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Review

The biomechanics of heel ulcers

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Pressure ulcer;
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Bed sore;
Mathematical model;
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Abstract Heel ulcers are common, dangerous and costly, but their etiology is poorly understood and no biomechanical studies were conducted to explore it. This paper describes a biomechanical investigation of heel ulcers using a theoretical model that characterizes the internal mechanical loading at the soft tissues of a supported heel. The study is aimed first at identifying some heel-ulcer-specific risk factors pointed out by the biomechanical theory, and second, at demonstrating the kind of support that biomechanical theory and computer modeling can offer in the conduct of clinical studies in the pressure ulcer field. The modeling demonstrated that atypical foot anatomies characterized by heavy-weight foot, sharp posterior calcaneus and thin soft tissue padding are theoretically more prone to heel ulcers. Diabetes and edema at the feet were also predicted to impose risks for heel ulcers, which agrees very well with clinical observations. This paper therefore demonstrated that a biomechanical theory can be used to explain and interpret clinical and epidemiological findings related to heel ulcers.

Introduction

In hospital settings, pressure ulcers most commonly occur in the heels, and overall, heel ulcers are reported to be the second most common type of pressure ulcers [1]. As with other pressure ulcers, heel ulcers might involve serious, life-risking complications such as osteomyelitis, septicemia, amputation of the affected limb, renal failure and multiple organ system failure [2]. Direct costs of treating a single heel ulcer are in the order of thousands to tens-of-thousands U.S. dollars [1]. Despite being so common, despite imposing such high risks, and in spite of being so costly, heel ulcers are considerably understudied in the pressure ulcer literature, with actually not even a single published paper to-date that addressed the biomechanical factors in their etiology.

Biomechanical computer-aided modeling has come a long way from serving merely scientists interested in basic research, to a practical tool that supports clinical and epidemiological studies in pressure ulcers [3—7]. This paper describes a biomechanical investigation of heel ulcers using a theoretical model that characterizes the internal mechanical loading at the soft tissues of a supported heel. The study is aimed first at identifying some heel-ulcer-specific risk factors pointed out by the biomechanical theory, and second, at demonstrating the kind of support that

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biomechanical theory and computer modeling can offer in the conduct of clinical studies in the pressure ulcer field.

Heel ulcers can be open wounds at the time of diagnosis, or there might be evidence of necrotic tissue damage under intact skin — a condition previously termed "the purple heel" — which is in fact a deep tissue injury [8,9]. It has been suggested that even the open wounds could initiate subdermally, and remain undiagnosed until breakdown of the skin occurred [10,11]. This "silent progress" pathway of heel ulcers from subdermal tissues onto the skin indicates how important it is to screen patients on admission to hospitals, nursing homes and rehabilitation centers, in attempt to estimate the internal mechanical loads in the soft tissues of their heels while the heels are being supported, and based on that, to assess the patients' individual risk of developing heel ulcers.

Based on epidemiological studies or clinical observations and experience, some co-morbidities were previously proposed to be associated specifically with heel ulcers, namely: immobile legs (e.g. due to fractured hips, joint replacement surgeries, spinal cord injuries and stroke), diabetes, lower limb atherosclerosis and other peripheral vascular diseases, leg spasms, leg pain and dementia [12—14]. However, the available quantitative data correlating co-morbidities and risks are rather sparse and the etiology is still essentially unexplained, probably because it is difficult, costly and labor-intensive to conduct large scale studies on heel ulcers.

It is a common problem of epidemiological studies in pressure ulcer research at large, that only a fraction of the patients who are being followed-up actually develop ulcers during the study period. It is also typically difficult to obtain patient cooperation as susceptible patients are, many times, critically ill or unable to consent. Development of risk assessment tools for heel ulcers also appears to lag behind legal actions, and so, an increasing number of lawsuits in the U.S. as well as in Europe claim that patients were neglected despite the possibility that caregivers actually followed the risk assessment guidelines available to them, but that these guidelines were inadequate for protecting vulnerable individuals from heel ulcers. This is why it is so critical to backup epidemiological studies in this field with biomechanical theory that explains or justifies the observations at the clinical setting.

