muscles, force per unit cross-sectional area was ~22.5N/cm^2, which serves as a nominal value of specific tension for mammalian muscle.

A potential problem with eq. 1 is that it assumes muscle fiber pennation angle is constant during muscle contraction. Experimental measurement of muscle fiber pennation angle in the unipennate rat gastrocnemius muscle have revealed that this is not the case.[70] Zuurbier and Huijing[69] placed small wire markers across the gastrocnemius surface and filmed marker movement during muscle contraction. Using these data, the authors measured fiber pennation angle and showed that muscle fiber angle varied considerably as muscle length was altered throughout the normal physiological range of motion (Fig. 3A). While resting fiber θ was approximately 30°, during isotonic contraction, θ increased to almost 60° and the angle between the muscle aponeurosis and the muscle axis rotated from about 10° to 15°. The fact that muscle fibers appear to be free to rotate during contraction has a number of implications. First, PCSA as calculated from eq. 1 may be somewhat arbitrary in its ability to predict force. Second, fiber

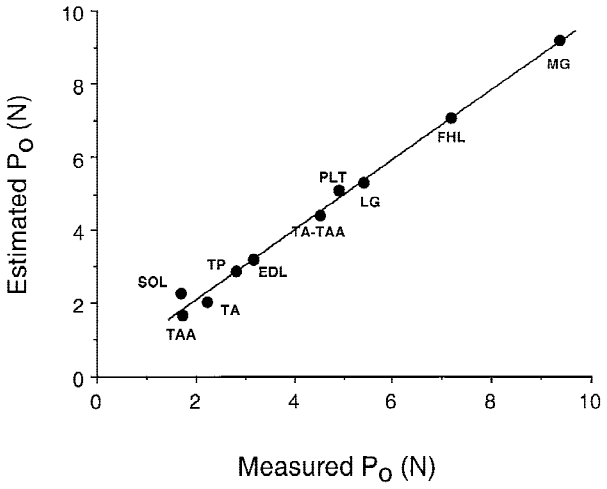

FIGURE 2. Comparison between measured P_o and estimated P_o, based on measurement of PCSA in guinea pig muscles. Note that PCSA and P_o are proportional for all muscles except the soleus, the one muscle with a preponderance of slow muscle fibers. (Abbreviations: EDL, extensor digitorum longus; FHL, flexor hallucis longus; LG, lateral gastrocnemius; MG, medial gastrocnemius; PLT, plantaris; SOL, soleus; TA, tibial anterior; TA–TAA, combined tibialis anterior accessorius and tibialis anterior; TAA, tibialis anterior accessorius; TP, tibialis posterior.) Note the close linear correlation between PCSA and measured P_o. The slope of the line 22.5N/cm^2 represents a reasonable value for muscle-specific tension. (Data replotted from Powell et al.[50])

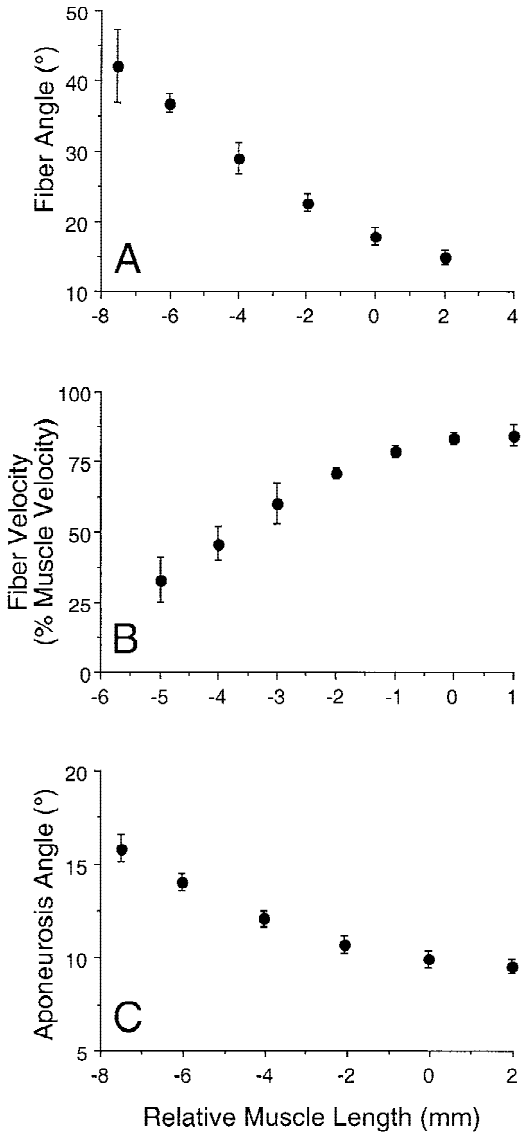

FIGURE 3. Changes in muscle structural properties as a function of muscle length in rat medial gastrocnemius muscle. (A) Fiber pennation angle. This angle increases as the muscle shortens. (B) Fiber velocity expressed relative to muscle velocity during isotonic shortening. Notice that at shorter lengths, fiber velocity is a decreasing percentage of muscle velocity, due to fiber rotation. (C) Aponeurosis angle. (Data replotted from Zuurbier and Huijing.[70])

rotation has a significant impact on the estimation of muscle fiber contraction velocity based on the measurement of whole muscle velocity. During shortening of the rat gastrocnemius, if fiber contraction velocity is calculated holding pennation angle constant, it will simply scale with whole muscle velocity. However, if fiber rotation is included, fiber velocity is actually much lower than whole muscle velocity, which, functionally, results in prediction of greater force generation by the muscle fibers (Fig. 3B). Thus, fiber rotation during muscle shortening provides a degree of freedom that allows muscle fibers to maintain a higher level of force generation than when fibers are constrained to maintain a constant pennation angle.

Does significant fiber rotation occur in human muscles? An affirmative answer to this question was provided in a most dramatic example by Fukunaga and colleagues[19] and Kawakami and colleagues,[28] who measured fascicle plane orientation during voluntary contraction of the human quadriceps, dorsiflexor, and plantarflexor muscles. Their measurements revealed that during low-level voluntary contraction, vastus lateralis pennation angle increased from 14° with the knee extended to 21° with the knee flexed, and, during this voluntary contraction, fascicle length decreased from 126 mm to 67 mm. Similarly, the medial gastrocnemius increased from about 20° to 45°, with a similar significant degree of shortening (Fig. 4). These data provide a real-time picture indicating that muscle fiber shortening and rotation are simultaneous and normal events that occur during muscle contraction. Fiber rotation during muscle contraction permits tensile force transmission to occur even when muscle fibers are oriented at an angle relative to the muscle's force-generating axis. The fact that pennation angles are small at muscle resting lengths (0–30°) probably accounts for the agreement between experiment and theory observed by Powell et al.[50] Thus, in predicting maximum muscle force–producing capacity, correction for fiber angulation may not be necessary.

Muscle Fiber Length. Even though it is often stated that muscle fiber length is proportional to fiber excursion (or velocity), there has not been a comprehensive study in mammalian muscle, analogous to the study described above for PCSA that confirms this relationship quantitatively. However, there is a good deal of experimental evidence available in the literature to suggest that this relationship is valid. First, in mechanical studies of isolated frog single muscle fibers, in which fiber length, and thus the number of sarcomeres in series, is easily measured, maximum contraction velocity is directly proportional to fiber length, as is the width of the isometric length–tension relationship.[15,67] This is the reason that muscle contraction velocities are often normalized and expressed in "fiber length/s" or "sarcomere lengths/s." Second, in a mechanical and anatomical study of the cat semitendinosus muscle, which represents a unique model in that it is composed of distinct proximal and distal heads separated by a tendinous inscription and which has distinct innerva-

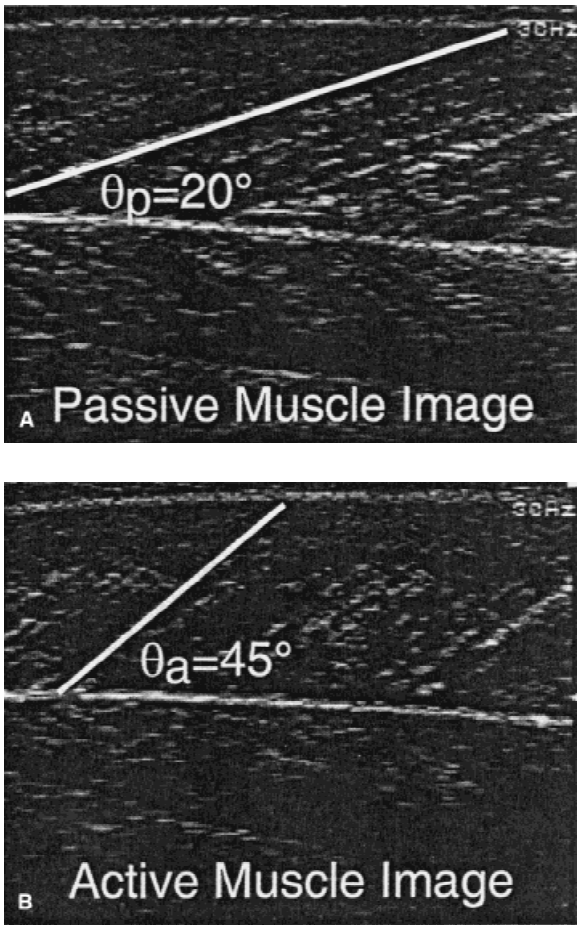

FIGURE 4. Ultrasound image of human medial gastrocnemius measured in the sagittal plane. (A) Muscle image at rest demonstrating passive fiber angle (θ_p) of 20° relative to the aponeurosis. (B) Muscle image with the muscle activated demonstrating active fiber angle (θ_a) of 45° relative to the aponeurosis. (Ultrasound images courtesy of T. Fukanaga and Y. Kawakami.)

