11-1-2000

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Original Citation

Bellchamber, T. L., and van, d. B., 2000, "Contributions of Proximal and Distal Moments to Axial Tibial Rotation during Walking and Running," Journal of Biomechanics, 33(11) pp. 1397-1403.

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Contributions of proximal and distal moments to axial tibial rotation during walking and running

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1. Introduction

It has been suggested that certain knee injuries are caused by excessive internal tibial rotation. (Clement et al., 1981; James and Jones, 1990; Tiberio, 1987) or a delayed external tibial rotation (McClay and Manal, 1997). As external rotation is linked to knee extension, excessive internal rotation during the stance phase of walking or running may delay the natural external rotation as the knee begins to extend. This has the potential to increase torsional joint stresses at the knee or within the tibia and in turn cause knee injury. For further comprehension of this potential injury mechanism, it is essential to understand where the cause of tibial rotation is located.

Tibial internal rotation is coupled to eversion of the foot through the ankle joint complex. The anatomy of

the ankle joint complex can be modelled as two separate hinge joints, at the talocrural joint and one at the subtalar joint. The subtalar joint between talus and calcaneus is approximately 45° inclined from the horizontal. As the calcaneus everts, this mechanism should transfer into a similar amount of tibial internal rotation (McClay and Manal, 1997; van den Bogert et al., 1994). Experimental work has verified this coupling between the internal rotation of the tibia and the eversion of the foot. Nigg et al. (1993) found that the coupling of the eversion of the foot to the internal rotation of the tibia had an extremely high correlation of $r^2 = 0.991$ for running. If tibial rotation is linked to knee injuries and is coupled to eversion of the foot this may imply that controlling eversion with foot orthoses could reduce tibial rotation and thus knee pain. The assumption in this is that foot eversion causes internal tibial rotation. However, if tibial rotation originates proximally to the tibia, and the foot merely follows the tibia, controlling eversion may increase the resistance against tibial rotation and cause more stress on the tibia and knee. One could use an analogy between the tibia

and the driveshaft in an automobile. If the brakes are used when the engine is powering the vehicle, stress in the driveshaft will increase. If the brakes are used when the engine is slowing down the vehicle, for instance when going down a steep downhill slope, stress in the driveshaft will decrease.

Clinically, orthotics are a common treatment for patients with knee pain due to running injuries. Orthoses used in conjunction with conventional therapies such as rest, medication, and physiotherapy are more effective than conventional treatments alone (Kilmartin and Wallace, 1994). Yet, there is evidence in the literature to suggest that eversion may not always cause the internal rotation of the tibia. Studies of orthotic effectiveness on controlling the lower limb have found varying results on tibial rotations and negligible effects on knee kinematics (Kilmartin and Wallace, 1994). The effect of orthoses on knee range of motion has been found to be related to the type of activity being performed (Eng and Pierrynowski, 1994) and the effect of orthoses on tibial rotation shows large intersubject variability (Cornwall and McPoil, 1995). Lafortune et al. (1994) found that changes in tibial internal rotation due to orthotic intervention were not matched with changes in knee internal rotation thus implying compensation at the hip joint.

These conflicting clinical and biomechanical conclusions about orthotic effectiveness and the widespread use of orthoses suggest further questions. Research is needed to examine whether eversion causes tibial rotation in walking and running or if tibial rotation is caused proximally for some patients during all or some parts of the support phase of the locomotion and foot eversion is a result of this. In the latter case, orthotic therapies may not be effective. It is the purpose of this study to determine the cause and effect relationship between tibial internal/external rotation and forces acting on the foot during walking and heel-toe running in the normal population.

2. Methods

Data were collected from twenty adult subjects, ten male and ten female, who had no current lower extremity injuries (mean age: 26.4 ± 6.7 years, mean mass: 77.7 ± 9.7 kg for male subjects, 61.7 ± 9.6 kg for female subjects). Each subject provided written informed consent to participate in the study. Male subjects used the Adidas Response shoe and female subjects used the Adidas Argonaut XS shoe. Each subject performed ten running and ten walking trials. The following anthropometric measurements were taken using sliding callipers: (1) distance from the lateral epicondyle to the medial epicondyle, (2) height of the midpoint of the lateral malleolus to the ground (3) height of the midpoint of the medial

malleolus to the ground, and (4) the distance between the medial and lateral malleoli.