The present paper was therefore written mostly for clinicians in the field of tissue viability, and accordingly, less focus is put on the mathematical formulation of the model and the derivation of its equations (though for completeness and reproducibility, the most important equations and key steps in derivation were included herein). The emphasis here is on the interpretation of the model predictions for identification of risk factors, as well as on the generic use of biomechanical modeling to support risk factor studies by providing the theoretical framework to explain epidemiological findings or clinical observations. Consistent with these purposes, the present biomechanical model of the heel was designed to be relatively simple in terms of mathematical formulation as well as physical interpretation, that is, the model was designed to contain a limited number of parameters which all have physiological or pathophysiological relevance and a possible role in the etiology of heel ulcers, as explained below.

Mathematical model of soft tissue loads under the supported calcaneus

The mathematical formulation necessary for describing and reproducing the present heel model is provided below. This heel model builds on the work of Ning and colleagues [15], who, in a different field of research, developed a theory to study the endurance of surface coatings for material engineering applications. From a purely mathematical point of view, the formulation of the problem of a sphere that is compressed against a thin elastic layer which was used by Ning to describe wear of coatings [15], is also adequate for describing the pressure applied by the calcaneus bone on the overlying thin layer of soft tissues when the heel is supported. The assumptions that are needed for applying the Ning model [15] to the present heel ulcer biomechanical studies are: (i) that the posterior aspect of the heel can be simplified to the shape of a sphere, and (ii) that the soft tissues covering it are a thin homogeneous linear-elastic layer (Fig. 1). The latter assumption means that the skin, subcutaneous fat and possibly the distal attachment of the Achilles tendon are considered together as one effective "soft tissue" material with a given material stiffness, which does not depend on the intensity of the internal soft tissue loading. The mathematical and mechanical considerations used for formulating the theory were described in full by Ning [15] and are beyond the scope of this paper. Practically, we adopt one useful result from the Ning [15] paper for the present study, that given the assumptions of homogeneity and linear-elasticity for the soft tissues at the posterior calcaneus (as explained

