Knee joint malalignment has been considered to be one of the factors that may contribute to musculoskeletal pathology of the knee joint itself, as well as the hip and the foot. Hungerford and Barry (18) using the quadriceps angle (Q angle), the angle that is formed between the line of action of the quadriceps muscle and the direction of the patellar tendon (Fig. 1), as an indicator of knee joint malalignment, stressed that when the knee joint is not properly aligned, such as in the case of a valgus knee, the quadriceps pulls the patella more outward and laterally than normal. Other authors have shown that Q angles higher than 15° for men and 20° for women are related more to patellofemoral pain and dysfunction of the extensor mechanism of the knee (1, 21). The coexisting changes in the contact surface of the patellofemoral joint in these individuals may predispose to chondromalacia (17, 23) and patellar subluxation or dislocation (18). The mechanism that may underlie the abnormal drive of the patella into the trochlea notch of the femur leading to knee pain and dysfunction may be related to a less effective pull of the vastus medialis (VM) relative to the vastus lateralis (VL), probably due to pain inhibition and/or muscle atrophy.

To date, the majority of research studies focusing on the effectiveness of the quadriceps muscle have been performed on symptomatic individuals, regardless of knee joint malalignment (7, 25, 34, 41, 45). The results of these studies revealed a reduced EMG activity of the VM oblique (VMO) relative to VL, as it was shown by the alteration of VMO-to-VL ratio activation patterns (7, 41). A reversal in the firing sequence of these muscles has been also reported in patients with patellofemoral pain syndrome, compared with the control group (45). Based on this evidence, research suggests that the neuromuscular imbalance may have its origin in an abnormal mechanical alignment (7, 30, 41, 45).

Furthermore, it remains unclear whether the lower EMG activity of the VM relative to VL was due to pain inhibition and/or muscle atrophy that preceded the onset of pain symptoms. The development of computed tomography has enabled researchers to determine the force-generating capacity of a muscle, based on the widely accepted concept that the maximum force that can be produced by a muscle is directly proportional to its cross-sectional area (CSA) (3, 22, 26). Strength changes of a muscle based on changes of its CSA have been monitored in several previous studies to determine whether that particular muscle was affected by aging (11, 27), training, or detraining (15, 16, 32) and disuse (e.g., bed rest, immobilization) of a body part (4, 35). To our knowledge, there is no information concerning the possible effect of knee joint malalignment on the CSA of the quadriceps muscle, neither in healthy individuals nor in individuals with knee joint pain and dysfunction. The purpose of the present study was to determine whether the CSA of the individual parts, as well as the entire quadriceps muscle, was altered in individuals with knee joint malalignment. To establish whether such possible changes preceded, or followed the onset of pain symptoms, measurements of the CSA of the quadriceps were performed on a sample of nonsymptomatic individuals. Such information would be of clinical importance, because it can improve both prevention and rehabilitation programs in patients with knee joint pain and dysfunction.

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METHODS

Subjects

Thirty-six healthy men were selected for this study, based on the magnitude of the Q angle, from a population of 265 first-year healthy male students of the Department of Physical Education and Sports Sciences. Setting a cutoff Q angle of 15°, subjects were divided into two groups. The first group was consisted of 17 subjects with Q angles lower than 15°, and the second group included 19 subjects with Q angles higher than 15°. Before selection, each subject underwent a thorough clinical assessment and was questioned about past medical history and the level of habitual physical activity. The physical activity level was quantified, using the kinetic activity assessment questionnaire (2). Subjects with postural deviations, such as leg length discrepancy, recurvatum knees, muscle shortening, kyphosis and/or scoliosis, post surgery, injury of the lower limbs or patellofemoral pain syndrome, thyroid dysfunction or rheumatic conditions, obesity, use of anabolic drugs and/or participation in organized athletic activities, or increased level of kinetic activity (>8 h/wk), were excluded from the experimental procedure. All subjects participated voluntarily, after being informed of the purpose of the study, signing a written consent. The experimental procedure was approved by the ethics committee of the University of Athens.

