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BASIC RESEARCH

Extrinsic Muscle Forces Affect Ankle Loading Before and After Total Ankle Arthroplasty

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Abstract

Background Joint loading conditions have an effect on the development and management of ankle osteoarthritis and on aseptic loosening after total ankle arthroplasty (TAA). Apart from body weight, compressive forces induced by muscle action may affect joint loading. However, few studies have evaluated the influence of individual muscles on the intraarticular pressure distribution in the ankle.

Question/purposes The purpose of this study was to measure intraarticular pressure distribution and, in particular, (1) to quantify the effect of individual muscle action on peak-pressure magnitude; and (2) to identify the location of the center of pressure in the weightbearing native ankles and ankles that had TAA.

Methods Peak pressure and intraarticular center of pressure were quantified during force alterations of four muscle groups (peronei, tibialis anterior, tibialis posterior, and triceps surae) in 10 cadaveric feet. The pressure was measured with a pressure sensitive array before and after implantation of a three-component mobile-bearing TAA prosthesis. Linear mixed-effects models were calculated and the y-intercept (b_0) and the slope (b_1) of the regression were used to quantify the size of the effect.

Results Mean maximum peak pressures of 2 MPa (± 2.6 MPa) and 6.2 MPa (± 3.6 MPa) were measured for the native and TAA joint respectively. The triceps surae greatly affect the magnitude of peak pressure in the native ankle (slope $b_1 = 0.174$; $p = 0.001$) and TAA joint (slope $b_1 = 0.416$; $p = 0.001$). Furthermore, the force of most muscles caused a posterior and lateral shift of the center of pressure in both conditions.

Conclusions Our results suggest that muscle force production has the potential to alter the pressure distribution in the native ankles and those with and TAA.

Clinical Relevance Our study results help us to understand the effect of muscle forces on joint loading conditions which could be used in muscle training strategies and the design of better prosthetic components. Physical therapy or guided exercises may provide the potential to relieve areas in the joint that show signs of early osteoarthritis or reduce the contact stress on prosthetic components, potentially reducing the risk of TAA failure attributable to wear.

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Introduction

Osteoarthritis (OA) in the ankle is most commonly of secondary nature which usually is caused by trauma, such as ligament strain, bone fracture, or changes in muscle coordination [24]. All these factors are hypothesized to alter the loading pattern in the joint [1, 6], which is theorized to affect the genesis and progression of degenerative joint diseases [1, 6, 7]. Several studies have evaluated the effect of tibial fracture [1, 14], joint instability [23], and ligament injury and repair [20] on joint contact stresses and related it to the development of OA. However, the effective contribution of the muscles on the load distribution in the ankle during gait has not been fully explored. It has been suggested that muscle weakness can be a factor contributing to the genesis of OA in the knee [4, 11]; however, no similar literature exists for the ankle.

Muscle activation is assumed to play an important role in postsurgical joint stability, for instance, after total ankle arthroplasty (TAA) [9]. Current TAA implant designs rely on the soft tissue envelope for stabilization of the mobile bearing of the three-component prosthesis [10]. Therefore, muscle imbalance can affect how the prosthetic components are loaded, and the differences in loading magnitude may have an effect on the wear of the components. Furthermore, changes in the loading location might influence the stability of the prosthesis and eventually affect prosthesis-bone ingrowth, either in a positive or negative way.

Several in vitro studies have measured ankle contact characteristics using static [5, 8, 19, 20], quasistatic [15], or dynamic [22] simulations. Even though these studies are informative regarding loading conditions in the joint, they report only on the total effect of all muscles on contact loading and not on the contribution of individual muscles. Only Potthast et al. [19] performed static measurements where muscles were activated individually and reported on the individual effect of each muscle on intraarticular pressure distribution. However, nonfunctionally relevant muscle forces were applied to each muscle with an absolute maximum of 400 N for the triceps surae. Furthermore the effect of altered force on contact loading was not investigated for the triceps surae, the muscle delivering the higher forces during locomotion. Finally, Potthast et al. [19] studied the effect of muscle force with the foot in only one position (midstance).

We therefore sought to evaluate the effect of individual muscle forces on loading of the native ankle and TAA joint during three positions corresponding to different gait phases and relevant muscle-loading conditions. To achieve this, we measured intraarticular pressure distribution (1) to quantify the effect of individual muscle action on peak-pressure magnitude; and (2) to identify the location of the center of pressure in the weightbearing native ankles and ankles that had TAA.

