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The Architectural Design of the Gluteal Muscle Group: Implications for Movement and Rehabilitation

omplex interactions between the nervous, muscular, and skeletal systems produce the wide variety of movements available for human task execution. Each of these systems is often studied in isolation, but movement scientists and clinicians are forced to integrate the entire neuromusculoskeletal system to understand functional limitations and prescribe viable treatments. Although this approach is appropriate for clinical decision making, the details of each system are often overlooked, perhaps at the expense of accurate and/or innovative treatment approaches. The purpose of this paper was to describe the fundamental organization and physiology of the muscles controlling hip movement.

In the muscular system, the organization of fibers within a muscle is highly predictive of its functional capacity.^{3,4,25} The organizational parameters (eg, normalized fiber length and physiological cross-sectional area), termed muscle architecture, are often used to identify muscles important in movement and stability.^{6,21,30,32,34} While other physical parameters, such as muscle mass and volume, and other metabolic parameters, such as fiber type distribution, substan-

• SYNOPSIS: The organization of fibers within a muscle (architecture) defines the performance capacity of that muscle. In the current commentary, basic architectural terms are reviewed in the context of the major hip muscles and then specific illustrative examples relevant to lower extremity rehabilitation are presented. These data demonstrate the architectural and functional specialization of tially influence contractile properties, none predict muscle function as well as muscle architecture.^{4,20} Architectural data, particularly in humans, are used extensively to understand muscle-joint behavior⁵ and to make surgical decisions.¹⁷ Although the architecture of the hip muscles has been documented,^{6,34} these studies are limited in terms of size and, in some cases, lack appropriate methods that confound accurate interpretation of the data. Here we present the basic

the hip muscles, and highlight the importance of muscle physiology and joint mechanics when evaluating and treating musculoskeletal disorders. *J Orthop Sports Phys Ther* 2010;40(2):95-102. *doi:10.2519/jospt.2010.3302*

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structure-function relationships of skeletal muscle and discuss these features in the context of a comprehensive data set of hip muscle architecture.³⁰ To provide clinical context for these data, we also present a brief illustrative description of muscle-joint interactions during a common movement task and discuss their utility in rehabilitation.

MUSCLE ARCHITECTURE

HE VISUAL APPEARANCE OF SKELetal muscle is familiar to most clinicians. It is a mass of organized muscle fascicles, covered in connective tissue, connected at each end to bones through tendons. The sizes of muscles and tendons vary considerably; but a key concept is that, macroscopically, the eye is drawn to the volume or mass of a muscle. For example, the gluteus maximus is larger in volume than the gluteus medius or minimus. The volume of a muscle is dictated, in large part, by the total number of sarcomeres contained in that muscle. Sarcomeres are approximately 1 μ m in diameter and 2 to 3 μ m long. These functional units of muscle are stacked end-to-end (serially) to form myofibrils. Myofibrils are packed side-by-side (in parallel) to form muscle fibers (approximately 50 μ m in diameter in humans). Muscle fibers are packed in parallel to

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form fascicles (approximately 1 to 3 mm in diameter in humans), which are again packed in parallel to form muscles (centimeters in diameter in humans).

The organization of muscle fibers (and therefore sarcomeres) relative to the axis of force generation (tendon) is called muscle architecture.7 That is, muscle architecture defines how sarcomeres (the functional units of muscle) are packed into a muscle. There are essentially 2 extreme options: sarcomeres packed end-to-end (in series) or side-byside (in parallel). The first option, packing sarcomeres in series, yields very long muscle fibers, which would allow a muscle to change lengths over large distances and at relatively high velocities (FIGURE 1).^{3,19,29} This architectural parameter is termed normalized muscle fiber length. The normalization process, described below, simply allows fiber lengths to be compared at the same sarcomere length. The second option, packing sarcomeres in parallel, yields higher forces because more sarcomeres are simultaneously pulling on the tendons (FIGURE 1).²⁵ This calculated parameter is called physiological cross-sectional area (PCSA). In humans and animals, the architectural features of muscle are somewhere in between these 2 extremes, which allow muscles to change length and to generate enough force to accomplish the functional demands imposed on them. Importantly, these architectural features (fiber length and PCSA) provide a nearcomprehensive picture of the functional capacity of a muscle.^{3,4,19,25} Muscles with large PCSAs and short fiber lengths generate large forces over small length changes, while muscles with small PC-SAs and long fibers generate small forces over large length changes. Functionally, we would categorize these 2 examples as muscles designed for "stability" or "excursion," respectively.