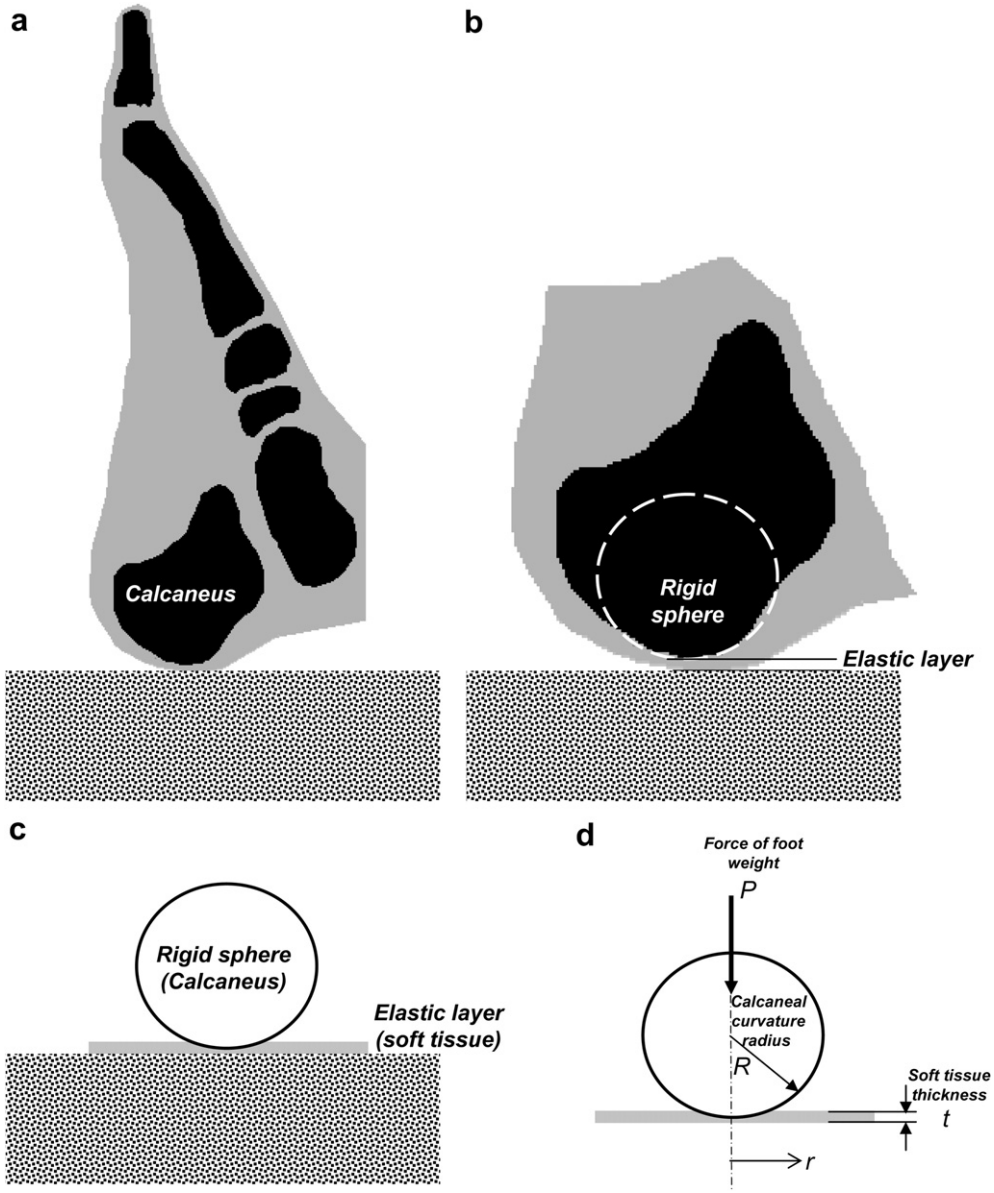

Figure 1 The biomechanical model of the heel used herein to study the risk for heel ulcers: (a) Real-world condition of a foot resting on a horizontal support so that the heel is compressing against the support. (b) For the purpose of biomechanical modeling, the curved shape of the posterior calcaneus can be approximated as a rigid sphere that compresses a soft tissue layer. (c) An engineering model (i.e. an idealized representation) of the heel at this posture would therefore be a rigid sphere (calcaneus) that is pressing against an elastic layer (soft tissues). (d) The resting heel problem can now be described with a set of quantitative parameters, such as the force of the foot weight, radius of curvature of the calcaneus, and thickness and stiffness of the overlying soft tissues.

above), the internal pressure between the calcaneus and the overlying tissues is bounded between lower and upper limits:

$$k_1\left[\frac{2\lambda_1\sqrt{\frac{PRt}{\pi}-r^2}}{2Rt}\right]\le p(r)\le k_2\left[\frac{2\lambda_2\sqrt{\frac{PRt}{\pi}-r^2}}{2Rt}\right] \qquad (1)$$

where $p(r)$ is the pressure between the calcaneus bone and soft tissue at distance r from the point where the soft tissue is the thinnest, P is the force of weight of the foot (while it is hypothetically considered to be detached from the body), R is the radius of curvature of the posterior calcaneus, t is the thickness of overlying soft tissues (Fig. 1), and k_1, k_2, λ_1,λ_2 are model constants that characterize the elastic behavior of the soft tissues:

$$\begin{aligned} k_1&=\tfrac{E}{1-\nu^2};\quad \lambda_1=\sqrt{\tfrac{1-\nu^2}{E}}\\ k_2&=\tfrac{(1-\nu)E}{(1+\nu)\,(1-2\nu)};\quad \lambda_2=\sqrt{\tfrac{(1+\nu)\,(1-2\nu)}{(1-\nu)E}} \end{aligned} \qquad (2)$$

where E is the elastic modulus of the soft tissues, which is a measure of tissue stiffness, and ν is the Poisson's ratio of soft tissues, which is a measure of their compressibility. The point of maximal soft tissue pressures is where the tissues are thinnest, just below the lowest point of the posterior calcaneus, i.e. where in the model, $r = 0$. For this particular point in the soft tissues, Eq. (1) reduces to:

$$\sqrt{\frac{PE}{\pi(1-\nu^2)Rt}} \le p(r=0) = p_{\max} \le \sqrt{\frac{(1-\nu)PE}{\pi(1+\nu)\,(1-2\nu)Rt}} \quad (3)$$