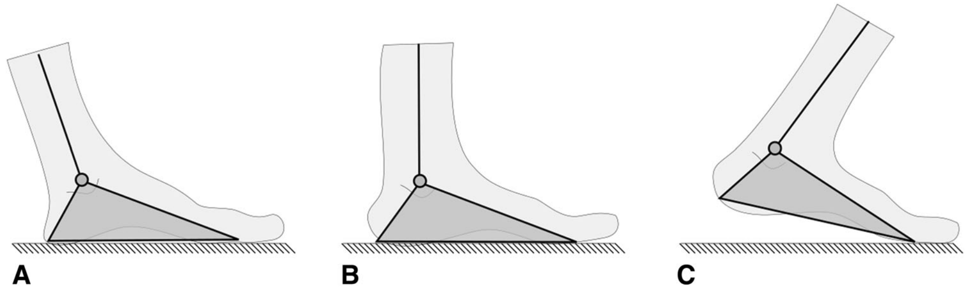
Materials and Methods

Ten freshly frozen cadaveric feet and lower legs (age and sex unknown, Institute for Orthopaedic Research and Training, Leuven, Belgium), amputated midtibially, were selected for study (six left, four right). The specimens were inspected radiographically for bony malformations or other visible disorders using CT and MRI and further inspection was performed on the preparations of the specimens. After thawing the specimens, the muscle belly was removed from nine tendons of the extrinsic muscles of the specimens (peroneus longus, peroneus brevis, tibialis anterior, extensor digitorum, extensor hallucis, tibialis posterior, flexor digitorum, flexor hallucis, triceps surae), and soft tissue was removed from the proximal part of the tibia. Two measurements were performed for each specimen: after measuring intraarticular pressure distribution in the native joint, a three-component TAA prosthesis (Hintegra®; Newdeal SA, Lyon, France) [3, 10] was implanted and the measurements were repeated. This prosthesis is a three-component mobile-bearing design consisting of talar and tibial components made of CoCr (ISO 5832/4). A highly congruent UHMWPE inlay is inserted between the components. The implantation was performed by an experienced surgeon (GD) using the instrument set provided by the manufacturer and following standard clinical procedure. All measurements were performed at room temperature.

To measure intraarticular pressure distribution, a Tekscan 5033 pressure sensor array (Tekscan Inc, Boston, MA, USA) was inserted in the ankle through an anterior incision of the skin, extensor retinaculum, and joint capsule. For measurements in the native ankle, the sensor was attached on the tibial side and was fixed with a metallic screw on the posterior side. For the TAA measurements, the sensor was inserted again through the anterior incision of the skin and was fitted between the tibial component and inlay of the prosthesis. The fixation point of the sensor was the same as for the native joint. Care was taken not to interfere with the path of any of the extensor tendons. The skin was stitched after insertion of the sensor.

Muscle force perturbation experiments were performed using a custom-built cadaveric gait simulator which has been described and validated [17]. To mount each specimen on the simulator, a metallic cylinder was fixed on the proximal side of the tibia using polyester resin (Motip Dupli BV, Wolvega, The Netherlands) and the mounting position was marked for further use. The tendons were attached to six pneumatic actuators using serrated clamps

Fig. 1A–C The three foot positions studied are shown. (**A**) Foot-flat corresponds to the beginning, (**B**) midstance to the middle, and (**C**) heel-off to the end-of-stance phase during gait.



to ensure proper force transfer from the actuators. Muscle tendons were grouped as (1) peroneus longus and peroneus brevis (peronei); (2) tibialis anterior, extensor digitorum, and extensor hallucis (tibialis anterior); (3) tibialis posterior; (4) flexor halluces; (5) flexor digitorum; and (6) gastrocnemius and soleus (triceps surae). To avoid excessive drying, the tendons were kept moist during insertion of the pressure sensitive array and implantation of the prosthesis. The actuators were operated in a force-feedback loop with the force measured by load-cells in series with the actuators. The gait simulator was operated with custom software programmed in LabVIEW 2013 (National Instruments, Austin, TX, USA).

Intraarticular pressure distribution was measured during three foot positions, representing three phases of stance during gait: at foot-flat, midstance, and heel-off (Fig. 1). The orientation and position of the tibia for each of the three positions were defined based on a specimen-specific tibial kinematic model [16] that accounts for the anatomic dimensions of each specimen. In each position, an upward force, corresponding to the vertical ground reaction force at that position, was applied by a plate beneath the specimen.

In each position, an initial force was imposed to each tendon corresponding to the muscle forces determined from inverse dynamics [13]. Using integrated three-dimensional motion capture data in combination with a geometric model of the lower leg, the muscle forces required to balance the external joint moments were calculated using a static optimization approach. The muscle force distribution was penalized for optimization solutions that introduce a high level of muscle cocontraction. To ensure cadaveric integrity, the forces calculated by the inverse dynamics analysis were reduced by 50%. A maximum force then was defined for each tendon at 1.2 times the maximum value determined from the inverse dynamics calculation for duration of the stance phase. The force delivered on the four main muscle groups (peronei, tibialis anterior, tibialis posterior, and triceps surae) was altered, one group at a time. During the alterations for each group, the force from the other groups was held constant. The force initially was increased, in four increments, until the maximum defined was reached, then decreased to zero in eight decrements, and then restored to the initial force in four increments. All increments and decrements were equally spaced, and the force was maintained constant for 3 seconds after each increment (Table 1).