tion to each head, the maximum contraction velocity of the two heads stimulated simultaneously was the same as the sum of the maximum contraction velocity of the two heads stimulated individually.[2]

The relationship between fiber length and whole muscle properties is also complicated by the observation that some very long feline muscles are composed of relatively short muscle fibers arranged in series.[41] The cat sartorius, tenuissimus, and semitendinosus muscles were microdissected and shown to be composed of relatively short fibers, 2–3 cm in length, that were arranged in series. By staining these specimens with acetylcholinesterase, the authors also showed that the endplates occurred in about the same serial arrangement. These data were combined with electromyographic data that demonstrated that the "in series" fibers were innervated by branches of axons also arranged in series, so that all serial fibers would be expected to be activated simultaneously. This led to the suggestion that short fibers arranged in series are simultaneously activated to act effectively like long muscle fibers. The reason this was viewed as a good design is that it is known that electrical conduction along muscle fibers is relatively slow (2–10 m/s).[13] Because of the long length of these cat muscles (10–15 cm), it was conceived that by the time the action potential reached the end of the fiber, the central region might already be relaxing. Mechanically, this is clearly unfavorable. This is not to say that longer fibers do not exist within cat muscles. In a histological study of the cat tibialis anterior, Ounjian et al.[47] traced single muscle fibers within single motor units along the muscle belly cross-section and clearly demonstrated that the fibers did not run the entire muscle length nor even the entire fascicle length (Fig. 5). Fibers within motor units ranged from ~8 mm to ~50 mm. Furthermore, the authors elucidated the general pattern that slow (S) units tended to have more uniform fiber lengths compared with fast (F) units. Fiber ends within the F units also tapered to a greater extent compared with S units. Additionally, some units actually began and ended within a fascicle, whereas others extended all or part of the length of a fascicle (Fig. 5). These data, taken in concert with the motor unit stiffness data of Petit et al.[48] and Petit et al.,[49] imply that the higher stiffness of the S compared with F units could result from the relatively short muscle fibers within those units and could represent a beneficial design for postural control. Because the S units are implicated in posture and also have a relatively high stiffness, small positional perturbations would be effectively damped by the intrinsic properties of the fibers within those units. It would also be predicted that slow units would have a narrower length–tension relationship compared with fast units as a direct reflection of differences in muscle fiber length, but this suggestion has only received weak experimental support.[25]

To summarize the results of muscle fiber length determinations, most studies report fiber bundle lengths rather than fiber lengths. These may be actual anatomical lengths based on microdissection or a functional length based on a physiological phenomenon such as glycogen depletion of single fibers. However, because fibers may terminate end to end, functionally, a fascicle may perform like a single muscle fiber of equivalent total length. Fibers may terminate within the muscle belly in the complex extracellular matrix composed of endomysial connective tissue.[62] These connective tissues merge into a final "external tendon" where force can be applied

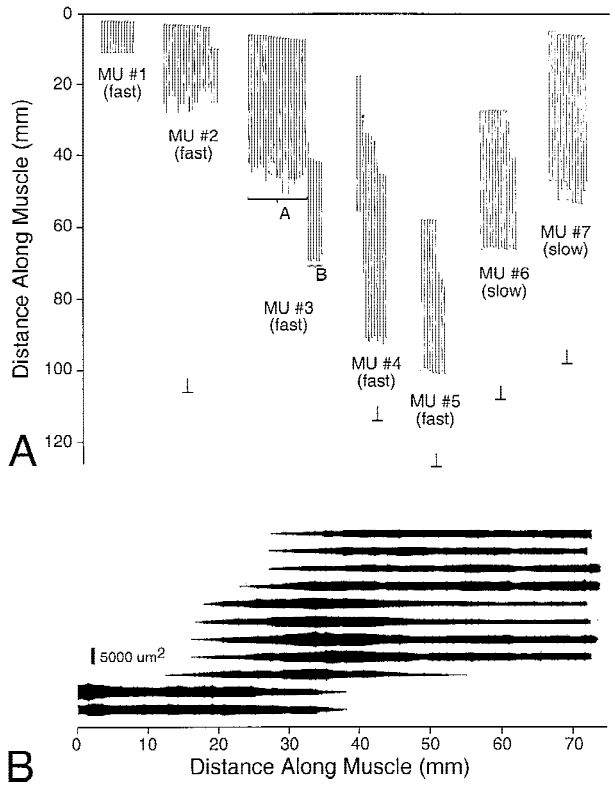

FIGURE 5. (A) Muscle fiber length and position determined by glycogen depletion within seven different single motor units of the cat tibialis anterior muscle. Motor units 1–5 were physiologically typed as fast, and motor units 6 and 7 were physiologically typed as slow. The most proximal end of the muscle is defined as 0 mm, whereas the most distal portion of the muscle is denoted by the ⊥ symbol. Note that some muscle fibers actually begin and end within the muscle fascicle itself. (B) Cross-sectional area of fibers within motor unit 4 shown in A. Note that, typically, fibers taper in ending within the fascicle. (Figure redrawn from Ounjian et al.[47])

to bone. It would be important to demonstrate quantitatively the relationship between fiber or fascicle length and measured maximum contraction velocity of muscle (V_{max}).

ARCHITECTURE OF HUMAN SKELETAL MUSCLES

Several architectural investigations have been performed in human upper and lower limb muscles.[5,18,34,36,65] Reported pennation angles normally range from 0° to 30°, and the ratio of muscle fiber length to muscle length ranges from ~0.2 to ~0.6. In other words, even the most "longitudinally oriented" muscles have fiber bundles that extend only about 60% of the muscle length.

Muscles of the Human Lower Limb. The two most important muscle architectural parameters are muscle PCSA (proportional to maximum muscle force) and muscle fiber length (proportional to maximum muscle excursion). The relationship between these two parameters for human muscles is shown in Figure 6. Although each muscle is unique in terms of its architecture, taken as functional groups (e.g., hamstrings, quadriceps, dorsiflexors, plantarflexors), a number of generalizations can be made regarding lower extremity muscles. Quadriceps muscles are characterized by their relatively high pennation angles, large PCSAs, and short fibers. In terms of design, these muscles appear suited for the generation of large forces. The hamstrings, on the other hand, by virtue of their relatively long fibers and intermediate PCSAs, appear to be designed for large excursions. Specifically, note that the sartorius, semitendinosus, and gracilis muscles have extremely long fiber lengths and low PCSAs, which permit large excursions at low forces (Fig. 6). Specialization of functional groups also appears to be true of the plantarflexors and dorsiflexors. A very general conclusion might be that the antigravity extensors are designed more toward force production, and the flexors are designed more for long excursions. The most extreme example of such a design is the soleus muscle, with its high PCSA and short fiber length, suitable for generating high force with small excursion. This would suit it well for a postural stabilization role.

The relatively high pennation angles of the quadriceps also have implications for muscle biopsy analysis and electromyogram (EMG) measurement. Because the fibers are relatively short, a biopsy obtained along the length of the muscle may not be representative of all the fibers along the muscle, which are staggered to a high degree. Experimental quantification of repeated biopsies from four rhesus monkeys was studied in three different locations along the length of the soleus, medial gastrocnemius, and tibialis anterior muscles.[56] The authors demonstrated a much greater fiber-type percentage variability between animal subjects than between biopsies within a subject. The authors also demonstrated that, in spite of sampling different fibers along muscles with very different architectures (L_f/L_m ratios ranging from 0.23 to 0.35), it was possible to obtain representative percentages within these different muscle regions. Fiber-type variability was 5–10% within a muscle but as much as 30% between the same muscles of different animal subjects.