Procedures

Q angle determination. The Q angle was measured in the dominant lower leg of the subjects, using a simple full-circle goniometer with a lengthened arm. Leg dominance was determined based on their individual preference when asked to kick a ball. Each subject was required to lie in supine position with the knees fully extended and the quadriceps relaxed. The foot was in a standardized position, so that the line connecting the middle of the heel with the second metatarsus was perpendicular to the ground (12), because the positioning of the foot in terms of inward-outward rotation influences the magnitude of the Q angle (33). Three landmarks were placed (8 mm in diameter), after palpation of the anterior superior iliac spine, at the center of the patella and the tibial tubercle. The patellar center was located at the intersection of a mediolateral line extending through the widest area of the patella and a superoinferior line connecting the base and the tip of the patella. The long arm of the goniometer was placed along the line connecting the anterior superior iliac spine with the center of the patella and the short arm along the line, connecting the center of the patella with the tibial tubercle. To determine interexaminer reliability, the landmarks were detached after each measurement, and the whole procedure was repeated a few minutes later by a second examiner who was not aware of the Q angle measured by the first examiner. A few days later, the whole procedure was repeated by the first examiner, to determine intraexaminer reliability.

Magnetic resonance imaging. A CSA of the quadriceps muscle of the dominant side was measured via magnetic resonance imaging (MRI) using a 1.0-T imager (Impact, Siemens, Erlangen, Germany). The MRI was used because it provides maximum accuracy of a muscle’s cross section in vivo measurements (31) and is radiation free. Each subject was placed in supine position. The knee of the dominant lower leg was placed in the commercial knee coil provided by the magnet manufacturer (quadrature-knee coil, a transmit-receive single-channel coil). The knee was supported and fixed in the coil using MRI-compatible cushions, also commercially available. The lower leg was also fixed, and the plantar surface of the foot was attached to a vertical MRI-compatible Plexiglas device to keep the foot fixed in this position. The hip was placed in a middle position in terms of rotation, while the knee was in a 10° flexion. The special knee coil was used for the static imaging (with the quadriceps relaxed at ~10° flexion) and dynamic imaging (with isometric quadriceps contraction) of the knee at 0° extension. Images were obtained using longitudinal relaxation time- and transverse relaxation time-weighted spin echo standard techniques. Additional three-dimensional volume images were obtained with 1.6-mm-thick slices. All measurements were performed using the Numaris MRI computer software and were performed by an experienced radiologist who was not aware of the Q angle group classification of each subject.

Axial images were obtained and the anatomic CSA (aCSA) of the following muscles were measured, in relaxation and contraction: VM, VL, vastus intermedius (VI), rectus femoris (RF), and the total...
Table 1. Descriptive anthropometric characteristics and physical activity level of each group

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>HQ Angle (n = 19)</th>
<th>LQ Angle (n = 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>20.0±1.6 (19.3)</td>
<td>19.3±0.9 (18.9)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.8±0.1 (1.8)</td>
<td>1.8±0.1 (1.8)</td>
</tr>
<tr>
<td>BW, kg</td>
<td>72.0±9.6 (70.0)</td>
<td>73.4±10.0 (72.0)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>23.0±2.4 (22.5)</td>
<td>23.7±2.3 (22.9)</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>18.6±4.5 (18.3)</td>
<td>18.0±3.7 (16.9)</td>
</tr>
<tr>
<td>Body fat, kg</td>
<td>13.7±4.9 (12.4)</td>
<td>13.4±4.5 (12.1)</td>
</tr>
<tr>
<td>LBM, kg</td>
<td>58.3±5.5 (57.5)</td>
<td>59.9±6.3 (59.2)</td>
</tr>
<tr>
<td>LA, 0–15 scores</td>
<td>8.9±1.1 (8.8)</td>
<td>8.9±1.1 (9.0)</td>
</tr>
</tbody>
</table>

Values are means ± SD with medians in parentheses; n, no. of subjects; LQ angle, Low quadriceps (Q) angle group; HQ angle, High Q angle group; BW, body weight; BMI, body mass index; LBM, lean body mass; LA, level of activity.