The reason for the existence of the upper and lower limits for $p_{\max}$ is that they define two extreme conditions of friction between the calcaneus and overlying soft tissues. The lower limit considers a condition where there is no such friction at all, and the upper limit assumes that this friction is so high that the soft tissues are actually bonded to the bone surface. The physiological condition is very likely in-between these two extremes. Hence, for the practical purpose of modeling, it is reasonable to estimate the maximal pressure in the soft tissues under the calcaneus $p_{\max}$ by averaging the upper and lower limits in Eq. (3). This yields a useful mathematical formula for estimating the maximal soft tissue pressure $p_{\max}$ when the heel is resting on its posterior aspect (Fig. 1), depending on important anatomical and (patho)physiological factors which could potentially relate to heel ulcers:

$$p_{\max} = \frac{1}{2}\sqrt{\frac{PE}{\pi Rt}}\left[\sqrt{\frac{1}{(1-\nu^2)}} + \sqrt{\frac{(1-\nu)}{(1+\nu)\,(1-2\nu)}}\right] \quad (4)$$

Specifically, the above Eq. (4) provides evaluation for the maximal pressure in soft tissues overlying the posterior calcaneus $p_{\max}$ as function of the weight of the foot (P), stiffness of the soft tissues (E), curvature of the posterior calcaneus (R), thickness of the soft tissues (t) and compressibility of the soft tissues (ν) (Fig. 1). We will now test the effects of these parameters, when associated with clinically meaningful factors including anatomical foot structures, diabetes and local edema or tissue dehydration at the posterior heel, on internal soft tissue pressures, in an attempt to identify risk factors for heel pressure ulcers based on this theoretical model. For this purpose, a bioengineering methodology of analysis called a "sensitivity analysis" will be used. In bioengineering research, sensitivity analyses of models are aimed at testing how variations in the output parameters of the model (being the maximal soft tissue pressure in the posterior heel $p_{\max}$ in this case) can be apportioned to variations in the input parameters (P, E, R, t, ν here). For example, how would $p_{\max}$ be affected by a sharper posterior surface of the calcaneus (low R), or by a thinner soft tissue padding at the posterior heel (low t). This essentially requires definition of a set of nominal values for the parameters, which are representative of a normal foot/heel anatomy and physiology, and which can then be altered, one at a time (while keeping all the other parameters at their nominal values), to test the effects on $p_{\max}$. These nominal model parameter values are provided in Table 1.

Effects of anatomical, physiological and pathophysiological factors

Internal foot anatomy and body habitus

A first practical basic question which can be addressed using the present model is: Would a patient with motosensory impairments who is

Table 1 Model parameters and their nominal values.

Model parameter	Description	Nominal value	Reference
P	Force of foot weight	1 kg	[16,17]
E	Stiffness of soft tissues overlying the posterior calcaneus	100 kPa	[18]
R	Radius of curvature of the posterior calcaneus	20 mm	[19]
t	Thickness of soft tissues overlying the posterior calcaneus	5 mm	[20]
ν	Compressibility of soft tissues overlying the posterior calcaneus	0.495	[21]