The methods used to calculate these architectural parameters have been well described²¹ but are important to understand. First, muscles are isolated and connective tissue is sharply removed



compared to a longer-fibered muscle (B). A muscle with few fibers in parallel (B) produces less maximum isometric force compared to a muscle with more fibers in parallel (C). These concepts can also be illustrated in the context of force-length curves, where relative tension (expressed as a percentage of its maximum isometric tension; P_0) is plotted against length (D). For example, muscle B has twice the excursion and the same force as muscle A and muscle C will produce 3 times the force and will have the same excursion as muscle B.

from the contractile tissues. A series of small muscle fiber bundles are isolated and their pennation angles are measured. The fiber bundles are then dissected from the muscle and their lengths measured. Following this procedure, sarcomere length is measured using laser diffraction.³⁶ A normalized fiber length is then calculated for the isolated bundles according to the following equation²²:

$$L_f = L_f' \left(\frac{2.7 \,\mu \mathrm{m}}{L_s'} \right)$$

Where L'_f is the measured fiber length, L'_s is the measured sarcomere length in each fiber bundle, L_f is normalized muscle fiber length, and 2.7 μ m represents the optimum sarcomere length for human muscle.²² This normalization procedure is important because muscles are fixed at a variety of lengths in vivo. For example, a muscle fixed in tension, like the hamstring muscles, will appear to have relatively long fiber lengths compared to a muscle fixed in a slack position, like

the quadriceps. These long (hamstring) and short (quadriceps) sarcomere lengths are dictated by the inherent sarcomerelength joint angle configuration of each individual muscle. In this specific case, when the hip and knee are at zero degrees of flexion, the hamstrings have relatively long sarcomere lengths compared to the quadriceps. The normalization procedure allows fiber lengths from any muscle to be compared, regardless of the amount of tension on that muscle at the time of fixation.

PCSA is calculated according to the following equation²⁵:

$$PCSA(cm^{2}) = \frac{M(g) \times \cos\theta}{\rho(g \div cm^{3}) \times L_{f}(cm)}$$

Where M is muscle mass, θ is pennation angle, ρ is muscle density (1.056 g ÷ cm³),³³ and L_f is the normalized muscle fiber length calculated above. This calculated value is an estimate of the sum of all of the muscle fiber cross-sectional areas in a muscle and it is dependent on

an accurate calculation of normalized fiber length (L_{a}) . As described above, calculation of L_{ℓ} requires a measurement of sarcomere length. Therefore, PCSA cannot be determined by simply measuring the anatomical cross-sectional area of a muscle (eg, from an MRI cross-section). Determination of L_f requires invasive measurements, and true calculations of PCSA have, to date, been limited to cadaveric specimens. Assessment of PCSA in individual patients has therefore been impossible. However, new minimally invasive imaging techniques²³ may allow for in vivo sarcomere length determination, enabling us to calculate PCSA from noninvasive MRI-based measurements of muscle volume and L_c

Recently, we studied the architecture of the major hip muscles in 20 cadaveric specimens.³⁰ These data demonstrate a number of important concepts. First, the trade-off between PCSA and fiber length can be readily observed (FIGURE 2). For example, the gluteus medius muscle has a large PCSA and relatively short fibers compared to the sartorius muscle, which has very long fibers and a very small PCSA. Using these 2 muscles as an example, the gluteus medius appears to be designed to stabilize the hip by generating very large forces over a narrow range of lengths, in contrast to the sartorius, which appears to be designed to generate much smaller forces over a much wider range of lengths. A few muscles, like the gluteus maximus and adductor magnus, have large PCSAs and long fibers, suggesting that they are expected to generate large forces over a wide range of lengths. These 2 muscles are likely to be clinically meaningful, as we may rely heavily upon them to generate force throughout the range of possible hip motions. Other muscles, like the short external rotators, have large PCSAs and very short fibers, suggesting that they are designed to stabilize the hip and pelvis. These muscles may also be clinically meaningful because they are, perhaps, responsible for providing minor position adjustments to the hip



FIGURE 2. Scatter plot of muscle normalized fiber length versus physiological cross-sectional area (PCSA) for the major hip muscles. Increasing muscle fiber length is associated with increasing excursion and velocity capacities in a muscle, while increasing PCSA is associated with increasing maximum isometric force-producing capacities. Gluteus medius (GMed), gluteus maximus (GMax), adductor magnus (Add M), adductor longus (Add L), adductor brevis (Add B), semitendinosus (ST), semimembranosus (SM), biceps femoris long head (BFLH), biceps femoris short head (BFSH), rectus femoris (RF), iliacus, psoas, sartorius (SR), gracilis (GR), piriformis (Piri), quadratus femoris (QF), obturator internis (OI), pectinius (Pect), internal gemellus (IG), obturator externus (OE), and superior gemellus (SG) are depicted. Data are presented as mean ± SE and adapted from Ward et al³⁰ and Friederich and Brand.⁵

and/or modulating joint stiffness, in effect, "tuning" the hip joint. Importantly, the masses of these muscles tell you relatively little about their performance. For example the gluteus maximus muscle has the largest mass, but not necessarily the largest force-producing capacity because it has very long fibers.