With regard to EMG measurements, a similar sampling problem may arise as a result of muscle fiber stagger. Electrodes placed in one region of the muscle may not provide an electrical signal that is

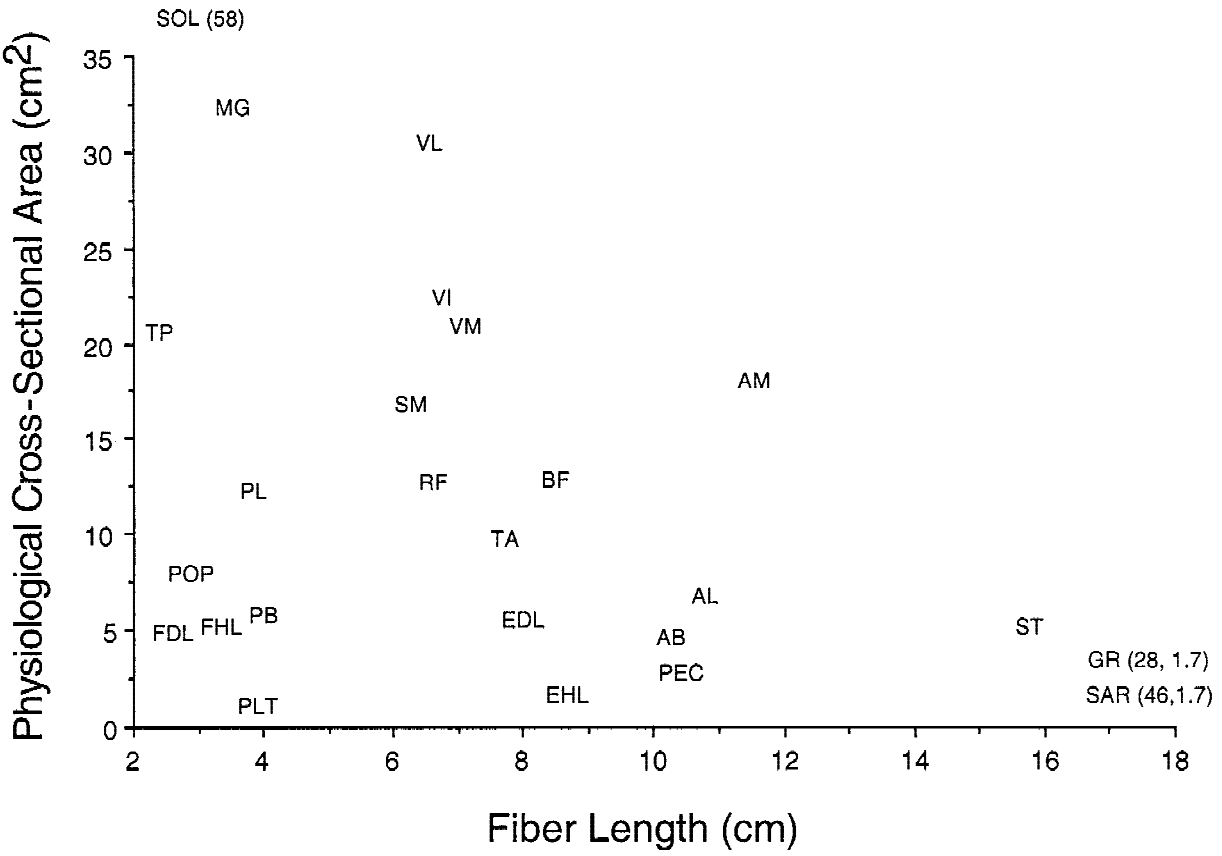

FIGURE 6. Scatter graph of the fiber length and cross-sectional areas of muscles in the human lower limb. Fiber length is proportional to muscle excursion, and cross-sectional area is proportional to maximum muscle force. Thus, this graph can be used to compare the relative forces and excursions of muscles within the lower limb. (Abbreviations: AB, adductor brevis; AL, adductor longus; AM, adductor magnus; BF_l, biceps femoris, long head; BF_s, biceps femoris, short head; EDL, extensor digitorum longus; EHL, extensor hallucis longus; FDL, flexor digitorum longus; FHL, flexor hallucis longus; GR, gracilis; LG, lateral gastrocnemius; MG, medial gastrocnemius; PB, peroneus brevis; PEC, pectineus; PL, peroneus longus; PLT, plantaris; POP, popliteus; RF, rectus femoris; SAR, sartorius; SM, semimembranosus; SOL, soleus; ST, semitendinosus; TA, tibialis anterior; TP, tibialis posterior; VI, vastus intermedius; VL, vastus lateralis; VM, vastus medialis.) (Data from Wickiewicz et al.[65])

representative of motor units from different regions of the same muscle.[40] This is due to two factors. First and most obvious, muscle fibers do not extend the length of the muscle, and second, a natural gradation exists in fiber-type percentage and thus motor unit types from superficial to deep within a muscle.[6] Because motor units are activated in a stereotypical fashion from slow to fast,[23] this may affect duration and amplitude of EMG signals measured at different depths. It should be noted, however, that the extent to which this inability to sample uniformly affects either clinical judgment or the understanding of muscle activation has not been clearly determined. A second level of complexity that may affect the extent to which an EMG signal is representative of muscle function arises from the fact that some muscles, such as the cat lateral gastrocnemius, demonstrate compartmentalization.[16,64] Under these conditions, separate portions of muscles with unique fiber-type distributions are innervated by distinctly different motor nerves. As a result, their activation pattern and general level of use can differ, in spite of the fact that they are in the same muscle.

Muscles of the Human Upper Limb. In light of the specialization observed in the lower limb, it is probably not surprising to note that there is also a high degree of specialization "built into" upper extremity muscles, by virtue of their architecture. Such specialization makes sense in light of the fact that a great deal more functional diversity is seen in upper extremity compared with lower extremity movement. From an architectural point of view, the superficial and deep digital flexors are similar to each other but are different from the digital extensors (Fig. 7). The flexors would be predicted to generate almost twice the force as the extensors and with a slightly greater active range. As another example, based on its very

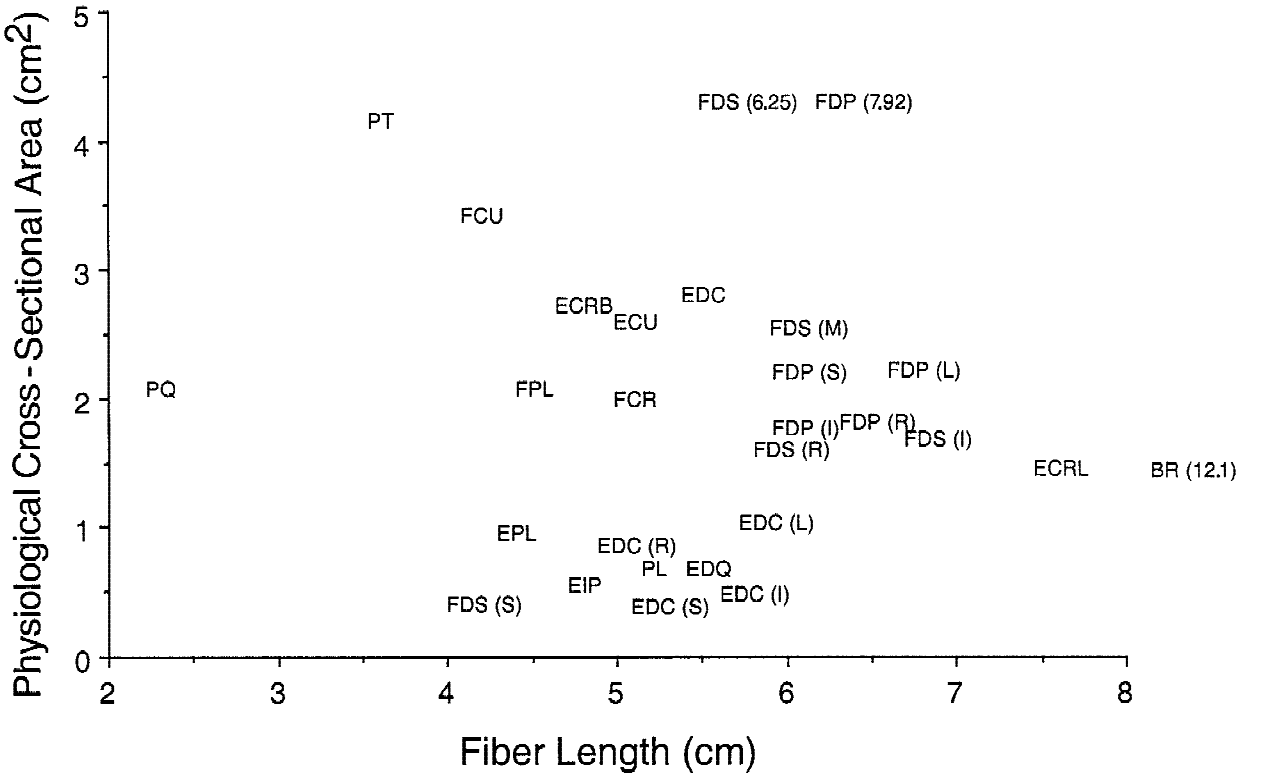

FIGURE 7. Scatter graph of the fiber length and cross-sectional areas of muscles in the human forearm. Fiber length is proportional to muscle excursion, and cross-sectional area is proportional to maximum muscle force. Thus, this graph can be used to compare the relative forces and excursions of arm and forearm muscles. (Abbreviations: BR, brachioradialis; ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; ECU, extensor carpi ulnaris; EDC I, EDC M, EDC R, and EDC S, extensor digitorum communis to the index, middle, ring, and small fingers, respectively; EDQ, extensor digiti quinti; EIP, extensor indicis proprius; EPL, extensor pollicis longus; FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; FDP I, FDP M, FDP R, and FDP S, flexor digitorum profundus muscles; FDS I, FDS M, FDS R, and FDS S, flexor digitorum superficialis muscles; FDS I (C), the combined properties of the two bellies as if they were a single muscle; FDS I (P) and FDS I (D), proximal and distal bellies of the FDS I; FPL, flexor pollicis longus; PQ, pronator quadratus; PS, palmaris longus; PT, pronator teres.) (Data from Lieber et al.[34,36])

high PCSA, the flexor carpi ulnaris is expected to generate very high forces. Examination of this type of information can be used to compare functional properties between muscles that might be surgically transferred to restore lost function (see below). Intuitively, it might be considered important to match the transferred muscle's architectural properties to the architectural properties of the muscle whose function was lost.