lying supine in bed and whose feet are bigger and heavier than normally, be theoretically at an increased risk for heel pressure ulcers because of the big feet? In typical real-world conditions, patients with big feet would either have a proportional body structure, or they may be overweight or obese. For an obese patient, the increased foot mass would be due to an increase in soft tissue mass (mostly fat) around the foot skeleton, whereas patients whose body structure is overall bigger would also have a bigger and heavier foot skeleton. In other words, the sizes of the foot bones (and the calcaneus among them) do not change when an individual becomes overweight. In qualitative terms, the internal foot anatomy of an obese patient should therefore typically show an averaged-sized calcaneus enveloped by thicker-than-normal soft tissue layers. This is as opposed to a subject with a congenitally bigger body structure, who is likely to keep a proportion between the dimensions of the calcaneus and overlying soft tissues. Corresponding to these qualitative considerations, the model was analyzed to test how p_{max} is being affected by changes in foot weight (P), alone or in combination with changes in the radius of curvature of the posterior calcaneus R (which increases when the calcaneal size grows), and with changes in thickness of the soft tissues overlying the posterior calcaneus (t). The model data in this regard, plotted in Fig. 2a, first shows that when just the foot mass increases, internal soft tissue pressures p_{max} increase as well. However, when the radius of curvature of the posterior calcaneus increases, p_{max} decreases (Fig. 2a, left plot). Accordingly, the model suggests that theoretically, patients who congenitally have a bigger body structure and hence bigger feet, are not necessarily at risk for heel pressure ulcers, because their larger posterior calcaneal surface spreads the loads imposed by their heavier feet. In other words, the heavy foot and wider posterior calcaneus factors have counteracting effects on p_{max}. Increasing the soft tissue thickness together with the foot weight, without changing the radius of curvature of the calcaneus, to study a process that is expected for feet of an overweight patient, shows a similar trend, where the effect of each added 150 g of foot weight is compensated by an increase of 1 mm in soft tissue thickness (Fig. 2a, right plot). Therefore, based on these model simulation data, an overweight patient whose soft tissues that cover the posterior calcaneus are thicker than normal is also not expected to be at an increased risk for heel pressure ulcers.

The model does indicate however that an atypical anatomy consisting of a heavier foot, together with sharp posterior calcaneus and thin soft tissue padding over that calcaneus theoretically imposes high risk (Fig. 2a). Though it is currently not straight-forward to identify such patients deterministically in the clinical setting, it is possible and practical to observe the feet at admission, and use palpation to assess whether the posterior calcaneus is particularly sharp or whether soft tissue padding over the posterior calcaneus appears especially thin. Pending additional studies as well as clinical validation, ultrasound scanning has the potential to become a conventional tool for estimating calcaneal curvature and soft tissue thickness in a quantitative manner [22]. Using ultrasound for this purpose is particularly convenient since Doppler measurements of blood flow at the heel tissues can be included in such an examination as well [23]. Establishing routine ultrasound scans of the heels might also lead to development of new ultrasound-based risk assessment tools that are more specific for identifying susceptibility to heel ulcers, particularly considering the individual's internal foot anatomy.

Diabetes

Of the multiple intrinsic risk factors for heel ulcers which are imposed by diabetes, neuropathy and inadequate tissue perfusion are the most well-known [12,14]. However, there could be additional risk factors that relate to the pathologically altered biomechanics of the diabetic foot, as diabetes might involve changes to the extents of loading in the soft tissues of the posterior heels, as well as to the soft tissue mechanical properties and sometimes to their geometry. First, diabetes is known to be associated with obesity, hence feet of diabetic patients might be heaver than normal. Second, the hyperglycemia gradually induces thickening and interlinking of collagen fibers in connective tissues, particularly in the soft tissues of the feet, which leads to progressive stiffening of these tissues that in turn, decreases their ability to spread and attenuate mechanical loads through tissue deformation [24,25]. Third, soft tissue displacements (migration) and atrophies have also been documented in the diabetic foot [26], which means that the soft tissue layer overlying the posterior calcaneus might be not only pathologically stiffer, but also pathologically thinner. The effects of these biomechanical factors on internal soft tissue pressures in the posterior heel, p_{max},