MUSCLE PHYSIOLOGY

LTHOUGH ARCHITECTURAL FEAtures are important for describing the functional capacity of muscle, it is important to recognize that muscles are exposed to length changes and velocities during movement, and both of these change absolute force production. For example, the previously described architectural feature PCSA is predictive of maximum isometric force-generating capacity at optimal muscle length.²⁵ However, force-generating capacity changes with muscle length. The well-described force-length relationship of muscle⁸ sug-

gests that it produces maximum force isometrically when there are a maximum number of cross-bridges formed between actin and myosin. This occurs at optimum myofilament (actin and myosin) overlap. In humans, this is between 2.6 and 2.8 μ m (FIGURE 3A).²² At longer lengths, fewer cross-bridges are formed and there is less active force production. This region of the length-tension curve is called the "descending limb." Importantly, passive force is also generated by the noncontractile elements of muscle in this region. At shorter lengths, again, there are fewer cross-bridges formed due to overlapping actin filaments and less force is produced. This region is called the "ascending limb."9,10,26

The force-generating capacity of a muscle is also greatly influenced by velocity. As muscle begins to actively shorten, force declines precipitously (**FIGURE 3B**). When muscle actively lengthens, force rises to levels greater than can be achieved isometrically (**FIGURE 3B**). These

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FIGURE 3. The shape of the force-length relationship (A) is dictated by the amount of actin and myosin overlap in a sarcomere. At optimal length (2.6-2.8 μ m in humans) there is maximum overlap and maximum isometric force production. At shorter sarcomere lengths, there are fewer cross-bridges formed and therefore less force production. This region is termed the ascending limb of the force-length curve. At longer lengths, again, fewer cross-bridges are formed and there is less active force production. This region is termed the descending limb of the force-length curve. Importantly, passive tension begins to rise on the descending limb of the length-tension curve. Muscle is also very sensitive to velocity and this physiological property of muscle is characterized by the force-velocity curve (B). Force-producing capacity falls rapidly as the muscle shortens at progressively higher velocities, while force increases to a level beyond isometric maximum when the muscle actively lengthens. Notice that muscle is not particularly sensitive to lengthening velocity, as force is relatively constant as lengthening velocity increases.

relationships have also been characterized extensively.¹³ The rationale for force decline during active shortening is thought to be related to fewer crossbridges being formed when actin and myosin are moving relative to each other. During active lengthening, the increase in force production is thought to be related to the mechanical energy needed to physically break the connection between actin and myosin, although this is the focus of much current work in basic muscle mechanics.

Although these relationships are dogmatic in the literature, it is important to understand that they are achieved under well-controlled, maximum activation of isolated muscle systems. In vivo, the relationships change slightly because of submaximal muscle activations,^{14,27} the interactions between muscles and connective tissues,¹⁵ and tendon elasticity.¹⁸ However, they should be considered as an accurate general framework for muscle physiology.

MUSCLE-JOINT INTERACTIONS

LTHOUGH MUSCLE ARCHITECTURE and physiology explain muscle function in isolation, human movement requires muscles to operate in the context of the skeleton. Specifically, the organization of a joint has a profound impact on the function of muscle. For example, most are familiar with the concept of torque (force \times moment arm), which is, in fact, how muscles generate motion. A simple yet important concept is that moment arms serve to magnify or attenuate the torque-generating capacity (strength) of a muscle or muscle group. However, it is also important to understand the moment arms change with joint position and they directly influence the amount of length change imposed on a muscle as a joint rotates. This has 2 key consequences on muscle force-generating capacity. First, large moment arms are associated with large muscle length changes. As muscles are forced to shorten and lengthen over large sarcomere length ranges, force-generating capacity is compromised (FIGURE 4). As an aside, the precise relationship between joint angle and sarcomere length for each lower extremity muscle is largely unknown but is the focus of current research in our laboratory. However, we do know from work

in the upper extremity and spine that optimal muscle length does not necessarily occur in the anatomically neutral joint configuration.^{22,31,32} Second, large moment arms are associated with higher muscle velocities for a given joint angular velocity. As muscles are forced to operate at higher velocities (at least shortening velocities), force-generating capacity is also compromised. Therefore, large moment arms do not necessarily guarantee high torque-generating capacity. Importantly, the complex interaction between muscles and moment arms is a key reason why musculoskeletal models are used to understand movement.