Table 1. Summary of muscle forces applied during the experiments*

Muscle	Phase	Maximum force (N)	Initial force (N)
Peronei	Foot-flat	187 ± 36	123 ± 26
	Midstance	187 ± 38	71 ± 57
	Heel-off	186 ± 37	91 ± 39
Tibialis anterior	Foot-flat	612 ± 95	473 ± 45
	Midstance	609 ± 86	196 ± 45
	Heel-off	593 ± 87	129 ± 46
Tibialis posterior	Foot-flat	216 ± 55	140 ± 45
	Midstance	212 ± 53	66 ± 34
	Heel-off	203 ± 51	56 ± 39
Triceps surae	Foot-flat	938 ± 331	303 ± 74
	Midstance	994 ± 266	421 ± 102
	Heel-off	1153 ± 309	835 ± 357

* Mean ± SD among all specimens of maximum and initial forces for each muscle, position, and case.

During the simulations, the pressure distribution was captured with the Tekscan sensor using I-scan software (Tekscan Inc) at 10 Hz. For each specimen and condition, a new sensor, calibrated before the experiment, was used (Appendix 1).

Data Analysis

To cancel sensor noise, the measured pressure distribution was averaged for the duration of each increment (3 seconds). For each increment the magnitude of the peak pressure and location of the center of pressure were calculated. To relate the peak pressure in the joint and the applied muscle force for the different specimens, all muscle forces were normalized against the initial force determined for each muscle and position from the inverse dynamics calculation. Additionally, the peak pressure measured at each increment was normalized against the initial value for each position before altering the muscle forces.

Table 2. Summary of peak pressure measured during the experiments*

Phase	Case	Maximum peak pressure (MPa)	Initial peak pressure (MPa)
Foot-flat	Native	1.57 ± 1.84	0.79 ± 0.86
	TAA	6.31 ± 3.48	3.04 ± 1.99
Midstance	Native	2.09 ± 3.02	1.18 ± 1.83
	TAA	6.21 ± 4.07	2.80 ± 1.72
Heel-off	Native	2.38 ± 3.13	1.80 ± 2.96
	TAA	6.15 ± 3.91	3.22 ± 1.71

* Shown as mean ± SD among all specimens of maximum and initial forces for each muscle, position, and case; TAA = total ankle arthroplasty.

To define a relationship between the muscle actuation and the peak-pressure magnitude and the relationship between muscle actuation and center of pressure location, linear mixed-effect models were used for each muscle, phase, and condition of the foot [18]. The linear mixed-effect models accounted for random variability owing to the 10 specimens selected for these measurements in our study. Three models were formulated for each muscle, phase, and condition: one with the response variable of peak-pressure magnitude; one with the response variable of center-of-pressure location in the AP direction; and one with the response variable of the center-of-pressure location in the mediolateral direction. The fixed effect of the model was the actuation of each muscle and the random effect was the 10 different foot specimens. The model outputs are the two parameters of a regression line, one describing the y-intercept (b_0) and one as the slope (b_1) of the regression. These parameters can be used to predict a response of the model (L) for a specific actuation of a muscle (A) based on equation 1.

$$L = b_0 + b_1 A \quad (1)$$

The estimates of the regression optimized the restricted maximum likelihood (criterion and an intercept and a coefficient were calculated for each muscle, phase, and condition). Statistical analysis was performed using R v3.1.1 [21] and the nlme v3.1.117 [18] package. Statistical significance was set at p less than 0.05. Only the muscles that had a significant influence on peak pressure or center-of-pressure location are presented in our results.

Results

Peak-Pressure Magnitude

For the native ankle, the mean peak pressure measured was 1.57 MPa (SD ± 1.84 MPa), 2.09 MPa (SD ± 3.02 MPa), and 2.38 MPa (SD ± 3.13 MPa) for the foot-flat, midstance, and heel-off positions (Table 2) (Fig. 2). The actuation of the peroneal muscles did not reveal any influence on peak pressure for any of the three positions (Table 3) (Fig. 3). Actuation of the tibialis anterior increased the peak-pressure magnitude at the foot-flat ($b_1 = 0.18$; $p = 0.001$) and midstance ($b_1 = 0.06$; $p < 0.001$) positions, but not during heel-off ($b_1 = -0.005$; $p = 0.107$). Actuation of the tibialis posterior decreased the peak-pressure magnitude at the foot-flat position ($b_1 = -0.052$; $p = 0.001$), whereas the triceps surae actuation increased the peak-pressure magnitude at the foot-flat position ($b_1 = 0.17$; $p < 0.001$). No other influences were found for the native ankle.

