Children with spastic cerebral palsy often walk with a troublesome, in-toed gait. Excessive internal rotation of the hip, usually accompanied by excessive femoral anteverision, causes tripping (Hoffer 1986, Taedhjian 1990) and may lead to lateral patellar subluxation (Samilson 1981) or external tibial torsion (Root 1987, DeLucia 1991). Hence surgery, such as tendon lengthening or derotational osteotomy, aimed at decreasing excessive internal rotation is often warranted.

Unfortunately the outcome of surgery to correct an in-toed gait is unpredictable and sometimes unsatisfactory (Bleck 1987). This problem exists, in part, because identifying the underlying cause of a rotational deformity is difficult; hence the most appropriate treatment is often unclear. For example, spastic muscles are frequently cited as a source of excessive internal rotation based on evidence from electromyographic (EMG) recordings. However, EMG recordings indicate only that a muscle is active — not why it is active. Consequently, there is no consensus among investigators as to which muscles contribute to abnormal internal rotation during walking or which muscles should be adjusted during surgery; medial hamstrings (Sutherland et al. 1969, Chong et al. 1978, Ray and Ehrlich 1979), adductors (Majestro and Frost 1971, Tyllkowskii et al. 1982), iliopsoas (Samilson 1981, Taedhjian 1990), and the anterior fibers of gluteus medius (Steel 1980, Bleck 1990) have all been proposed.

Other investigators have speculated that the source of excessive internal rotation in many patients with cerebral palsy is not spasticity of the internal rotators, but apparent weakness of the abductors, caused by bone deformity (Merchant 1965, Bleck 1987). According to this theory, excessive femoral anteverision decreases the moment arms of the abductors, and internal rotation of the hip is a compensatory mechanism that restores these moment arms and preserves abduction capacity. This explanation seems plausible; however, few investigators have actually quantified how femoral geometry affects the moment arms of the abductors, and thus the capacity of the muscles to generate moments about the hip (Spoor et al. 1989, Delp et al. 1994). No study has determined how abduction moment arms vary with anteverision angle and hip internal rotation angle for the ranges of deformities commonly found in children with cerebral palsy. Thus, whether this ‘compensatory mechanism’ theory is correct, or even feasible, is not known.

The ‘compensatory mechanism’ theory has been difficult to verify because conventional methods for measuring abduction moment arms are not sufficiently accurate. Planar radiographs are routinely used to estimate abduction moment arms after hip replacement (Weisman et al. 1978, Borja et al. 1985). However, large errors can be introduced when moment arms are measured from radiographs, because the planar projections do not accurately characterize the three-dimensional geometry of the femur. This problem is especially troublesome when bone deformities are present (Miller et al. 1993). Thus standard clinical measurements are not adequate for quantifying how abduction moment arms are altered by femoral deformities, hip rotation, or surgical procedures aimed at restoring moment arms.

In this study, a three-dimensional computer model of the hip and the surrounding musculature was developed to determine how variations in anteverision angle, neck-shaft angle, and hip internal rotation angle affect the abduction moment arm of the gluteus medius, one of the primary abductors of the
hip. We hypothesized that excessive femoral anteversion substantially decreases the abduction moment arm of the gluteus medius and that this moment arm is restored with internal rotation. Our goal in implementing the computer model was to test this hypothesis and to evaluate the feasibility of the 'compensatory mechanism' theory for internal rotation gait.

Method

We used a graphics-based model of the lower extremity (Delp et al. 1990b) to examine how femoral deformities and excessive internal rotation affect the abduction capacity of the gluteus medius (Fig. 1). This model characterizes the geometry of the femur, the path of the gluteus medius, and the kinematics of the hip for a nominal adult subject (height approximately 1.8 m) so that the abduction moment arm of the muscle can be estimated for a range of bone deformities and limb positions. The surfaces of the pelvis and femur are described by a mesh of polygons. The gluteus medius is represented by three straight-line segments; attachment coordinates for these segments were defined by Delp et al. (1990b) based on the muscle's anatomical relations to the three-dimensional representations of the bones. The hip model, which defines relative motion between the pelvis and femur, is assumed to be a ball-and-socket joint.

We studied the relation between anteversion angle, neck-shaft angle, and abduction moment arm by deforming the femur of the lower-limb model. Our method of specifying femoral geometry is similar to the classic method described by Murphy et al. (1987). On the femur we located the positions of five points: H, O, P, L, and M (Fig. 2). These points define the neck axis (NA), the condylar axis (CA), and the femoral-shaft axis (FA). Anteversion of the femur (α) is defined as the angle between the plane of the neck axis and the plane of the condylar axis. The neck-shaft angle (β) is the angle formed by the neck axis and the femoral-shaft axis. Undeformed, the model has an anteversion angle of 20° and a neck-shaft angle of 130°, which are within the normal range (Clark et al. 1987, Upadhyay et al. 1987).