Ground reaction forces were sampled at 2400 Hz from a Kistler force plate (model 9281B) flush with the surface of an indoor track. Synchronized three-dimensional (3-D) kinematic data were collected using four cameras at 240 Hz (Motion Analysis Corporation, Santa Rosa, California) positioned around the force plate such that each kinematic marker would be visible in at least three cameras in each frame. Calibration was done with a calibration cube with an approximate volume of 1 m^3 defining the laboratory coordinate system (LCS). The position of the transducers in the force plate with respect to the LCS was obtained by collecting the 3-D coordinates of spherical markers set on top of each of the four transducers of the force plate.

Spherical reflective markers were placed on the right shank and shoe of the subjects. Shank markers (12 mm diameter) were located at five sites; 2 cm below the head of the fibula, on the tibial tuberosity, on the anterior tibia halfway down the shank, 2 cm proximal to the lateral malleolus, and anterior on the lower shank. For the shoe segment, three 20 mm markers were placed on the posterior lateral aspect of the calcaneus, lateral metatarsal head, and superiorly on the navicular. Two more 20 mm markers were placed on the lateral epicondyle of the knee and lateral malleolus of the ankle for the neutral trial only and removed for the dynamic trials.

A neutral trial was collected for each subject standing on the force plate for one second. The right foot was placed in the centre of the force plate with the long axis of the foot (connecting the centre of the calcaneus and the second toe) aligned with the forward (x) axis of the LCS. Data collection of the dynamic trials was done using photocells to trigger the data collection and record the time taken for the subjects to pass between them. Sufficient practice trials were given to the subjects to obtain consistent speed and foot placement. Acceptable running speeds were in the range $4.0 \pm 0.4 \text{ m/s}$ and acceptable walking speeds were in the range $2.0 \pm 0.2 \text{ m/s}$. Data collection continued until 12 acceptable trials had been collected. After data collection, the first ten acceptable trials of each movement along with a neutral trial and a force plate calibration trial were tracked in the EVa software (Motion Analysis Corporation, Santa Rosa, California) for each subject.

3. Data analysis

The 3-D coordinates of the neutral trial were averaged in each subject to give a set of reference coordinates for all markers. Using these and the anthropometric measurements, the knee and ankle joint centres were defined as described by Vaughan et al. (1992). The tibial axis was defined as the vector from the ankle joint centre

to the knee joint centre in the neutral frame. With this axis the segment coordinate system (SCS) of the tibia was defined such that the y -axis was the tibial axis, and the yz plane was defined by the tibial axis and the lateral malleolus. For the foot coordinate system, the axes were assumed to be aligned with the LCS during the neutral trial such that the x -axis pointed anteriorly and the y -axis was the vertical axis. These coordinate systems were used to compute the static coordinates of the markers with respect to the SCS for later use in the analysis of the dynamic trials.

The 3-D kinematic data from the dynamic trials were filtered using a quintic smoothing spline (Woltring, 1986) with cut-off frequencies of 10 and 15 Hz for walking and running, respectively. For each frame the rotation matrix (${}^{\text{lab}}\mathbf{R}_{\text{SCS}}$) and position vector (${}^{\text{lab}}\mathbf{p}_{\text{SCS}}$) for each segment reference system with respect to the laboratory reference system was obtained using a least squares algorithm (Soderkvist and Wedin, 1993). The angular velocity vector for the shank segment relative to the LCS was found using the first derivative of ${}^{\text{lab}}\mathbf{R}_{\text{SCS}}$ (Berme et al., 1990). The angular velocity component along the tibial axis corresponding to internal and external rotation is the y component of this angular velocity vector.

In order to compare the measured tibial internal rotation with previous studies the internal rotation of the tibia and the eversion of the foot were also calculated from the rotation matrices as described by Nigg et al. (1993).

Force plate output was arranged into X , Y , and Z components for each of the four transducers in the force plate coordinate system (FCS). Heel strike and toe off were determined to be the point at which the vertical ground reaction force from the plate first exceeded and last exceeded 20 N respectively. Moments of force in the FCS were determined using the known distances to the transducers from the force plate origin.

Calculation of the moment of force at the ankle joint centre involved two coordinate transformations. Position and orientation of the force plate with respect to the LCS was determined from the 3-D coordinates of each of the force plate transducers (Soderkvist and Wedin, 1993). The forces and moments in the FCS were then transformed to the LCS by rotating the force and moment vectors and adding the additional moment from the cross product of the point of application of the force vector in the LCS. The final coordinate transform from the LCS to the tibia coordinate system was done similarly using the individual rotation matrix and position vector for the tibia in each frame, resulting in a 3-D intersegmental force and moment vector acting on the tibia at the ankle joint centre. The mass of the foot was neglected. The angular velocity and moment data were time-normalized to 100 data points for each trial, from heel strike to toe-off and the power flow associated with axial tibial rotation was calculated at each data

point:

$$\text{Power} = \omega_y \cdot M_y.$$

Positive power represents power flow from foot to tibia and therefore a distal cause of axial tibial rotation. It should be noted that this is only one term in the total power flow which is the sum of six terms, one for each degree of freedom. This axial power flow term indicates the power source of axial rotation, using the analogy with a driveshaft in an automobile transmission. It should also be noted that the axial moments at proximal and distal end of the tibia are equal and opposite, since the axial moment of inertia is negligible.