SIGNIFICANCE OF MUSCLE ARCHITECTURE IN SURGICAL TENDON TRANSFER

In addition to improving our understanding of muscle anatomy and function, elucidation of muscle architecture may ultimately provide information useful for selection of muscles used in tendon transfers. To substitute lost muscle function, the distal tendons of muscles are often transferred from one position to another,[3,4,22,51] a procedure known as a "tendon transfer." It would seem reasonable to select a donor muscle with architectural properties that are similar to the original muscle in order to perform the original muscle's function. (Numerous other factors also influence donor selection, including donor muscle availability, donor muscle morbidity, preoperative strength, integrity, expendability, synergism, transfer route and direction, and surgical experience and preference.)

Surgical Restoration of Digital Extension. We envision that architectural differences might be useful in tendon transfer when making a choice involving multiple donors or when a combination of transfers is available for selection. For example, in the surgical restoration of digital extension following high radial nerve palsy, described and accepted potential donor muscles (which are transferred to the extensor digitorum longus) include the flexor carpi radialis, the flexor carpi ulnaris, the flexor digitorum superficialis to the middle finger, and the flexor digitorum superficialis to the ring finger. From the standpoint

of architecture alone, the flexor digitorum superficialis to the middle finger most closely resembles the extensor digitorum communis in terms of force generation (i.e., cross-sectional area) and excursion (i.e., fiber length). This is emphasized by the relatively close position in "architectural space" of the flexor digitorum superficialis to the middle finger to the extensor digitorum communis (Fig. 7). If individual architectural properties are compared, it is clear that the flexor digitorum superficialis to the middle finger has more than enough excursion compared with the extensor digitorum communis, whereas the flexor carpi ulnaris has excessive force-generating potential but may lack sufficient excursion. Thus, if the concern were sufficient force, the flexor carpi ulnaris might be chosen, whereas if the concern were excursion, the flexor digitorum superficialis to the middle finger might be chosen. Either way, a knowledge of muscle architecture permits an informed decision to be made. It should be noted that architectural mismatch between the flexor carpi ulnaris and extensor digitorum communis has been blamed for the poor clinical result of this transfer.[39]

Surgical Restoration of Thumb Extension. To restore thumb extensor function in high radial nerve palsy, potential donors include the flexor digitorum superficialis to the middle finger, flexor digitorum superficialis to the small finger, and palmaris longus. Again, in terms of architecture, the flexor digitorum superficialis to the small finger and the palmaris longus are similar to the extensor pollicis longus and therefore should provide the force generation and excursion required to restore lost function (Fig. 7).

Surgical Restoration of Thumb Flexion. As a final example, following high median nerve palsy, anterior interosseous nerve injury, or isolated, irreparable flexor pollicis longus muscle injury, multiple potential donors for transfer to restore thumb flexion are available. These donors include the brachioradialis, extensor carpi radialis brevis, extensor carpi radialis longus, extensor carpi ulnaris, extensor digiti quinti, and flexor digitorum superficialis to the ring finger. From an architectural standpoint, the extensor carpi radialis brevis, flexor digitorum superficialis to the ring finger, and extensor carpi ulnaris are most similar to the flexor pollicis longus (Fig. 7).

Quantification of Architectural Differences between Pairs of Muscles. To perform a quantitative comparison between muscles in terms of their architectural properties, a method was developed to determine which architectural properties best discriminated between functional groups and then a scaled calculation was made comparing pairs of muscles using these parameters.[33] The multivariate method of the discriminant analysis first determined architectural properties which uniquely characterized muscle groups in either the rabbit hindlimb[32] or the human wrist and hand musculature.[34,36] For both species, it was possible to identify five specific discriminating parameters that successfully characterized the appropriate functional groups. The best discriminators were (in order): L_f, PCSA, L_m, L_f/L_m ratio, and muscle mass. These five parameters were then used to calculate an "architectural difference index" for pairs of muscles, according to a modification of the distance formula for two points in Cartesian coordinates. The difference is calculated from linear combinations of discriminating parameters in order to obtain a single number, the magnitude of which describes the architectural difference between two muscles (high values implying architectural differences).

SIGNIFICANCE OF MUSCLE ARCHITECTURE IN MOTOR CONTROL

One consequence of the architectural specialization noted throughout the body is that the neuromuscular system is not the only means available to modify muscular force and excursion. Although neural input to muscles can change muscle force, the effectiveness of neural input is increased, because individual muscles are intrinsically designed for a specific function, large excursion, for example. The nervous system provides the input signal to modulate the timing and intensity of the activation signal to the muscle. It is as if the nervous system acts as the central control and the muscle interprets the control signal into the actual external action by virtue of its intrinsic design.

MECHANICAL PROPERTIES OF MUSCLES WITH DIFFERENT ARCHITECTURES

As stated above, muscle force is proportional to PCSA and muscle velocity is proportional to fiber length. By stating that velocity is proportional to fiber length, it is implicit that the total excursion (active range) of a muscle is also proportional to fiber length.

Comparison of Two Muscles with Different PCSA. Suppose that two muscles had identical fiber lengths and pennation angles but one muscle had twice the mass (equivalent to saying that one muscle had twice the number of fibers and thus twice the PCSA). Also

suppose that their fiber-type distributions are identical and that they generate the same force per unit area (specific tension). The functional difference between these two muscles is shown in Fig. 8. The muscle with twice the PCSA has a length–tension curve with the same shape but is amplified upward by a factor of two. Thus, the maximum tetanic tension (P_o) of the larger muscle will be twice that of the smaller muscle. Similarly, comparison of schematic force–velocity curves indicates that the differences between muscles are simply an upward shift in P_o for the larger muscle but the curve retains the same shape. If both curves were plotted on relative scales (i.e., percent maximum tension instead of absolute tension), the two muscles would appear to have identical properties. This demonstrates that although architectural properties profoundly affect the extrinsic muscle properties (i.e., the properties that vary with absolute muscle size, such as P_o, PCSA, or mass), they have no effect on its intrinsic properties (i.e., the properties that are independent of absolute muscle size, such as the shape of the length–tension or force–velocity curves or fiber length/muscle length ratio).

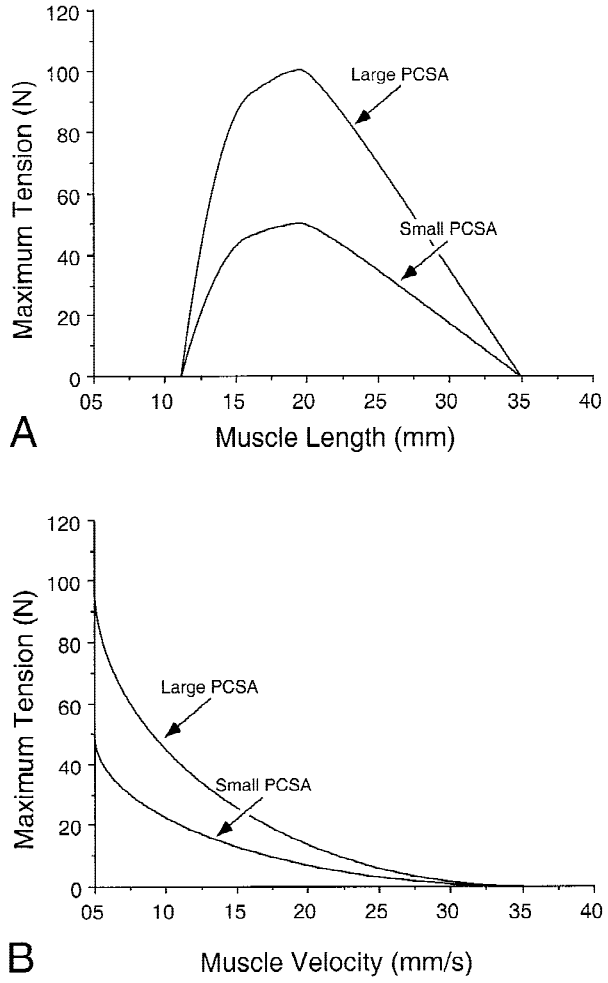

FIGURE 8. Schematic drawing of two muscles with different PCSAs but identical mass. (A) Comparison of isometric length–tension properties. (B) Comparison of isotonic force–velocity properties. The effect of increased PCSA with identical fiber length is to shift the absolute length–tension and force–velocity curves to higher values but with retention of the same range and intrinsic shape.