We altered the anteversion and neck-shaft angles of the model by rotating the bone vertices that make up the surfaces of the femoral neck and head. The angle of anteversion was varied between 0°, which is at the low end of the normal range (Anda et al. 1988, Lausten et al. 1989), and 60°, which is at the high end of the abnormal range in children with cerebral palsy (Bleck 1987, Laplaza et al. 1993). The neck-shaft angle was varied between 150°, which is considered excessive (Bleck 1987), and 110°, which is a common postoperative goal after varus derotational osteotomy (Root and Siegel 1980, Tylkowski et al. 1980, Hoffer et al. 1985). Muscle insertions on the greater trochanter were displaced along with the bone vertices; hence, the lengths and moment arms of the gluteus medius segments were changed by these alterations. The position of the center of the hip with respect to the pelvis was not changed.

We determined the degree to which internal rotation of the hip alters the abduction moment arm of the gluteus medius by rotating the (deformed) femur of the lower limb model about an axis of rotation (RA) through the center of the hip (see Fig. 2). The angle of rotation (γ) was varied between 0° and 30° of internal rotation. Hip flexion and abduction angles were kept constant in this study at angles corresponding to the anatomical position of the limb.

We calculated the abduction moment arm of each gluteus medius segment i (ma_i) as the partial derivative of the segment's origin-to-insertion length (L) with respect to the abduction angle (θ) of the hip (An et al. 1984): ma_i = dL/θ.

Moment arms of the three segments were averaged to obtain an estimate (ma_bar) of the moment arm of the muscle. To facilitate interpretation of the data, moment arms were expressed as percentage of change from the moment arm of the model in its undeformed and unrotated state.

![Figure 1: Graphics-based model of lower extremity with (A) 20° and (B) 50° of femoral anteversion. Both femurs have a neck-shaft angle of 150° and a hip rotation angle of 0°. Paths of the three gluteus medius segments are highlighted.](image-url)
Results
Excessive anteversion of the femur decreases the abduction moment arm of the gluteus medius (Fig. 3). The decrease of the moment arm is minimal for small increases in the anteversion angle but is substantial for large increases in the angle. Our model indicates that a 10° increase in anteversion angle (i.e., an anteversion angle of 30° and a neck-shaft angle of 130°) decreases the abduction moment arm of the gluteus medius by 10%. A 40° increase in anteversion angle (i.e., an anteversion angle of 60° and a neck-shaft angle of 130°) decreases the moment arm by 54%.

The abduction moment arm of the gluteus medius is also affected by the neck-shaft angle (see Fig. 3). A valgus deformity of 20° (i.e., an anteversion angle of 20° and a neck-shaft angle of 150°) decreases the moment arm of the model by 26%. A combination of excessive femoral anteversion and a valgus femoral neck (e.g., an anteversion angle of 60° and a neck-shaft angle of 150°) decreases the moment arm of the model by as much as 69%. ‘Correction’ of the deformity, perhaps simulating the outcome of a varus derotational osteotomy (e.g., an anteversion angle of 10° and a neck-shaft angle of 110°), increases the abduction moment arm of the model above normal by as much as 30%. However, varization concurrently decreases the origin-to-insertion length of the muscle to about 5% below normal, which may substantially lessen force-generating capacity.

Internal rotation of the hip, in the presence of excessive anteversion, restores the abduction moment arm of the gluteus medius (Fig. 4). An anteversion deformity of 30° (i.e., an anteversion angle of 50°, a neck-shaft angle of 130°, and a rotation angle of 0°) decreases the abduction moment arm of the model by 37%. With 10° of internal rotation, however, the abduction moment arm is decreased only 22%. With 30° of internal rotation, the abduction moment arm is increased by less than 1%. Internal rotation of the hip does not counteract the adverse effects of a valgus femoral neck (not shown).

Discussion
The results of our computer simulations confirm that (1) anteversion and valgus deformities of the femur decrease the abduction moment arm of the gluteus medius, and (2) internal rotation of the hip in the presence of an anteversion deformity restores the abduction moment arm of the gluteus medius and improves abduction capacity. These results are consistent with the ‘compensatory mechanism’ theory for internal rotation gait. Delp et al. (1990a) estimated that a 45% reduction in abduction capacity is enough to cause a limp; that estimate agrees with the clinical observation that near full strength of the hip abductors is required for normal walking (Perry 1985).

In our study, 30 to 40° of excessive femoral anteversion caused a 40 to 50% decrease in the abduction moment arm of the gluteus medius—a functionally significant reduction. In children with spastic cerebral palsy, deformities this size are not unusual (Bleck 1987, Ruwe et al. 1992, Laplaza et al. 1993). Our analysis also indicates that the abduction moment arm of the gluteus medius is greatly improved with 20 to 30° of internal rotation; this amount of in-toeing occurs frequently.