A further calculation was made in order to compare the pattern of the moment of force with one in the literature (Holden and Cavanagh, 1991). The moment of force at the tibia was expressed in the lab coordinate system such that the vertical moment of force about the ankle joint centre could be obtained.

4. Results

Fig. 1 shows the results for all running trials in a typical subject, indicating the consistency between trials which justified representing each subject's data by an ensemble average of all trials.

Axial tibial moments during walking showed similar patterns for all subjects (Fig. 2a). Amplitudes were different, indicating possible effects of body weight and movement style. Larger interindividual differences were found for running (Fig. 2b).

The angular velocity pattern during walking (Fig. 3) showed a large internal rotation velocity peak near the beginning of the stance phase reaching speeds of 2 rad/s and a large external rotation velocity peak near the end of stance. Smaller oscillations were seen around midstance and some subjects having large external rotation velocities at approximately 30% of stance. Women appeared to have larger oscillations throughout midstance, therefore further results will be separated by gender. Power curves for axial tibial rotation mainly gained their intersubject variability from the angular velocity variability as all subjects had similar moment curves. Power flow patterns tended to have numerous oscillations between -20 and 7 Watts for the first 70% of stance phase, again women tending to have larger oscillations than men. All subjects showed large negative power flow in the last 30% of stance phase (Fig. 4) indicating a proximal power source for the external rotation of the tibia at that time.

Angular velocity curves for running (Fig. 5) were similar for male and female subjects, again with slightly larger midstance oscillations for females. After heel strike, the tibia increased its velocity corresponding with tibial internal rotation in the first part of stance. A maximum

Fig. 1. Moment, velocity, and power curves during running (10 trials) for a typical subject.

velocity of about 3 rad/s was reached at 20% of stance, followed by a negative peak at approximately 35% of the stance phase when the tibia was externally rotating at about 2.5 rad/s. The tibia then had a much lower velocity, perhaps briefly internally rotating in females, followed by accelerated external rotation during takeoff. Three male subjects and two female subjects had patterns that were clearly abnormal.

Power flow for normal subjects during running (Fig. 6) remained small for the first 10–20% of stance. It then became mainly negative, indicating a proximal source of power for tibia rotation. Positive peaks occurred and larger negative peaks occurred in the abnormal subjects. Female subjects again tended to have larger oscillations and also a brief positive power flow from foot to tibia between 40 and 60% of stance.

5. Discussion

The purpose of this study was to determine the contributions of proximal and distal segments to axial tibial rotation. Angular velocity and moment of force with

Fig. 2. Average axial tibial moment curves for all twenty subjects: (a) walking average moment curves, (b) running average moment curves.

Fig. 3. Average axial tibial angular velocity curves, walking trials. Positive velocities are internal rotations.

Fig. 4. Average power curves for each subject during walking. Positive power indicates distal control of the tibial rotation.

Fig. 6. Average power curves for each subject during running. Positive power indicates distal control of the tibial rotation.

Fig. 5. Average axial tibial angular velocity curves for each subject during running. Positive velocities are internal rotations.

respect to the tibial axis were calculated in order to quantify power flow from foot to shank associated with axial rotation. Proximal control was indicated by angular velocities in the opposite direction of the moment of force, distal control by angular velocities in the direction of the moment of force.

Rapid oscillations in the angular velocity curves for walking and running suggested errors due to marker movement relative to the bone. Pilot studies were done with varying sizes and locations of reflective markers but no clear differences were found in the velocity curves. Similar oscillation patterns have been found in other studies (Stergiou, 1996). These oscillations may be a combination of true tibial velocity oscillations and artifacts from muscle and skin movement. Reinschmidt et al. (1997) reported kinematics of femur and tibia using both skin and bone markers. The largest axial angular velocity difference between skin and bone markers at any time in any subject was 2° per 10% of stance time, or 1.4 rad/s. A similar value was obtained for walking (Reinschmidt, 1996). Therefore, whenever the magnitude of angular velocity in our results is greater than 1.4 rad/s, we know that this represents true tibial rotation. Values less than this error bound may be soft tissue artifacts, and consequently there will also be an uncertainty of up to 1.4 rad/s, divided by the slope of the angular velocity curve, in the time of zero crossing. The errors, however, are likely to be smaller on average than this worst case

estimate. The larger oscillations in female subjects could have been caused by the shoes.