Table 2. Differences in each anatomic cross-sectional area of muscles and patellar tendon between the HQ angle and LQ angle group in static and dynamic imaging

<table>
<thead>
<tr>
<th></th>
<th>Static Imaging</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>aCSA, cm²</td>
<td>HQ Angle</td>
<td>LQ Angle</td>
<td>%Diff</td>
<td>HQ Angle</td>
<td>LQ Angle</td>
<td>%Diff</td>
</tr>
<tr>
<td>TQ</td>
<td>74.9±8.9 (74.9)</td>
<td>83.1±7.9 (81.9)*</td>
<td>9.9</td>
<td>72.0±8.7 (73.3)</td>
<td>80.6±7.6 (80.7)†</td>
<td>10.7</td>
</tr>
<tr>
<td>VM</td>
<td>19.3±2.6 (19.0)</td>
<td>21.5±3.0 (20.5)</td>
<td>10.2</td>
<td>20.0±2.5 (20.0)</td>
<td>22.7±2.7 (22.5)†</td>
<td>11.9</td>
</tr>
<tr>
<td>VI</td>
<td>24.3±4.7 (23.6)</td>
<td>27.9±4.0 (28.2)*</td>
<td>12.9</td>
<td>23.3±4.2 (23.5)</td>
<td>26.9±3.9 (27.0)†</td>
<td>13.4</td>
</tr>
<tr>
<td>VL/VM</td>
<td>1.3±0.3 (1.3)</td>
<td>1.3±0.3 (1.3)</td>
<td>0.0</td>
<td>1.2±0.2 (1.2)</td>
<td>1.2±0.2 (1.8)</td>
<td>0.0</td>
</tr>
<tr>
<td>RF</td>
<td>8.2±2.1 (8.0)</td>
<td>8.2±2.0 (7.8)</td>
<td>0.0</td>
<td>7.4±1.9 (7.5)</td>
<td>7.5±1.9 (7.6)</td>
<td>1.3</td>
</tr>
<tr>
<td>PT</td>
<td>1.8±0.3 (1.7)</td>
<td>1.6±0.4 (1.7)</td>
<td>11.1</td>
<td>1.5±0.3 (1.5)</td>
<td>1.5±0.2 (1.5)</td>
<td>0.0</td>
</tr>
</tbody>
</table>

Values are means ± SD with medians in parentheses; n, no. of subjects. aCSA, anatomic cross-sectional area of muscles; TQ, total quadriceps area; VM, vastus medialis; VL, vastus lateralis; VL/VM, aCSA ratio of the vastus lateralis-to-vastus medialis; VI, vastus intermedius; PT, patellar tendon; %Diff, percent differences of the aCSA. *P < 0.05. †P < 0.01. ‡P < 0.001.

Discussion

The findings of the present study revealed a 9.1–13.4% lower aCSA (muscle atrophy) of the entire quadriceps muscle, as well as the VM, VL, and VI in individuals with HQ angle compared with LQ angle. The muscle atrophy that was found in the HQ angle may be attributed to a neural mechanism by which length-tension changes in the capsuloligamentous and musculotendinous structures result in proprioceptive deficits, alterations in theafferent information to the central nervous system (CNS) and modifications in the efferent information. The role of capsuloligamentous mechanoreceptors is very important because they provide afferent information to the CNS via specific reflex arcs (10, 36) and thus contribute to the dynamic stability of a joint by facilitating and adjusting muscle kinetic response. In individuals with HQ angle, contraction of the quadriceps will tend to produce a “valgus vector” because of the patella’s tendency to move laterally (18). This lateralization is resisted by the VMO, the passive medial soft tissue restraints, and the prominence of the lateral facet of the trochlea (8, 9, 14). In the face of chronic malalignment, the capsuloligamentous structures of the medial knee surface may become stretched (23) or loose due to accumulated creep (40), whereas those of the lateral surface become shortened and tight (23). These alterations have a direct effect on the receptors’ threshold, resulting in a degradation of kinesthesia and proprioceptive perception (40). Abnormal afferent information may in turn decrease γ-motoneuron excitability, inhibiting muscle kinetic response (19, 20) and leading to muscle atrophy. Williams et al. (43) have shown, in a previous study, that proprioceptive deficits may be one of the factors that can cause impairment of voluntary contraction, muscle weakness, and nonuniform atrophy among the parts of the quadriceps in individuals who do not compensate well after anterior cruciate ligament injury (noncopers). These authors showed signifi-
cantly lower volume and peak aCSA of the quadriceps with the VL and VI disproportionately atrophied compared with the uninjured side.