For the TAA joint, the peak pressure measured was 6.31 MPa (SD ± 3.48 MPa), 6.21 MPa (SD ± 4.07), and 6.15 MPa (SD ± 3.91) for the foot-flat, midstance, and heel-off positions. All muscles revealed influences in at least one position. Actuation of the peroneal muscles decreased the peak pressure at the heel-off position ($b_1 = -0.1$; $p = 0.01$). Actuation of the tibialis anterior decreased the peak pressure at midstance ($b_1 = -0.06$; $p = 0.043$) and heel-off ($b_1 = -0.02$; $p = 0.003$) positions. Peak pressure also decreased with actuation of the tibialis posterior at foot-flat position ($b_1 = -0.08$; $p < 0.001$); however, it increased at midstance ($b_1 = 0.01$; $p = 0.007$). Finally, actuation of the triceps surae increased the magnitude of peak pressure in all three positions ($b_1 = 0.41$, $p < 0.001$; $b_1 = 0.71$, $p < 0.001$; and $b_1 = 0.35$, $p = 0.001$ at foot-flat, midstance, and heel-off, respectively).

Center-of-Pressure Location

For the native joint (Table 4) (Fig. 4), the peronei and triceps surae shifted the center of pressure in the ankle posteriorly in the foot-flat position ($b_{1AP} = -0.42$, $p = 0.007$; $b_{1AP} = 0.64$, $p = 0.001$), whereas the tibialis posterior shifted it posteriorly during midstance ($b_{1AP} = -0.08$, $p = 0.008$). In the mediolateral direction, the peronei, triceps surae, and tibialis posterior shifted the center of pressure laterally in midstance, foot-flat, and heel-off positions, respectively ($b_{1ML} = -0.06$, $p = 0.041$; $b_{1ML} = 0.66$, $p = 0.015$; $b_{1ML} = 0.03$, $p = 0.024$), although the tibialis anterior shifted the center of pressure medially in midstance position ($b_{1ML} = -0.2$, $p = 0.007$).

Similar effects were observed for the TAA joint (Table 4). The peronei, tibialis posterior, and triceps surae shifted the center of pressure posteriorly in foot-flat position ($b_{1AP} = -0.16$, $p = 0.038$; $b_{1AP} = -0.37$, $p < 0.001$; $b_{1AP} = -0.28$, $p = 0.008$). Additionally, the peronei caused a posterior shift in the heel-off position ($b_{1AP} = 0.09$; $p = 0.007$). In the mediolateral direction, the tibialis anterior caused a lateral and tibialis posterior medial shift in

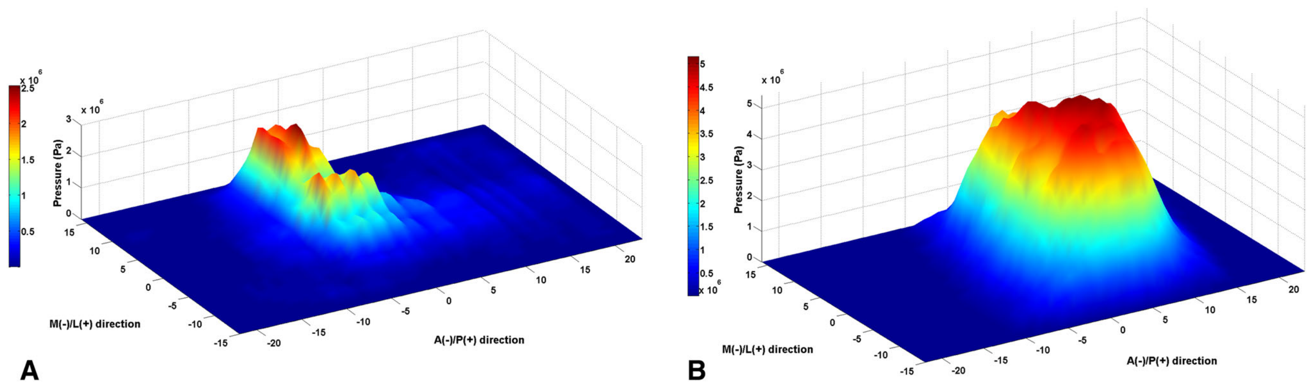


Fig. 2A–B Pressure distribution in the (**A**) native ankle and (**B**) ankle that had total ankle arthroplasty are shown. The mediolateral (ML) and AP directions are indicated in the axes. The different color and height of the surface indicate the magnitude of the pressure measured at each location.