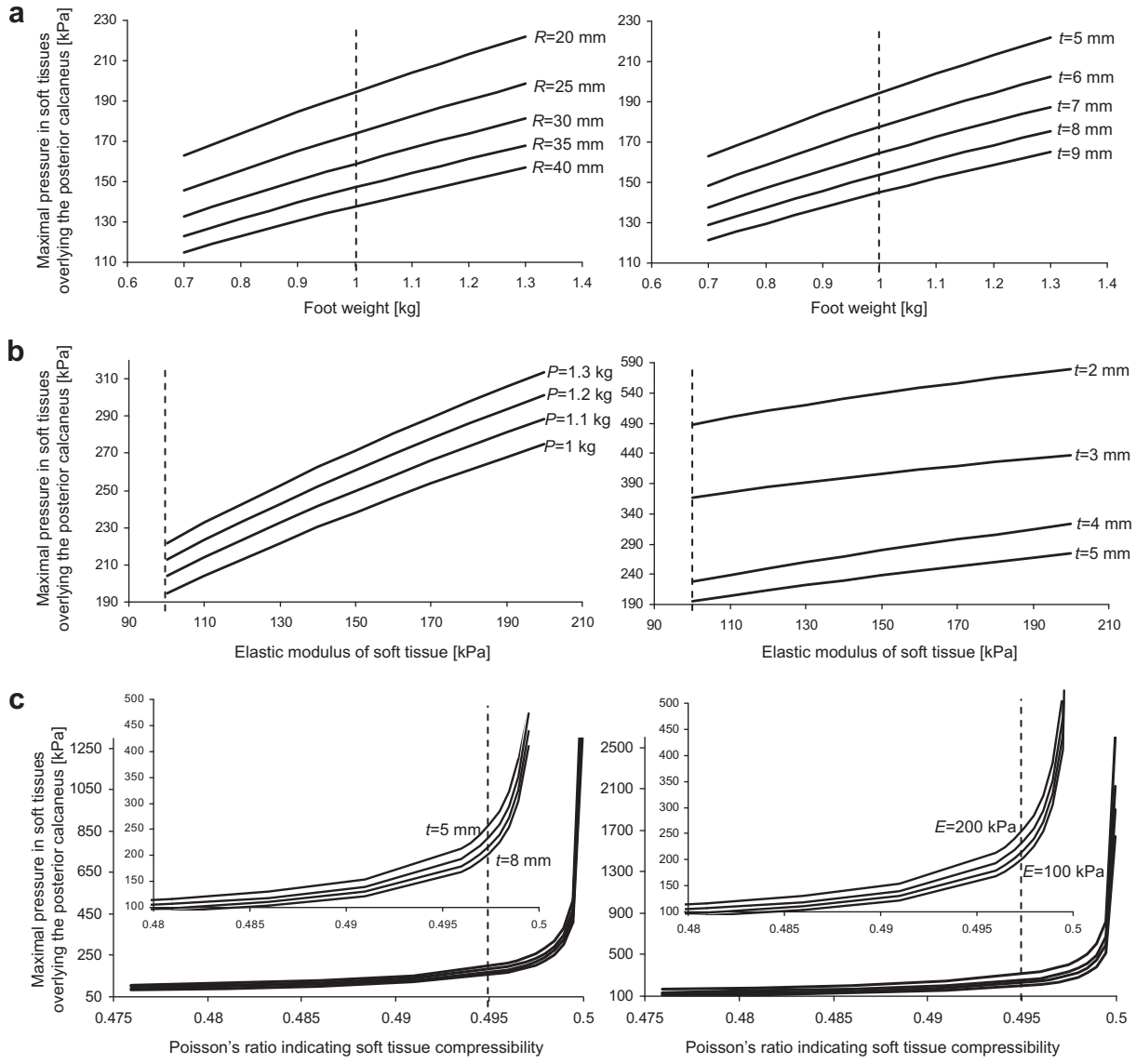

Figure 2 Model simulation data of maximal internal pressures in the soft tissues overlying the posterior heel, in regard to (a) foot and heel anatomy, (b) diabetes, and (c) edema and dehydration of the soft tissues of the heel.