Alterations in the musculotendinous receptors threshold and differentiation in afferent and efferent neural signals may also contribute to the VM and VL muscle atrophy, in the individuals with HQ angle. These alterations may attribute to the different tensile loads applied along the myofibrils and the musculotendinous junction as joint angulation forces VL to function from a shortened position, whereas VM functions from a stretched position. Animal studies have shown that muscles immobilized in a shortened position developed a higher degree of atrophy than those immobilized in a stretched position (6). The muscle atrophy that is found is also accompanied by length-dependent changes in the cell shape. When a muscle is immobilized in a lengthened position, the number of the sarcomeres is increased and the length is reduced; the opposite occurs when a muscle is immobilized in a shortened position (44, 46). These findings also supported our observations regarding greater atrophy in the VL compared with VM.

Muscle atrophy of the entire quadriceps as well as the VL compared with VM may also be explained by the alterations in the efferent neural information originated from higher neural centers. Such changes have been reported in situations such as joint immobilization (29, 48) and ligamentous injury (24), which have caused sensory deprivation and led to brain reorganization. Prolonged abnormal joint loading due to side-to-side differences in the length of the capsuloligamentous structures, as in individuals with HQ angle, may induce similar changes in the CNS, because the human body has the ability to ensure optimal joint function whenever this joint is dysfunctional (39). In our study, quadriceps muscle activation may be lowered, and, therefore, atrophied through the years, to protect the joint structures from wear.

Another possible explanation for the differences in the cross sections of the quadriceps between the two groups is the different pennation angle of the quadriceps muscle fibers. The CSA of the quadriceps in the present study was measured based on axial images (aCSA) therefore the pennation angles of the individual parts of the quadriceps were not taken into account. The physiological CSA, on the other hand, is a method which has been used to calculate indirectly the perpendicular CSA of a muscle based on the muscle volume and length. This method has been used mainly in the assessment of the of muscle’s capacity to generate force assuming that the muscle fiber pennation angle and the fiber-to-length ratios remain consistent (47). These conditions, however, do not apply to muscles with a complex architecture, such as the RF and VI, because their muscle fibers do not contract at the same rate (5). Therefore, the aCSA was favored over the physiological, taking into consideration that both the anatomical and physiological CSAs are highly correlated to the maximum voluntary strength produced by a muscle (3).

Based on our findings, the aCSA of the RF remained unaffected, compared with the other parts of the quadriceps in the HQ angle group. This may be explained by the fact that the biarticular RF is stronger as a hip flexor, rather than as a knee extensor, because the produced active force is determined by the fiber length of its proximal part (13). On the other hand, the VM and VL function to control the tracking of the patella in the trochlea groove (28) and may be more susceptible to muscle atrophy, as has been shown in previous studies (43).

The method used for the MRI of the quadriceps was one of the limitations in the present study. The aCSA of the quadriceps in our study was measured from images obtained in the middle of the distal third of the femur on both HQ and LQ angle groups. Other researchers (42), who, measuring the peak aCSA using a trapezoidal integration algorithm in young athletes, have reported a greater CSA of the quadriceps, compared with our findings. It is possible that performing the MRI on a different location of the quadriceps may result in a different aCSA between an HQ and an LQ angle group.

Our findings are also limited in the present study due to subjects’ positioning for Q angle and MRI measurements. Q angle in our study was measured in the supine position to be consistent with the supine position that each subject assumed during the MRI measurements. It should also be noted that, despite the fact that the lower extremity was fixed in the supine position, muscle contraction during a dynamic MRI could produce slight outward or inward movements, affecting our results. Q angle measurements performed in the standing position could reveal different values, because they can be influenced by the alignment of the adjacent joints (e.g., hip, ankle). Although such measurements would be more appropriate from a functional point of view, they would be inconsistent with the MRI, because such measurements are not possible in the standing position.

In conclusion, the results of this study revealed a lower aCSA in individuals with HQ angle. It is hypothesized that these changes may be attributed to neural alterations in the peripheral and CNS, which leads to muscle atrophy and possibly lowers muscle activation, to protect the joint from excessive loading and wear.

Further research is needed to determine the possible effect of knee joint malalignment on the strength, the activation pattern, the electromechanical efficiency, or fiber microstructure of the quadriceps muscle.

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GRANTS

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REFERENCES

804 QUADRICEPS CSA DIFFERENCES IN MEN WITH DIFFERENT Q ANGLE