Table 3. Summary of results of linear mixed-effects modeling*

Phase	Muscle	Case	Intercept (b_0)	Coefficient (b_1)	p value
Foot-flat	Peronei	Native	1.382	−0.036	0.166
		TAA	1.318	−0.104	0.01†
	Tibialis anterior	Native	1.215	0.186	0.001†
		TAA	1.068	0.157	0.081
	Tibialis posterior	Native	1.263	−0.052	0.001†
		TAA	1.116	−0.085	0†
	Triceps surae	Native	1.238	0.174	0†
		TAA	0.89	0.416	0†
Midstance	Peronei	Native	1.06	0.003	0.517
		TAA	1.168	−0.02	0.378
	Tibialis anterior	Native	0.935	0.062	0†
		TAA	1.232	−0.064	0.043†
	Tibialis posterior	Native	1.016	0.005	0.128
		TAA	0.984	0.018	0.007†
	Triceps surae	Native	1.071	0.036	0.204
		TAA	0.818	0.718	0†
Heel-off	Peronei	Native	1.096	0.019	0.722
		TAA	1.07	−0.049	0.072
	Tibialis anterior	Native	1.165	−0.005	0.107
		TAA	1.175	−0.026	0.003†
	Tibialis posterior	Native	1.07	−0.002	0.501
		TAA	1.093	−0.009	0.356
	Triceps surae	Native	0.968	−0.087	0.429
		TAA	0.67	0.355	0.001†

* Intercept (b_0) and coefficient (b_1) estimates are presented for when the peak pressure was the response variable. A positive coefficient estimate corresponds to an increase of peak-pressure magnitude with increase in muscle force; $^\dagger p < 0.05$; TAA = total ankle arthroplasty.

the center of pressure during midstance ($b_{1ML} = -0.9$, $p = 0.032$; $b_{1ML} = 0.11$, $p = 0.002$). Finally, the tibialis posterior and triceps surae caused a medial shift during foot-flat position ($b_{1ML} = 0.23$, $p = 0.012$; $b_{1ML} = 0.2$, $p = 0.015$).

The data discussed in this study are provided in the form of a table in long format (Appendix 2. Supplemental material is available with the online version of CORR®. The supplementary material can be opened with the program R, which is free; you can get R at: www.r-project.org.). The

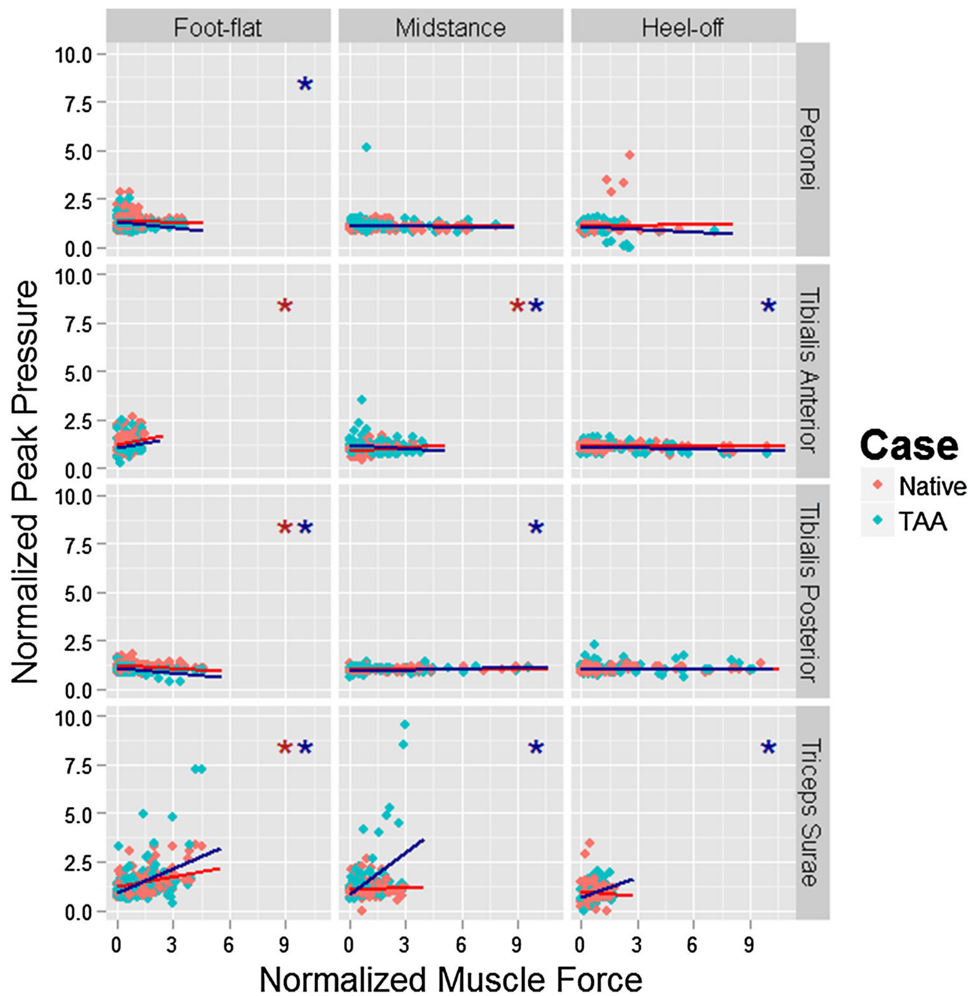


Fig. 3 The relationship between muscle force and peak-pressure magnitude for the native ankle (red) and ankle that had a total ankle arthroplasty (TAA) is shown (blue). The dots represent the measured peak pressure (y-axis) for a specific applied muscle force (x-axis). The values for peak pressure and muscle force are normalized with respect to the initial values at the beginning of each position. The relationship is presented for the three positions (horizontal spacing) and four muscles (vertical spacing). For each position and muscle, the regression line also is presented. *Significant influence of muscle force on the magnitude of the peak pressure ($p < 0.05$) for the native (red) and TAA joint (blue).