Comparison of Two Muscles with Different Fiber Lengths. If two muscles have identical PCSAs and pennation angles but different fiber lengths, then the effect of increased fiber length is to increase muscle velocity (or muscle excursion), as demonstrated in the schematic in Figure 9. Peak absolute force of the length–tension curves is identical between muscles, but the absolute muscle active range is different. For the same reason that an increased fiber length increases active muscle range of the length–tension relationship, it results in an increase in the muscle's V_{max}. As in the example with altered PCSA, an increase in fiber length increases absolute properties but has no effect on the intrinsic properties of the muscle. Experimental support for this concept was obtained indirectly in the study mentioned above on the cat semitendinosus muscle.[2] When only the proximal semitendinosus head was activated, its V_{max} was 224 mm/s, whereas when only the distal semitendinosus head was activated, its V_{max} was 424 mm/s. Then, when both heads were activated simultaneously, the whole muscle V_{max} was 624 mm/s, or the sum of the two velocities. The values for V_{max} were proportional to the different lengths of the proximal and distal heads. These data indicate that the longer are the fibers in series (equivalent of saying the greater number of sarcomeres in series), the greater is the muscle contraction velocity. As expected, maximum isometric tension was essentially the same regardless of which activation pattern was used.

RANGE OF MOTION (ROM) AS A FUNCTION OF ARCHITECTURE

Muscles with longer fibers have longer functional ranges than do muscles with shorter fibers. This does not necessarily indicate that muscles with longer fibers are associated with joints that have larger ranges of motion. It is true that a muscle with longer fibers does have a longer working range, but the amount of change in muscle fiber length that occurs as a joint rotates depends on the muscle moment arm, the "mechanical advantage" that a muscle has at a particular joint. This idea can be explained by compar-

ing the situation in which a simulated "muscle" is attached to a joint using two different moment arms but muscles with identical fiber lengths. In the case where the moment arm is greater, the muscle fibers will change length much more for a given change in joint angle compared with the same change in joint angle in the system with shorter fibers. As a result, the active ROM for the muscle-joint system with the larger moment arm will be smaller compared with the system with the larger moment arm in spite of the fact that their muscular properties are identical. Therefore, the design of a muscle when considered in isolation may or may not be directly related to the actual function of a muscle once placed in the skeletal system.

It is now important to qualify the previous statement about muscle design and architecture. Muscles that are designed for speed based on their long fibers may not actually produce large velocities if they are placed in the skeleton with a very large moment arm. The increased moment arm results in a very large joint moment, so that the muscle would be highly suited for torque production but at low angular velocities. Similarly, a muscle that appears to be designed for force production due to the large PCSA, if placed in position with a very small moment arm, may actually produce high joint excursions or angular velocities. Differences between muscle-joint systems thus require complete analysis of both joint and muscular properties (see below).

ISOKINETIC DYNAMOMETERS USED IN PHYSICAL ASSESSMENT

It is necessary to characterize human performance objectively, not only to evaluate patient progress but also to ascertain the efficacy of clinical treatment. Using objective criteria such as maximum joint moment, shape of the moment–angle relationship, and ROM, it is possible to evaluate the efficacy of many surgical and rehabilitative procedures or the progression of neuromuscular diseases. One of the most commonly used tools for musculoskeletal assessment is the isokinetic dynamometer. If joint velocity is set to zero, an isometric joint moment versus joint–angle relationship is generated which, as described above, represents the interaction between muscle and joint properties. It is clearly not appropriate to ascribe any portion of a moment versus joint angle curve to either muscle or joint properties alone. Such moment–joint angle curves are also often generated at constant angular velocities, hence the term, "isokinetic." It is assumed that constant angular velocity corresponds to constant muscle velocity. However, this is a poor assumption for a number of reasons: (1) joint moment arm is not always constant, and therefore linear changes in joint angle do not correspond to linear changes in either muscle or muscle fiber length; (2) muscle activation is neither instantaneous, constant, nor maximal, all of which are required to generate the classic force–velocity relationship; and (3) the compliance of the tendon permits muscle length changes that are not proportional to joint angle changes. Although these points have been known for some time, nonconstant muscle velocity was directly demonstrated experimentally by Fukunaga and colleagues,[19] who used ultrasonography to measure fascicle length changes during isokinetic tasks. The authors found that, for the reasons stated above, the only parameter constant about isokinetic motion was joint angular velocity. Muscle fiber velocity, tendon velocity, and muscle velocity all changed in a highly nonsteady state manner.

Influence of PCSA and Fiber Length on Isokinetic Torque. It is reasonable to assume that joint kinematics are velocity independent and, thus, that variations in the moment–angle curves as a function of velocity represent variations in muscle force. The moment achieved during isokinetic contraction (concentric muscle contraction) must necessarily be less than that achieved during isometric contraction, due to the muscle's force–velocity relationship. Also, muscle force generated during an isokinetic contraction is a function of the muscle's PCSA and fiber length. The reason that isokinetic muscle force varies with PCSA is obvious because force is directly proportional to PCSA. The reason that muscle force varies with fiber length is less obvious, although straightforward. The isokinetic moment measurements are obtained while the joint is limited to a specific isokinetic movement. This means that the muscle is also forced to maintain a certain (but not necessarily constant) shortening velocity. Because shortening velocity is fixed (because excursion is fixed), the longer are the muscle fibers, the higher is the muscle force that can be sustained during shortening. This is because longer muscle fibers have more sarcomeres in series and, with longer fibers, each sarcomere has a slower absolute contraction velocity, allowing it to stay higher on its force–velocity curve (i.e., closer to P_o). This idea is illustrated in the force–velocity curve of Figure 9, where force is measured from two hypothetical muscles that are shortening at the same velocity. Note that, for a given shortening velocity (vertical dotted line), the muscle with longer fibers maintains a higher force compared with the muscle with shorter fibers.

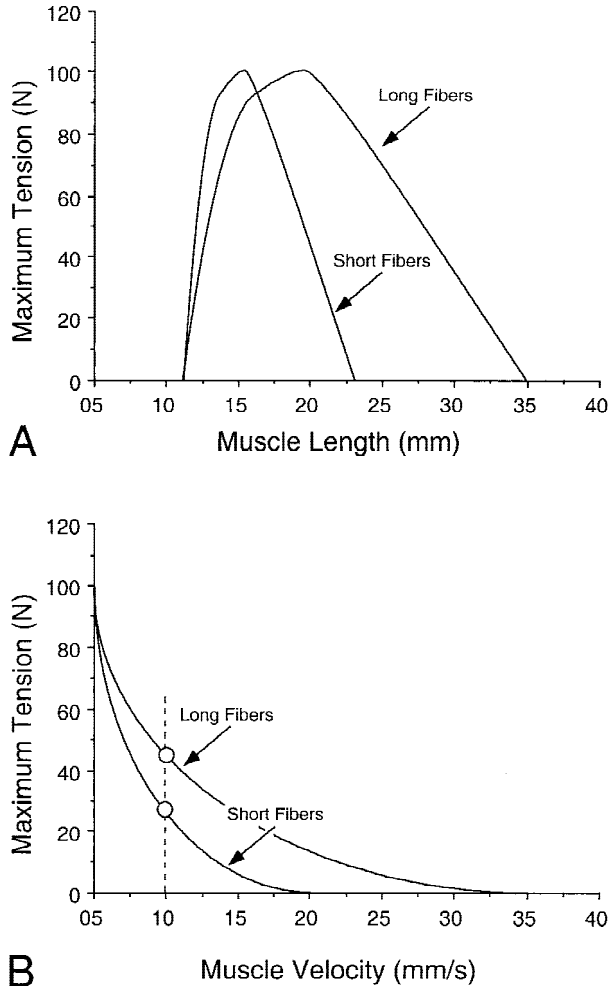

FIGURE 9. Schematic drawing of two muscles with different fiber lengths but identical PCSAs. (A) Comparison of isometric length–tension properties. (B) Comparison of isotonic force–velocity properties. The effect of increased fiber length is to increase the absolute range of the length–tension curve and absolute velocity of the force–velocity curve but with retention of the same peak force and intrinsic shape. Dotted vertical line demonstrates that, for an equivalent absolute velocity, the muscle with longer fibers generates a greater force.

This means that the muscle with longer fibers generates the greater force at all velocities.

Earlier, it was stated that a muscle with longer fibers was designed for speed or excursion, whereas a muscle with a high PCSA was designed for force production. It is now apparent that in the intact musculoskeletal system, this is not necessarily the case.