HIP MUSCLE ARCHITECTURE, MUSCLE-JOINT INTERAC-TIONS, AND MOVEMENT

0 ILLUSTRATE THE COMBINED effects of muscle architecture, moment arms, and force-velocity relationships, we have modeled the potential moment-producing capacities of the gluteus maximus (sagittal plane), gluteus medius (frontal plane), adductor magnus (sagittal plane), and the hamstrings (sagittal plane), during a simple squat task (FIGURE 5). We use the term "potential" specifically because we are modeling the moment-generating capacity of each muscle, assuming maximum activation of that muscle, which is clearly not the case during normal movements. However, this exercise does allow us to compare the relative strength capacity of each muscle. The task involves squatting to 90° of hip flexion and 90° of knee flexion, and is performed relatively slowly. This speed allows us to consider the effects of muscle architecture, moment arms, and muscle length quite easily within the theoretical framework of the model. Although more sophisticated musculoskeletal models are available for the hip,² here we have used the standard SIMM (software for interactive musculoskeletal modeling) model.5

These data presented in **FIGURE 5** illustrate a few key concepts. First, the gluteus maximus and adductor magnus exert sim-



FIGURE 4. The relationship between moment arm and muscle fiber length is complex. The amount of length change imposed on a muscle (dashed lines) is directly related to the size of the muscle moment arm. Therefore, if we consider 2 hypothetical muscle-joint systems (left and right) with identical muscle fiber lengths but different moment arms, we would expect the muscle with the larger moment arm to operate over a wider range of the length-tension curve (P_0 is maximum isometric tension). Additionally, assuming the angular velocity of the joint is the same, we would expect the muscle with the larger moment arm to be operating at higher velocities. Both of these interactions would be expected to compromise the force-generating capacity of the muscle.

ilar maximum extension torques at the hip in the sagittal plane, despite the fact that gluteus maximus has a larger PCSA. This is due to the fact that adductor magnus has a larger hip extension moment arm when the hip is flexed. Importantly, the adductor magnus retains its ability to generate hip extension as the hip is flexed, unlike the gluteus maximus (FIGURE 5A AND 5C). Second, the combined efforts of the hamstring muscles produce a much larger hip extension moment (nearly 2-fold higher) than the gluteus maximus. This is due to the larger combined PCSA of the hamstrings compared to the gluteus maximus and the fact that the hamstrings have larger hip extension moment arms when the hip is flexed. This is important because the combined effect of hip flexion (which tends to lengthen the hamstrings) and knee flexion (which tends to shorten the hamstrings) offset each other enough

to limit net muscle length changes. Third, the gluteus medius produces nearly the same torque as the gluteus maximus but in the frontal plane (abduction).

To illustrate the effect of kinematic changes on torque-generating capacity, we simply added 15° of internal rotation to the hip during the same motion. Based on the model, it can be readily observed that this movement change impairs the hip extension torque generated by the gluteus maximus, but increases the abduction moment capacity of the gluteus medius when the hip is in flexion (FIGURE 5A AND 5B). This is an intriguing concept, because it is commonly believed that this movement pattern is indicative of hip weakness, particularly in patients with patellofemoral joint dysfunction28 and subjects at risk for anterior cruciate ligament injury.¹⁶ Perhaps it is a compensation for weak abductor muscles, as internal rota-

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FIGURE 5. The interaction between muscle architecture, muscle physiology, and joint mechanics is complex. Therefore, the influence of each of these properties is often evaluated within the context of a musculoskeletal model. Using a simple lower extremity model,⁵ we characterized the hip extension moment-generating capacities of the gluteus maximus (A), adductor magnus (C), and hamstrings muscle group (D) and the hip abduction moment-generating capacity of the gluteus medius (B) during a simple squatting task (solid blue line). These data are compared to a squatting task with 15° of internal rotation at the hip (dashed orange lines). It can be observed that the hip extension capacity of the gluteus maximus declines, while the hip abduction movement capacity of the gluteus waiting to variation. Although this is a hypothetical scenario depicted with a generic musculoskeletal model, it illustrates that small changes in movement can influence the physiology of the system.

tion will increase the torque-generating capacity of the gluteus medius, without changing the architectural features of the muscle. This has been demonstrated previously in children with cerebral palsy.¹² Also, the net effect on hip extension would be relatively minor, as the hamstrings, gluteus maximus, and adductor magnus all contribute to hip extension moment-generating capacity. Although these muscle-level changes seem to fit previously described movement patterns observed in patients, it should be noted that within the framework of this model the changes are fairly small and appear to occur deeper in hip and knee flexion

than expected.