Moments of force and rotations for running were consistent with those reported in the literature. Average vertical moments of force at the ankle joint centre (Fig. 7) were similar to results of Holden and Cavanagh (1991). While no data in the literature could be found to compare with the velocity pattern of tibia rotation, the amount of internal rotation measured in running was in agreement with previous literature. The eversion of the foot and internal rotation of the tibia showed a nearly linear correlation (Fig. 8) as found by Nigg et al. (1993). Patterns of rotation also agreed with previous studies (Nigg et al., 1993) with a maximum average change in internal rotation of $15^\circ \pm 4^\circ$ (Fig. 9). The average maximum change in eversion was $18 \pm 5^\circ$ which was smaller than the value of $28 \pm 7.2^\circ$ measured by Nigg et al. (1993). However, it should be noted that their values were approximately 10% higher than results of other studies.

Fig. 7. Average vertical moments of force about the ankle joint centre for each subject during running.

Fig. 8. Tibial rotation versus calcaneal inversion-eversion for running, average of all trials in all subjects. Note the linear relationship between the variables in the latter part of the curve.

Fig. 9. Average rotations during running: (a) Calcaneal inversion-eversion, positive angles are everted, (b) tibial internal-external rotations, positive angles are externally rotated.

Axial moment curves (Fig. 2) were positive in most subjects during running and in the latter 70% of walking stance. This shows that the ground reaction force, in combination with the ankle mechanism, caused mostly an internally rotating moment at the tibia during all or 70% of stance phase respectively. Moment curves had a slightly higher magnitude in running than in walking, and much more variability between subjects. Running curves also peaked earlier in stance phase at approximately 50% of stance while walking curves peaked later at 80% of stance. Axial moment during the first 25–35% of stance phase showed an externally rotating moment of about 3 Nm.

For walking, the angular velocities (fig. 3) were not always larger than the error bound of 1.4 rad/s. Since the angular velocities between 35 and 80% of stance were relatively small, conclusions about velocity or power flow during this time period may be influenced by soft tissue movement. Power flow until 50% of stance was small due to the low moments and oscillated from positive to negative (Fig. 4). The last 20% of stance phase contains a large peak of negative power flow indicating that there is a clear proximal source of tibial rotation. At no time during walking stance was there a positive (distal to proximal) power flow large enough to indicate that the foot was driving tibial rotation. This suggests that the use of foot orthoses during walking may increase stress at the knee during the internal rotation phase by creating a moment which opposes the motion induced by the

proximal segments. Clinical success using orthoses to treat knee pain in walking may instead be due to a redistribution of forces at the knee, rather than through an effect on tibial rotation.

The velocity curves for running (Fig. 5) all tended to have peak internal and external velocities greater than 1.4 rad/s indicating that the curve did represent the velocity of the tibia and expected internal and external rotation patterns were seen. There was minimal power flow until 15% of stance (Fig. 6) indicating that the moment from the ground reaction force did not contribute to or oppose tibial rotation. There were brief periods of positive power flow up the tibia in most subjects between 20 and 60% of running stance, indicating that the movement of the foot may have contributed to the tibial rotation. Negative power flow in the remaining part of stance indicated that the tibial rotation was usually being driven proximally, especially near the end of stance. The most remarkable finding is that five subjects had very different power flow patterns during running, clearly showing a potential for different responses to orthoses. We suggest that individuals with large negative power flow early during stance will respond poorly to orthotics, with the possibility of further aggravating knee problems.

Our conclusions are:

- (1) During walking, axial tibial rotation has a proximal power source and foot orthoses may increase stresses in the knee that are associated with tibial rotation torques.
- (2) During running, there are brief periods where axial tibial rotation is driven by the foot, indicating a potential beneficial effect of foot orthoses.
- (3) 25% of the subjects had abnormal power flow patterns during running, indicating the potential for patient-specific responses to orthotics.

This research was limited to a population of subjects who did not have knee pain. Further studies looking for differences between healthy individuals and subjects with knee pain are needed to look for systematic patterns that may help diagnosis and treatment. We speculate that patients with significant positive axial power flow from foot to tibia could benefit from foot orthoses and we suggest that the protocols described in this study may be used to predict and evaluate effectiveness of orthotic treatment for knee pain.

Acknowledgements

This research was financially supported by the Whitaker Foundation. We thank Adidas America for donating the running shoes used in this study.

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