Theoretical Calculation of Optimal Architecture. The previous discussion highlights the general principle that force production in muscle is highly dependent on architecture, although not necessarily in ways that are intuitively obvious. This is because muscle velocity achieved during motion has a very large effect on force production. It is not effective to "design" a high-force muscle by giving it a huge PCSA and short L_f, if it is placed in a situation where shortening velocity is sizable. This is because the short fibers have a high sarcomere shortening velocity and thus force depression. We investigated the trade-off between PCSA and L_f in producing muscle force for the mouse tibialis anterior muscle, whose architectural[7] and kinematic properties were known.[10] We considered the various ways in which this muscle could be "designed," given the constraint of constant volume (or mass) within the shank. The main result was that an optimal fiber length can be shown to be long enough to permit low sarcomere velocity but short enough to permit high PCSA. The logic is as follows: If muscle fiber length of the tibialis anterior is increased while maintaining muscle mass, PCSA (and thus isometric muscle force) necessarily decreases (Fig. 10A). This is a detriment to force production. In contrast, as fiber length increases at constant muscle mass, sarcomere velocity decreases, resulting in increased muscle force at this velocity (Fig. 10B). These two effects oppose one another, resulting in a theoretically "optimal" fiber length, representing the simultaneous satisfaction of both criteria. By calculating the tension expected during muscle shortening as a function of calculated fiber length variation, the interaction between isometric force production (resulting from PCSA) and dynamic force production (sarcomere number and velocity) can be seen (Fig. 10C). The two opposing effects in Figures 10A and 10B result in the existence of an "optimal" fiber length, because at longer fiber lengths, PCSA continues to decrease hyperbolically, whereas sarcomere velocity decreases linearly. This diminishing sarcomere velocity increases force as fiber length increases, but the concomitant reduced PCSA decreases force with increasing fiber length, and the latter effect outweighs the former. At shorter fiber lengths, the increased sarcomere shortening velocity overpowers the increased isometric force potential, again resulting in lower dynamic force production. For the mouse tibialis anterior, dynamic tension was calculated to maximize at a fiber length (8.1 mm) that was very close to that observed by direct measurement (7.6 mm).[30] The tension produced by the observed 7.6-mm fiber was still 99% of maximum. These data reinforce the idea that in some muscles that experience cyclic and stereotypic activity, architecture is optimized to achieve maximal force production under the precise conditions that the muscle experiences. In the previous study considered,[30] fiber length was optimized in the soleus and medial gastrocnemius muscles, as well.

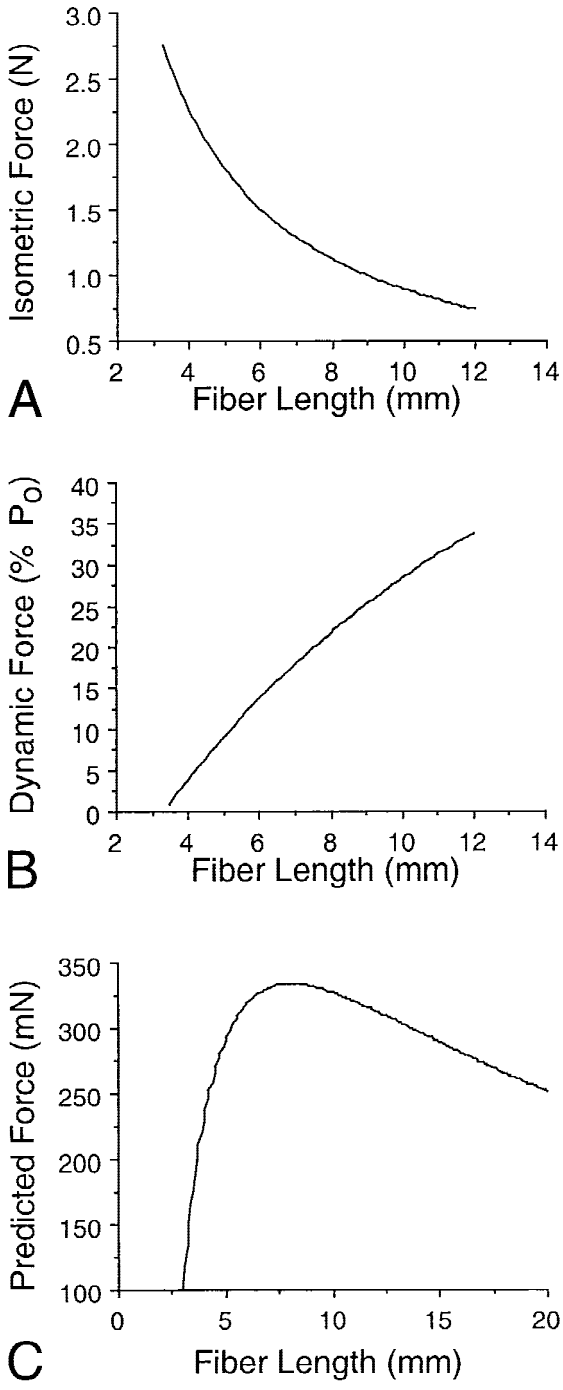

FIGURE 10. (A) Theoretical relationship between muscle fiber length and isometric force for a constant muscle mass. As fiber length increases, PCSA and thus P_o decreases nonlinearly. (B) Theoretical relationship between muscle fiber length and dynamic muscle force at a constant shortening velocity. Dynamic force increases due to decreasing sarcomere velocity. (C) Predicted force calculated to be generated by a tibialis anterior as fiber length is altered. Optimal fiber length results from a trade-off between decreasing sarcomere shortening velocity (which increases force) and decreasing muscle isometric force capacity as fiber length increases. (Data from Lieber.[31])

ARCHITECTURAL DESIGN OF SYNERGISTIC MUSCLES

Given the specific design of individual muscles, it might be expected that muscle placed in similar locations within the body might have similar architectures. This is a difficult statement to support and, in fact, a number of examples in the literature demonstrate that two muscles of divergent architectures are placed in synergistic locations.

Human Wrist Extensors. One fairly detailed example of divergent architecture was seen in the long and short extensors of the human wrist: the extensor carpi radialis longus (ECRL) and extensor carpi radialis brevis (ECRB). The ECRL muscle is the shorter muscle of the two but contains longer muscle fibers. The L_f/L_m ratio is relatively high (~0.8) and PCSA relatively low (~1.5 cm^2), leading to the assertion that the ECRL is a muscle designed for high excursion or velocity. The ECRB, on the other hand, is a longer muscle with shorter fibers and, therefore, a much lower L_f/L_m ratio (~0.4) but a higher PCSA compared with the ECRL (~2.7 cm^2). These data suggest that, from a muscle point of view, the ECRB is designed preferentially for high force production. Using rigid body kinematics, we quantified the moment arms of both muscles acting in wrist extension.[42] We wondered whether architectural differences between muscles might actually be compensated for by changes in wrist moment arm (r). We also measured the sarcomere length change in each muscle during wrist rotation in patients undergoing surgery for tennis elbow.[37] Sarcomere length change during joint rotation (i.e., $dSL/d\omega$) is a direct reflection of the relative ratio of the L_f:r ratio in a musculoskeletal system.[68] The interesting result of this study was that the skeletal kinematics actually accentuated the architectural differences between muscles. The extensor moment arm of the ECRB was much greater throughout the range of motion compared with that of the ECRL. Thus, muscle fiber length change with joint rotation was expected to be greater in the ECRB compared with the ECRL. This was confirmed by the intraoperative sarcomere length measurements that showed an average $dSL/d\omega$ for the ECRB of 9.1 nm/degree, whereas that of the ECRL was approximately half this value, or only 4.7 nm/degree. This led to a mechanical model of the wrist extensors (Fig. 11) explaining that the large $dSL/d\omega$ of the ECRB was due to the large moment arm and short fibers, whereas the small $dSL/d\omega$ of the ECRL was due to the small moment arm and long fibers. Using the architectural properties of the two muscles,[34] along with nominal values for the

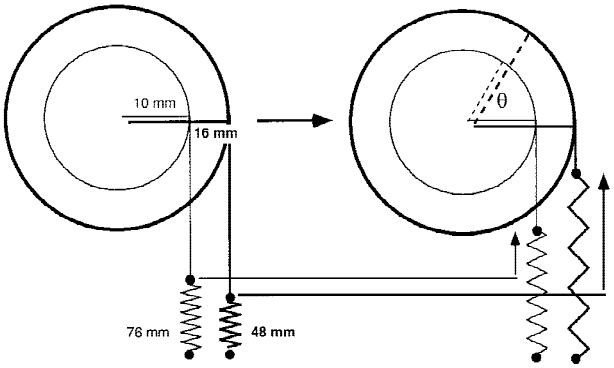

FIGURE 11. Schematic diagram of the interrelationship between fiber length and moment arm for the ECRB and ECRL torque motors. The ECRB (bold print, thick lines) with its shorter fibers and longer moment arm changes sarcomere length about 2.5 times as much as the ECRL, with its longer fibers and smaller moment arm. (Adapted from Lieber et al.[37])

V_{max} of mammalian muscle,[11] the force–velocity relationship for each muscle was also calculated. The contraction velocity at which the ECRL becomes stronger was calculated to be ~80 mm/s which, on the basis of the two muscle moment arms, corresponded to an angular velocity of ~240°/s. This results in a design in which the ECRB is stronger isometrically but, as angular velocity increases, the ECRL becomes the stronger muscle.