Before discussing the clinical implications of these results, it is important to consider some of the limitations of the study. First, the musculoskeletal model used in this study represents an adult subject, whereas surgery to treat excessive internal rotation is commonly performed on children. Unfortunately there are insufficient data in the literature to determine how muscle moment arms differ between subjects of different sizes and ages; thus, the model’s ability to predict changes in moment arm for a range of subjects is not known. A related limitation stems from the fact that the changes in moment arm reported here reflect our definition of normal femoral geometry. Anteversion angles vary with age (Fabry et al. 1973), and anteversion measurements depend upon the specific measurement techniques used (Murphy et al. 1987, Hoidal et al. 1988, Miller et al. 1993). Thus the decrease in abduction moment arm caused by a particular degree of anteversion in a particular patient may not correspond exactly to the percentages reported here. We believe, however, that the trends obtained from the model are representative and accurate.

Second, we considered only the gluteus medius in this

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Figure 2: Description of femoral geometry. H = center of femoral head; O = center of base of femoral neck (determined by locating centroid of femoral diaphysis on a cross-section through base of femoral neck); P = attachment of posterior cruciate ligament; L, M = posterior aspects of lateral and medial condyles, respectively; NA = neck axis (defined by points O and H); FA = femoral-shaft axis (defined by points P and O); CA = condylar axis (defined as the vector parallel to LM that passes through point P); RA = rotation axis (defined by point H and midpoint of a line joining medial and lateral condyles); AP = anteversion plane, formed by axes NA and FA; CP = condylar plane, formed by axes CA and FA. Anteversion angle (a), neck-shaft angle (b), and internal rotation angle (c) are determined based on these definitions. Figure adapted from Murphy et al. (1987).
study, although other muscles contribute to the abduction moment at the hip. We focused on the gluteus medius because it is the primary abductor of the hip (Delp et al. 1990a), and because its line of action can be adequately described by straight-line segments for a range of femoral deformities and limb positions. The abduction moment arm of the gluteus minimus is expected to vary with femoral geometry in a similar manner due to its similar insertion on the greater trochanter. Together, these two muscles provide a large proportion of total abduction capacity (Delp et al. 1990a).

Third, in this study we assessed only muscle moment arms; we did not estimate muscle forces. Even if internal rotation of the hip improves the abduction moment arm of the gluteus medius, it may not eliminate abduction weakness if the muscles are activated inadequately or if they cannot generate sufficient force. Internal rotation gait is sometimes associated with a gluteus medius limp (Tachdjian 1990). In these patients, insufficient abduction force — in addition to an insufficient moment arm — may contribute to abductor weakness. Weakness of the abductors has also been observed after varization osteotomy (Tachdjian 1990), although our simulations show that decreasing the neck–shaft angle of the femur increases the abduction moment arm of the gluteus medius (see Fig. 3). Certainly changes in muscle lengths and numerous other factors may influence the abduction capacity of patients after surgery; hence the results of our study must be interpreted with care. Our aim, in analyzing the computer model, was to isolate the changes in abduction moment arm that result from alterations in femoral geometry and hip rotation.

Finally, our results only suggest that the 'compensatory mechanism' theory is feasible; they do not prove that it is true. Certainly other causes of an internal rotation gait are possible.

Anatomical studies based on cadaver specimens with normal femoral geometry have shown that the moment arms of many internal rotators increase with hip flexion while the moment arms of many external rotators decrease with hip flexion (Dostal et al. 1986). Since flexion deformities frequently accompany an internal rotation gait (Bleck 1987, Gage 1991), excessive hip flexion may disrupt the balance between the internal and external rotators and contribute to abnormal internal rotation. Much more work is needed to understand how the moment arms of the hip muscles change with flexion, adduction, and internal rotation—and how these relations are altered by bone deformities. This study, by quantifying the abduction moment arm of the gluteus medius over a range of anteversion angles, neck–shaft angles, and internal rotation angles, is an important first step.

The results of this study emphasize the need to consider musculoskeletal geometry when considering the causes of movement abnormalities. Although cerebral palsy is caused by damage to the central nervous system, mechanical changes at the periphery, such as bone deformities or muscle contractions, may play an important role. Muscle spasticity is often cited as the source of an in-toed gait on the basis of EMG recordings. These recordings, however, indicate only that a muscle is active. Premature or prolonged muscle activity could reflect CNS pathology, as is often assumed, or it could instead indicate that the muscle is activated to compensate for some other abnormality. If the abductors are in effect weakened by an anteversion deformity, for example, the internal rotators may be activated as a compensatory mechanism to achieve the abduction moment arm needed for walking.

Figure 3: Change in abduction moment arm vs anteversion angle for neck–shaft angles of 110° to 150°. Moment arms are expressed as percentage of change from moment arm of model in its undeformed state (20° anteversion, 130° neck–shaft angle). Hip rotation angle was kept constant at 0°; hip flexion and adduction angles were kept constant at angles corresponding to anatomical position of limb.

Figure 4: Change in abduction moment arm vs anteversion angle for hip internal rotation angles of 0° to 30°. Moment arms are expressed as percentage of change from moment arm of model in its undeformed state (20° anteversion, 0° internal rotation). Neck–shaft angle was kept constant at 130°; hip flexion and adduction angles were kept constant at angles corresponding to anatomical position of limb.
Acknowledgements
This work was supported by a National Science Foundation National Young Investigator Award to S Delp and a National Science Foundation Graduate Research Fellowship to A Arnold.

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