table consists of nine columns describing for each row the foot number (Foot), the condition (Case), the muscle that is being activated (Muscle), the phase of stance that the measurement was obtained (Phase), the normalized actuation applied on the specific muscle (Actuation), the peak pressure measured (PP), the normalized peak pressure calculated (PPnorm), the location of the center of pressure in the AP direction (CoPAP), and the location of the center of pressure in the mediolateral direction (CoPML).

Discussion

Muscle forces have been hypothesized to influence generation and progression of ankle OA by influencing the loading conditions in the joint. Furthermore, muscle force transfer is crucial for the success of current designs of TAA prostheses as it can influence the contact stress between the components and subsequently their wear. The main focus of this study was to document the influence of individual muscle forces on loading conditions in the native ankle and the ankle that had TAA in postures representative of three phases of the stance portion of the gait cycle. More specifically, we sought to examine the influence of individual muscle forces on the peak-pressure magnitude and the location of the center of pressure. We discuss the specific muscles that can cause either an increase or a decrease of the magnitude of peak pressure (triceps surae and tibialis posterior respectively). Furthermore, we showed that several muscles can affect the location of the center of pressure by translating it medial and laterally in most cases.

This study has numerous limitations. First, the reported pressure distribution might be affected by the use of a cadaveric model with the inherent changes in the material properties of the cartilage postmortem and the loss of fluid of the joint capsule after the incisions. However, in vitro experimentation is currently the only method for directly measuring intraarticular pressure distribution and for actuating muscles individually. Furthermore, extra care was taken to restore the retinaculum and skin by suture, minimizing the influence on the loading conditions. Second, the study is limited by the static nature of the

Table 4. Summary of results of linear mixed-effects modeling*

Direction	Phase	Muscle	Case	Intercept (b_0)	Coefficient (b_1)	p value
AP	Foot-flat	Peronei	Native	−1.297	−0.424	0.007†
			TAA	4.039	−0.16	0.038†
		Tibialis anterior	Native	−0.81	0.064	0.907
			TAA	3.39	0.29	0.312
		Tibialis posterior	Native	−1.24	−0.144	0.355
			TAA	3.881	−0.377	0†
		Triceps surae	Native	0.403	−0.644	0.001†
			TAA	3.815	−0.282	0.008†
	Midstance	Peronei	Native	0.19	−0.003	0.934
			TAA	3.623	−0.04	0.086
		Tibialis anterior	Native	−0.013	0.155	0.098
			TAA	3.369	−0.048	0.468
		Tibialis posterior	Native	0.161	−0.088	0.008†
			TAA	3.317	−0.014	0.637
		Triceps surae	Native	0.084	−0.048	0.717
			TAA	3.647	0.087	0.504
	Heel-off	Peronei	Native	−0.43	−0.111	0.458
			TAA	3.572	−0.094	0.007†
		Tibialis anterior	Native	−0.321	0.003	0.839
			TAA	3.447	−0.019	0.513
		Tibialis posterior	Native	−0.215	−0.021	0.344
			TAA	3.482	−0.021	0.524
		Triceps surae	Native	0.707	0.149	0.856
			TAA	3.886	0.235	0.221
ML	Foot-flat	Peronei	Native	1.271	−0.046	0.749
			TAA	−0.566	0.235	0.093
		Tibialis anterior	Native	0.963	0.179	0.733
			TAA	−0.149	−0.302	0.132
		Tibialis posterior	Native	0.929	−0.208	0.082
			TAA	−0.568	0.236	0.012†
		Triceps surae	Native	−0.685	0.665	0†
			TAA	−1.111	0.204	0.015†
	Midstance	Peronei	Native	0.696	0.065	0.041†
			TAA	−0.708	−0.01	0.637
		Tibialis anterior	Native	1.351	−0.202	0.007†
			TAA	0.034	−0.096	0.032†
		Tibialis posterior	Native	0.858	−0.034	0.372
			TAA	−0.676	0.11	0.002†
		Triceps surae	Native	1.068	0.009	0.927
			TAA	0.067	−0.228	0.079
	Heel-off	Peronei	Native	0.991	−0.025	0.65
			TAA	−0.426	0.023	0.457
		Tibialis anterior	Native	1.19	−0.019	0.289
			TAA	−0.508	0.007	0.72
		Tibialis posterior	Native	0.859	0.036	0.024†
			TAA	−0.318	0.041	0.098
		Triceps surae	Native	1.16	0.083	0.886
			TAA	0.228	−0.232	0.368

* The intercept (b_0) and coefficient (b_0) estimates are presented for the center of pressure in the AP and ML directions as the response variable. A positive estimate corresponds to displacement of the center of pressure toward the anterior and lateral direction with increase in muscle force; †$p < 0.05$; AP = Anteroposterior; ML = mediolateral; TAA = total ankle arthroplasty.