This modeling approach is useful for guiding further research in the area of movement disorders, but should not be considered empirical data. The model itself has a series of assumptions built into it (for example, the architectural features of the muscles, the range in which they operate on the length-tension curve, the moment arms of each muscle, and the assumption of maximal activation for each muscle). Additionally, it is important to recognize that this amount of internal rotation is greater than the amount reported in patients with patellofemoral dysfunction²⁸ and is constant throughout the squatting task. However, internal rotation is also a difficult measurement to make in vivo, so a precise representation of patient-specific kinematics is problematic. All of these factors can change on a subject-by-subject basis. Although many of these assumptions have been tested in cadaveric specimens, others are unknown (ie, the sarcomere length-joint angle relationship of each muscle). These problems are the focus of much work in the area of musculoskeletal biomechanics. While such models are being validated, such thought exercises should be used to guide empirical experiments.

IMPLICATIONS FOR REHABILITATION

USCLES ARE THE ORGANS THAT directly produce movement. Therefore, a comprehensive understanding of their design and physiology is critical for the precise identification of (1) sources of pathologic movement and (2) targets of exercise interventions. For example, understanding that the gluteus maximus, adductor magnus, and hamstrings have the potential to generate the vast majority of hip extension torque will allow the clinician to rapidly assess the strength of these muscles when hip extension weakness is suspected. If hip extension weakness is found when the hip is flexed, adductor magnus and the hamstring muscles would be the most likely culprits, because they are capable of generating the most torque in this joint position. Similarly, gluteus medius is the dominant hip abductor and should be assessed when hip abduction weakness is suspected.

The architecture of these muscles is also important when assessing and targeting these muscles. For example, the gluteus medius produces an exceptional amount of force given its size. This is accomplished by packing many short fibers in parallel. The consequence, however, is that the muscle is not designed to produce very large forces over a wide range of lengths (or hip positions). Basically, it is designed to stabilize the femur and pelvis. As such, force production is likely to be compromised at extreme joint positions. This is important because a clinician may choose to test the muscle in a position that impairs muscle force production, like hip abduction or adduction. However, overloading the muscle for hypertrophy is most efficiently accomplished at relatively long lengths²⁴ and during eccentric exercise,11 which means hip adduction for this muscle. Efficient strengthening of the gluteus maximus would require eccentric contractions with the hip in flexion (activation of the muscle at a relatively long length). Similarly, because hip and knee flexion work cooperatively to dampen length changes in the hamstrings, squat exercises may not be the most efficient strengthening intervention. In this particular case it may be more efficient to place the hip in a fixed amount of flexion and load muscle during dynamic knee flexion (ie, leg curls). However, these are all ideas based on the physiology and mechanics of the muscles and should be tested experimentally.

Muscles operate within the context of a particular joint system. The hip is capable of motion in all 3 planes, so uniplanar and multiplanar movements are likely to change the force-length characteristics and moment arms of each muscle. Although the details of how each muscle changes is the focus of current research, it should be appreciated that kinematic changes may positively or negatively impact the force-producing capacity of a muscle. This is illustrated in the squatting example presented above. The addition of hip internal rotation negatively impacts the gluteus maximus but positively impacts the gluteus medius. The cause of this common movement problem (particularly subjects with patellofemoral joint dysfunction²⁸) remains enigmatic, meaning it is unclear if internal rotation is a strategy to improve the momentgenerating capacity of a weak gluteus medius or the consequence of a weak gluteus maximus. However, it is clear that this movement solution may impair hip

extension moment generation and it allows the gluteus medius to generate less force. Both should be considered problematic because it is clear that muscles negatively adapt (atrophy) when loading is reduced.³⁵ However, we do know that muscles positively adapt (hypertrophy) when subjected to new, higher loading environments.^{1,24}

SUMMARY

ONTROL OF THE HIP DURING MOVEment involves complex interactions between the nervous, muscular, and skeletal systems. Here we have illustrated that architectural features of muscle are directly related to the functional demands imposed on that muscle. These features characterize a muscle in isolation and should be considered in the context of the entire musculoskeletal system when movement disorders are being analyzed. However, the individual characteristics of a muscle shed light onto the functional "design" of a muscle. These characteristics can be used to identify muscles that are potentially weak and can be used to target muscles for strengthening when movement disorders are identified.

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