Differential muscle strength that is velocity-dependent may provide insight into the diversity of architectural design among muscles. The ECRB and ECRL muscles as a synergistic group can generate a maximum tetanic tension (based on the sum of the PCSAs) of 25.6 kg and can produce a maximum angular velocity (based on the length of the ECRL fibers) of approximately 2800°/s. In order for a single muscle to generate that much force while maintaining the same V_{max}, the fiber length would have to be 76 mm and the cross-sectional area would have to be 4.2 cm^2. Using eq. 1 to calculate muscle physiological cross-sectional area,[59] this single muscle would weigh 33.7 g, which is over 30% greater than the sum of the two muscle masses.[34] Having two muscles as synergists thus accomplishes the same task at the velocity extremes but with a lower mass than a comparable, single, "supermuscle." The design lesson is that it is more efficient from a mass point of view to have more highly specialized muscles than a single "supermuscle" that can accomplish the task of multiple muscles.

Rabbit Dorsiflexors. A similar disparity of architectural properties is also seen in the rabbit dorsiflexor muscles: the tibialis anterior and extensor digitorum longus. The tibialis anterior has relatively long fibers (~4.0 cm) and a small PCSA (0.6 cm^2), whereas the extensor digitorum longus has shorter fibers (~1.5 cm) and a higher PCSA (1.4 cm^2).[32] Interestingly, the former muscle crosses only the ankle joint, whereas the latter crosses the ankle, tarsal, metatarsophalangeal, and phalangeal joints. One might expect that, in general, muscles that cross multiple joints would have longer muscle fibers in order to provide the functional range required when numerous joints move simultaneously. In the case of the tibialis anterior and extensor digitorum longus of the rabbit, this is not the arrangement. The rationale for such diversity is not clear. As with the human ECRB/ECRL muscles, were a single "supermuscle" to be designed with the force-generating capacity of the sum of the two muscles and the overall excursion of the long tibialis anterior, the total mass would be significantly greater by 45%. Additionally, if synergistic motion of the ankle and toes were to routinely occur during the gait cycle, then short fibers of the extensor digitorum longus would not be a limitation and might simply represent a design that permits packing of a large number of fibers into a small volume, to generate high force.

Cat Triceps Surae. Architectural disparity in the cat triceps surae between the medial gastrocnemius and soleus muscles may have a more obvious functional role.[60,63] Cat soleus muscle fibers are fairly long: the soleus is composed of 100% slow muscle fibers and the muscle crosses only the ankle joint. In contrast, the medial gastrocnemius is composed of about 80% fast fibers, has relatively short fibers, and crosses both the ankle and knee joints. Based on joint kinematics measured during walking, it has been shown that the cat knee and ankle joints move in opposite directions during normal gait, which means that the medial gastrocnemius muscle length change during locomotion is minimized. Based on the known mechanical properties of muscle, high shortening velocities are unfavorable because they result in low muscle force, even when the muscle is fully activated. Therefore, the gastrocnemius can produce high muscle force, due to its large number of fibers packed at a relatively high pennation angle, even though the fibers are relatively short.

The soleus muscle is fairly active even during stationary standing in the cat and is nearly maximally activated when the cat is walking relatively slowly.[63] Because the soleus is uniarticular, moderate length changes and thus high shortening velocities are observed during locomotion, which can compromise muscle force. Long soleus muscle fibers "oppose" this tendency. That the soleus is composed of only

slow fibers makes the force depression due to shortening even more pronounced, as the muscle has a relatively low V_{max}. The triceps surae in the cat thus represents a system in which fiber type and muscle architecture appear to have developed in a complementary manner.

MUSCULOSKELETAL BALANCE

The previous discussion focuses primarily on torque production of a single musculoskeletal system. However, much of normal movement is predicated on effective and coordinated interaction between opposing muscle groups. Detailed muscle and skeletal data on agonist–antagonist pairs are largely lacking. However, based on the relatively complete data set regarding wrist muscle[34] and joint[42] properties that we have collected, a few general trends have emerged. Based on the intraoperative sarcomere lengths measured,[37,38] wrist extensors were predicted to operate primarily on the plateau and descending limb of their sarcomere length–tension curve, with all muscles generating maximal force in full extension. Only the ECRB was predicted to operate at sarcomere lengths corresponding to less than 80% P_o in the normal range of motion. Wrist flexors (flexor carpi ulnaris and flexor carpi radialis) were predicted to operate predominantly on the shallow and steep ascending limbs of their length–tension curve, with both major flexors generating maximal force in full wrist extension. In full flexion, it was possible for wrist flexors to generate forces that were less than 50% P_o only.

Such a design presents interesting implications for the design of the wrist as a torque motor. Both flexor and extensor muscle groups generate maximum force with the wrist fully extended. As the wrist moves from flexion to extension, maximum extensor force increases due to extensor shortening up the descending limb of the length–tension curve and maximum flexor force increases due to flexor lengthening up the ascending limb of the length–tension curve. This effect is superimposed upon an increasing extensor moment arm as the extensor muscles elevate off of the wrist under the extensor retinaculum and a decreasing flexor moment arm as the flexors juxtapose the wrist from the flexor retinaculum. Combining muscle and joint effects, extensor muscle force is amplified by an increasing extensor moment arm and flexor muscle force is attenuated by a decreasing flexor moment arm. Interestingly, because the flexors as muscles develop significantly greater force than do the extensors (due to their larger PCSA), the net result is a nearly constant ratio of flexor to extensor torque over the wrist range of motion. In fact, although at the muscular level, the flexors are considerably stronger than the extensors,[5,34] when including the wrist kinematics, extensor moment actually slightly exceeds flexor moment. Additionally, wrist resistance to angular perturbation increases as the wrist is moved to full extension, because both flexor and extensor moments increase in a similar fashion. This is another way of saying that the wrist is most mechanically stable in full extension, which is probably the reason that individuals who are asked to perform a power grip operation do so with the wrist extended. It should be noted that wrist torque balance is achieved at the expense of maximum moment generation at the wrist, and, therefore, we conclude that this musculoskeletal system is not designed simply to operate near maximum force, as is often assumed in musculoskeletal models.

PLASTICITY OF MUSCLE ARCHITECTURE

The fact that architecture varies between muscles within a species and is extremely consistent within a species implies that regulation of serial sarcomere number in muscles is ongoing. It is easy to demonstrate that muscle serial sarcomere number (and thus fiber length) varies rapidly with such treatments as immobilization,[66] tenotomy,[1] moment arm alteration,[29] and even eccentric exercise.[45] However, although muscle adaptation to these conditions is consistent and well described, the mechanical and cellular factors to which a muscle responds under these conditions are not yet fully understood.

In muscles that are used in stereotypical fashion, muscle architecture and joint kinematics seem to interact such that muscles produce near-maximal power during normal use, as described above. For example, the complex architecture of fish muscle[54] and the spatial segregation of fish red (slow) and white (fast) muscle[53] enable fish to maintain near-optimal sarcomere lengths and near-optimal power production at velocities normally seen during locomotion.[53] The very large frog semimembranosus muscle, which powers hopping, appears to have the appropriate architecture such that during the hop, the muscle generates near-maximal power at near-optimal sarcomere lengths.[43,44] Optimization of this relationship is not trivial. Because the moment arm transforms the linear muscle motion into angular joint rotation, the moment arm influences both the muscle length range and shortening velocity required to produce a given joint motion. Thus, the interaction between muscle architectural properties and joint moment arms is critical for producing the correct joint kinematics during locomotion.

Immobilization Studies of Architecture Alteration. One of the most simple and straightforward demonstrations of architectural change occurs secondary to limb immobilization. In a classic study that has greatly influenced modern thinking on this subject, Williams and Goldspink[66] immobilized the ankles of young (8-week-old) mice. Ankles were held in a fixed position for 3 weeks by means of a plaster cast, and the properties of the monoarticular soleus muscle was measured at the end of the immobilization period. The result was straightforward: regardless of the position of immobilization, the muscle changed its length–tension properties so that maximum isometric tension was observed at the muscle length corresponding to the angle of immobilization. These data could indicate that muscles "adapt" to chronic length changes by resetting sarcomere length to the optimum or simply that muscles adapt to reset the muscle to a specific sarcomere length. Architectural analysis revealed that this change in resting length was accompanied by a change in serial sarcomere number within the soleus fibers (Fig. 12). For example, whereas normal soleus muscles had about 2200 serial sarcomeres, when the ankle was immobilized in dorsiflexion (thus stretching the muscle), sarcomere number increased to almost 2600, and when the ankle was immobilized in plantarflexion (thus shortening the muscle), sarcomere number decreased to ~1800. Thus, the mouse soleus muscle had adjusted to its new mechanical environment by adding or deleting the appropriate number of sarcomeres such that optimal muscle length coincided with the length at which the muscle was immobilized.