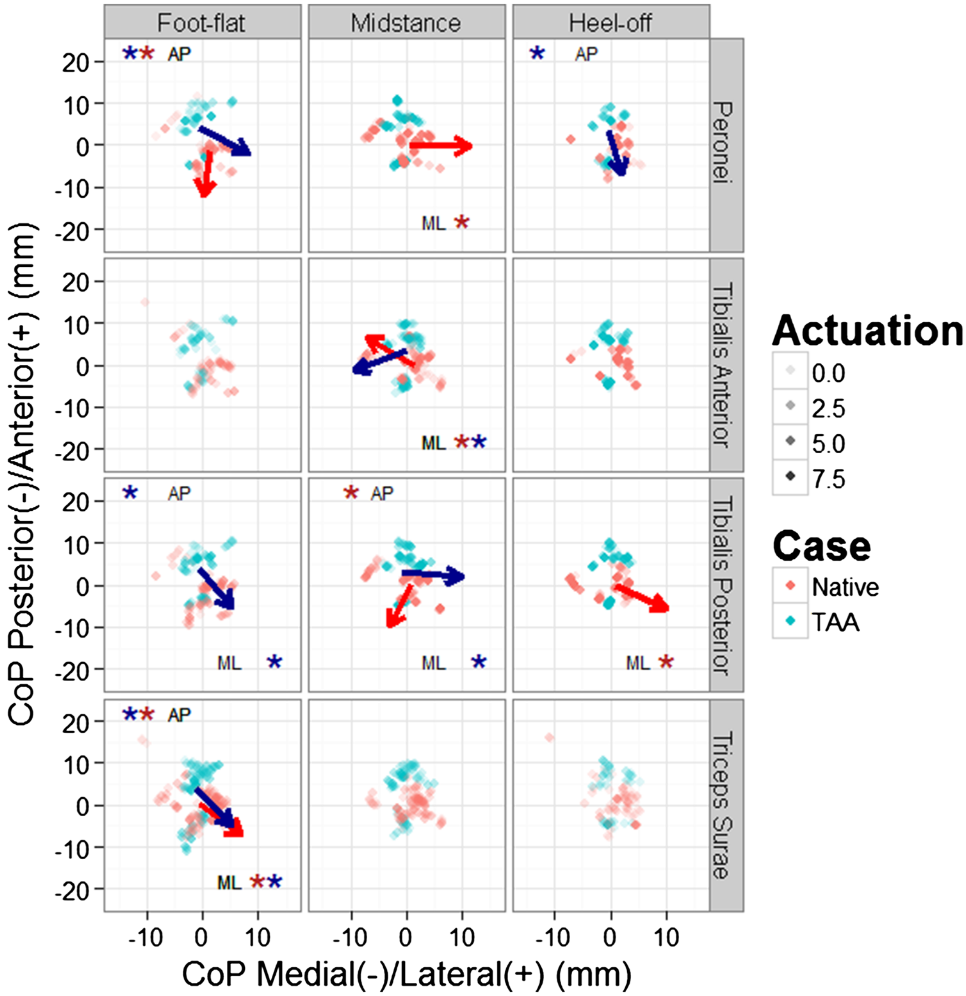


Fig. 4 The location of the center of pressure (CoP) and the influence of the muscle actuation on the location for the native ankle (red) and the ankle that had a total ankle arthroplasty (TAA) (blue) are shown. The intensity of the colors indicates the magnitude of the muscle actuation. Arrows are visible for instances when significant influence was detected in either of the two directions, indicated by (*) on the top left for the AP and on the bottom right for the mediolateral (ML) direction ($p < 0.05$). The red and blue stars correspond to the native and TAA joint respectively. The arrows are constructed based on the equation $L_j = b_{0j} + b_{1j} {*}A$, where L_j is the location of the center of pressure, b_{0j} is the estimated intercept, and b_{1j} is the estimated coefficient, j, corresponding to AP and ML directions.

measurements which might not adequately reflect the changes in the joint loading conditions during dynamic walking. However, the applied muscle action was representative of the phase of the gait cycle, therefore reducing to some extent this liability. To fully address this issue, numerous dynamic roll-offs must be performed, each imposing a change of a specific muscle force in a specific phase of the gait cycle. These subsequent repetitions can be harmful for the integrity of the cadaveric specimens, therefore requiring the measurements to be terminated prematurely. Finally, reduced forces (50% body weight) were applied on the tendons during the measurements, which is an inherent limitation of in vitro simulations. Some researchers performing in vitro simulations use reduced muscle forces to ensure cadaveric integrity [22, 25], with a typical reduction of the muscle forces by 50%, with reported limited effect on bone kinematics [2].

In the native joint, muscle actuation was found primarily to increase the peak pressure for three of the four muscle groups studied; only the peroneal muscles had no influence with the numbers available in any of the three foot positions. Increasing the force of the triceps surae increased the peak pressure, whereas increasing the force of the tibialis posterior decreased it in the foot-flat position. This finding is in contrast to the findings of Potthast et al. [19], who reported increased peak pressure with increased force production of the tibialis posterior in the midstance position. This difference could be attributable to the absence of synergistic muscle forces [19], which could alter the point of contact and therefore the moment of the muscle. The ankle that had TAA showed similar behavior as the native joint, and the peak pressure was affected by all four muscle groups studied. Increased force from the triceps surae resulted in increased peak pressure in all positions studied, whereas increased muscle force from the peroneal and tibialis posterior muscles resulted in decreased peak pressure. The direction of the influence was the same as in the native joint in almost all cases, however it was more pronounced. This might be related to the higher congruency of the surfaces after TAA and the increased stiffness of the materials that limit load redistribution on adjacent joint surfaces.

Furthermore, in the native ankle, most muscles shifted the center of pressure toward the posterior and lateral directions, with the exception of the tibialis anterior, which caused a medial shift during midstance. This finding again is in contrast to the observations of Potthast et al. [19], who reported a medial shift of the joint load with increased force of the tibialis posterior. However in their study, the data were evaluated qualitatively only and no statistical analysis was performed. Furthermore, their experimental protocol used considerably lower forces (maximum force for the triceps surae was 400 N compared with 1400 N in our study), and only one muscle was actuated at a time, with the rest being inactive. In contrast, we imposed synergistic muscle forces that were more representative for each studied position. Similarly, in the ankle that had TAA, the influence of muscle force on the center of pressure was detected although to a lesser extent than in the native joint. This less pronounced effect could be explained by the higher congruency and stiffness of the surfaces that cause the load to increase instead of redistributing.

Results of our study support those of previous studies [1, 8, 19, 22] that muscle forces are capable of modifying joint loading in magnitude and location. However, the effect differs among muscles and also depends on the foot position [5]. These findings are relevant as they support the use of muscle-training strategies to affect joint-loading magnitude and position. Such strategies might be beneficial in the prevention of OA or rehabilitation of patients with early OA, as it has been suggested [1, 6] that increased joint load is related to ankle OA. More specifically, force production of the triceps surae was found to shift the load toward the posterior and lateral regions of the native ankle. Therefore, strengthening the gastrocnemius muscle could be useful in patients with OA to migrate the center of pressure away from the more vulnerable areas of the ankle such as the anteromedial regions of the talus where higher percentages of OA onset are reported [12]. Furthermore, strengthening of the tibialis posterior might have a beneficial effect, as we found that increased muscle force from the tibialis posterior reduced the peak-pressure magnitude at the foot-flat position.

We showed that the muscles influence the contact loading magnitude and distribution in a native ankle and an ankle that has had a TAA between the tibial component and the inlay of a three-component prosthesis. Differences in the joint axis attributable to prosthesis design and implantation technique can cause differences in moment arms of the tendons that thereby will change the effect of most muscles on joint loading. As such muscle alterations after implantation of TAA have been reported [9], this might lead to wear and component loosening of the prosthesis.

We found that muscle forces have an important effect on the magnitude and topology of the loading conditions of the native ankle and on the ankle that has had a TAA. The TAA results refer to a specific TAA design and should be explored with other designs as well. The described effects possibly could be explored as part of training exercises and also could be useful in consideration of the design of new TAA devices.

Appendix 1: Tekscan sensor calibration

Each sensor was calibrated separately on a date close to the measurement date. It was fitted between two flat plates (DIN 6346, P40 × 12 × 160, AMF, Fellbach, Germany), which were mounted on an Instron 4467 compression bench (Instron, MA, USA). To ensure parallel alignment, a universal joint was placed between the compression bench and the bottom plate. Each sensor was loaded under 15 loading levels ranging from 350 N until 6500 N with an increment of 400 N. A piecewise cubic hermite interpolating polynomial was used between the points of the calibration to determine the calibration curve of each sensor. As the Tekscan measurement system allows setting the sensitivity of the sensors in six different levels (Low-3, Default, Mid-1, Mid-2, High-1, High-2), the calibration was performed for each of these levels. The suitable sensitivity level for the measurements was chosen so that a linear relationship was found in the area of interest (0–7 MPa), in this example the High-1 sensitivity level. For all the sensors, either the Mid-2 or High-1 sensitivity was used.

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