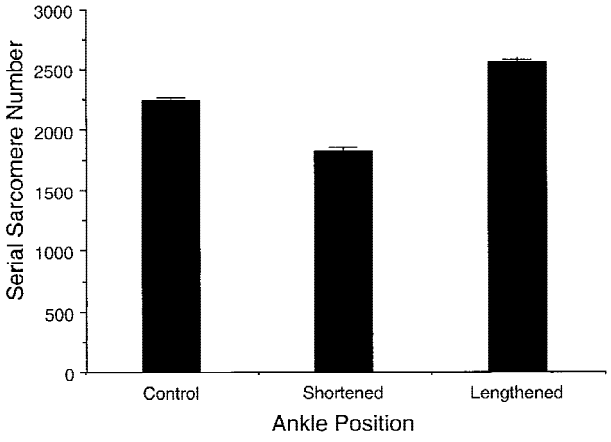

FIGURE 12. Sarcomere number in mouse soleus muscles of 8-week-old mice subjected to immobilization. Serial sarcomere number was determined after 3 weeks of immobilization with the muscle in the control, shortened, or lengthened position. (Data from Williams and Goldspink.[66])

Retinaculum Release Studies of Architecture Alteration. The studies of Williams and Goldspink[66] have been extended in concept by many physiologists and surgeons to indicate that muscles simply adjust serial sarcomere number to optimize force production. However, there are a number of reasons not to accept this generalization. First, it was demonstrated that although the soleus muscle may show such adaptations, not all muscles adapt in the same manner.[61] Thus, the basic nature of the adaptation was similar in the medial gastrocnemius as well as the tibialis anterior, but the magnitude of the response was greatly attenuated (Fig. 13). This study suggests that different muscles have different degrees of responsiveness to sarcomere number change. This should not be surprising, because different muscle groups demonstrate large differences in their tendency to either atrophy or hypertrophy in response to an altered mechanical environment.[14,35,57]

A difficulty in interpreting results from immobilization studies is that it is impossible to identify the particular signal to which sarcomere number adapts.[24] Sarcomere number may change within a muscle fiber so that optimal sarcomere length is determined by resting muscle length, by resting muscle tension, by an extreme of muscle length, or even by the length of most frequent or maximal muscle activation. Chronic length change models cannot distinguish between these possibilities. In an effort to extend this body of knowledge, length changes have been imposed upon muscles that are not immobi-

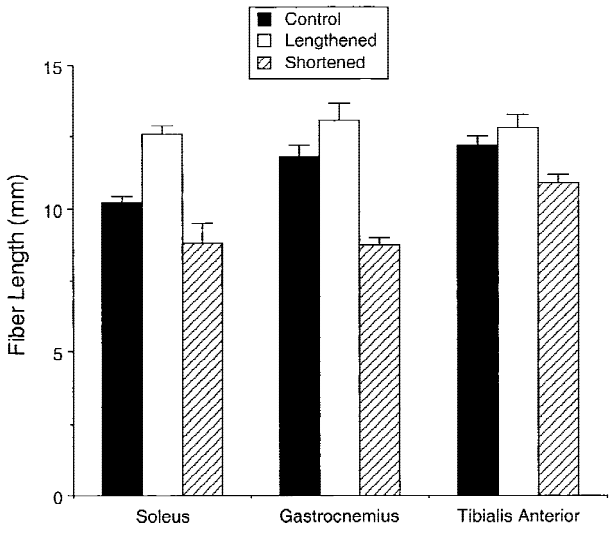

FIGURE 13. Fiber length of rat muscles immobilized in the neutral (filled bars), lengthened (open bars), or shortened (hatched bars) position. Note that muscles respond to immobilization in the same direction although with different magnitudes. (Data from Spector et al.[61])

lized, by transecting the ankle retinaculum and permitting the muscle an increased active excursion during normal movement. This procedure was imposed upon rapidly growing rabbits, and it was shown that muscle excursion was dramatically increased, as were whole-muscle length and fiber length, all in proportion to the increased excursion.[12] Unfortunately, the interpretation of these experiments is confounded by the rapid growth rate of these animals, which itself increases fiber length. Retinaculum release was performed in a separate stud, and the mechanical environment of the muscle was studied in vivo in much greater detail.[29] Interestingly, although the serial sarcomere number increase was also documented, the force generated by the released tibialis anterior as well as its PCSA actually decreased. These data may represent simultaneous adaptation of serial and parallel sarcomere number to complex mechanical stimuli. Neither the torque produced by the released tibialis anterior nor the ankle flexor moment arm were reported, but it is interesting to speculate that the tibialis anterior decreased its PCSA proportional to the increased moment arm to keep dorsiflexion torque constant.

In light of the functional importance of fiber length and the general lack of understanding of mechanical and biological factors that regulate fiber length, we developed a model in which the demands placed upon the muscle were altered so that sarcomere number could adapt to either length or excursion in a way that could be differentiated.[9] The ankle pretibial flexor retinaculum was surgically transected, and the tibialis anterior muscle was permitted to move away from the center of rotation of the ankle joint, increasing the tibialis anterior moment arm and decreasing muscle–tendon unit (MTU) length as the foot dorsiflexes. The mechanical effect on the muscle can be viewed as a chronic shortening as well as an increased excursion and velocity. Prior to retinaculum transection, the moment arm was relatively constant throughout the range of motion, as would be expected, because the tendon of the tibialis anterior passes beneath the flexor retinaculum. After transection, there was a notable peak during dorsiflexion due to the altered geometry allowing the tibialis anterior to "bowstring" in operated animals. The major effect of transection was to increase the moment arm at joint angles of less than 100°. Based on moment arm measurements, average MTU excursion increased from 1.4 mm in control animals to 1.9 mm in the transected groups over the full range of motion (50–150°). Despite the significant increase in maximum muscle length and significant decrease in minimum muscle length, both of which might be expected to lead to increased sarcomere number, sarcomere number decreased significantly over the 2-week experimental period, from 3200 ± 200 in control animals to 2900 ± 200 in mice 2 weeks after transection. Similar results were obtained after 2 weeks of swim training following retinaculum transection (2950 ± 170; $n = 4$), indicating that lack of tibialis anterior activation did not induce the results.

Video analysis of stepping in normal and retinaculum-transected mice revealed that 2-week transected mice preferentially dorsiflexed at slightly slower and more variable rates. Unoperated mice dorsiflexed fairly consistently, at 2200 ± 300°/s, which was significantly faster than 2-week retinaculum-transected mice (1900 ± 400°/s). Despite this 14% decrease in angular velocity after transection, the 30% increase in moment arm and significant reductions in dorsiflexion and plantarflexion muscle lengths resulted in a calculated increase of approximately 20% in the velocity of tibialis anterior shortening during dorsiflexion, from 2.7 to 3.3 $L_o\ s^{-1}$. The effect of altered sarcomere number is also reflected in the decreased power production calculated at the preferred gait speed. Whereas control mice were calculated to generate 92 ± 2% of peak tibialis anterior muscle power, 2-week transected mice were calculated to produce only 74 ± 5% of their peak muscle power in tibialis anterior. These data suggest that not all architectural adaptations that occur in a complex mechanical environment result in "reoptimization" of muscle properties. In the mouse retinaculum-release study, the only parameter that appeared to return to the initial condition was the minimum sarcomere length. Taken at face value, these data suggest that the muscle adapted in response to the underlying chronic change in length rather than the altered excursion or velocity. Obviously, the details of the mechanical factors regulating sarcomere number in muscle remain to be elucidated.

Architectural Change after Eccentric Contraction. One of the most extreme mechanical conditions under which a muscle may operate is forced lengthening of an activated muscle, i.e., eccentric contraction, when the muscle is not usually used in that manner. Chronic eccentric training of a muscle results in a number of changes, most of which are beyond the scope of this review. However, based on a theory that suggests that sarcomere length operating range should remain on the ascending limb and plateau of the length–tension curve, Morgan[46] predicted that a muscle subjected to eccentric training should add sarcomeres in series. This would have the

effect of shifting the muscle operating range to shorter lengths, perhaps even those on the ascending limb. This hypothesis was tested in an experiment in which rats were trained to either run downhill or uphill or remain sedentary. The downhill-running protocol, due to the extreme knee flexion angles reached, resulted in eccentric training of the vastus intermedius muscle. The uphill-training protocol controlled for the effect of exercise itself.[45] The authors demonstrated an increase in serial sarcomere number from about 3250 in sedentary controls to about 3500 in decline runners. This supported their underlying assumption that muscles adapt such that they will not be stretched on the descending limb of the length–tension curve. Although these data explain the behavior of the rat vastus intermedius, some muscles appear to be designed to operate on the descending limb of the length–tension curve. For example, based on intraoperative sarcomere length measurements, the human ECRB operates almost exclusively on the descending limb of the length–tension curve during wrist joint rotation.[38] Thus, it is difficult to define a stereotypical sarcomere length operating range for muscles. In a recent survey of 83 separate studies covering a total of 51 different muscles in eight different species, the average minimum sarcomere length was 82 ± 17% of optimal length (L_o) and an average maximum length of 119 ± 21% L_o[8]; 90% of maximum sarcomere lengths fell within 92–167% L_o. The average sarcomere length of 100 ± 14% L_o across the entire review suggests that L_o ± 14% L_o would make a reasonable first approximation for a "generic" muscle operating range in the absence of other data.

In summary, this review has presented basic definitions of skeletal muscle architecture as well as a summary of the architectural properties of human upper and lower limb muscles. The varying architectural design of human and other mammalian muscles was used to illustrate the fact that muscles can be "designed" to perform fairly specific functions. Finally, muscle architecture can change with altered muscle use or in the face of a new mechanical environment. A detailed understanding of the mechanical and biological factors that regulate such adaptation will undoubtedly be the source of future studies.

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