can be studied using the present model (Eq. (4)). Specifically, the model was tested for increasing values of the elastic modulus of the soft tissue (E), to simulate tissue stiffening due to hyperglycemia-induced collagen changes. The influence of changes in E on p_{max} was also analyzed in combination with increased foot weight associated with obesity (P), or in combination with decreased soft tissue thickness at the posterior heel, caused by tissue migration (t). Analysis of the model data concerning diabetes (Fig. 2b) clearly demonstrated that from a biomechanical theory perspective, diabetes imposes a substantial risk for heel ulcers. Not only that an increase in soft tissue stiffness always inflicts higher p_{max} values, obesity and tissue migration from the posterior heel region will amplify this trend and increase p_{max} further (Fig. 2b). Much like as said in regard to foot anatomy, future risk assessments may employ ultrasound scanning to test — other than geometrical features (e.g. soft tissue thickness) — also soft tissue stiffness properties. Technologically, this can be done using an emerging technique called ultrasound elastography, which measures the characteristics of the ultrasound waves while they are being attenuated within the tissues, and then provides evaluations of the local tissue stiffnesses based on the velocity of the propagating ultrasound waves [27]. It is expected that within the next few years noninvasive internal soft tissue stiffness measurements will be available to clinicians as part of routine ultrasound scans, which

will allow to include these tests in pressure ulcer, and, particularly, in heel ulcer risk assessments. Hence, in the future, ultrasound scans to estimate a patient's risk for heel ulcers should ultimately include anatomical assessment, blood flow assessment (Doppler) and tissue elasticity measurements (elastography). Together, these examinations should provide a complete picture of the susceptibility of an individual to heel ulcers or other pressure ulcers.

Edema and tissue dehydration at the posterior heel

Edema at the heels can develop as a result of an evolving pressure ulcer, or, it can be associated with a chronic circulation deficiency in the venous system of the legs. In either case, fluids are then accumulated at the posterior heel tissues, and this can possibly lead to local tissue swelling and/or stiffening due to potential rise in the osmotic tissue pressures. The heel tissues may also be subjected to the opposite imbalanced fluid volume state — tissue dehydration — which typically occurs due to systemic dehydration. As in the previous cases, the model described by Eq. (4) can be used to theoretically evaluate the effects of edema or dehydration on internal soft tissue pressures at the posterior heel, p_{max}, and thereby, predict whether a risk for heel ulcers is imposed by any of these two conditions. This is achieved by changing the compressibility parameter, i.e. Poisson's ratio ν in the model. As water are completely incompressible (i.e. water do not change their volume regardless of the level of external pressure that is applied), their ν value is the highest possible based on physical and mechanical engineering theories, being 0.5. Soft tissues are a mixture of solids (e.g. proteins) and fluids, but most of their mass weight, approximately 70—80% (depending on the specific tissue type), is fluids. Hence, soft tissues normally have a ν value that is a little less than that of water, but, due to their high fluid contents, is close to ν of water, being around 0.495 (Table 1). The computer simulation data in regard to edema and dehydration, depicted in Fig. 2c, demonstrate that pressures in the posterior heel p_{max} rise sharply when the tissue compressibility parameter ν increases even slightly above the normative 0.495 level. In stark contrast, when ν decreases from 0.495, p_{max} decrease mildly (Fig. 2c). Hence, the modeling predicts that in theory, edema at the heels is a serious biomechanical risk factor for heel ulcers whereas dehydration is not. Combining the edema with an increase in tissue thickness to simulate tissue swelling (Fig. 2c; left frame) or stiffening (Fig. 2c; right frame) that might accompany the edema indicated that the effects of these factors on p_{max} were small compared to the p_{max} rise caused directly by the elevated ν. Hence, the present biomechanical model (Eq. (4)) identifies edema in the heels as being, in theory, an important risk factor for heel ulcers. This indirectly implies that venous circulatory deficiencies in the legs, heart failure and renal failure conditions, which are all known to cause edema in the legs and feet, are risk factors for heel ulcers as well, which supports and explains clinical observations and experience in this regard [12—14].

Discussion

A relatively simple biomechanical model was developed here to calculate internal soft tissue pressures at the posterior heel when the foot is supported in a supine posture (Fig. 1). The model was used to explore the theoretical effects of the internal heel anatomy (posterior calcaneus curvature and overlying soft tissue thickness), as well as the effects of diabetes, edema and dehydration on internal soft tissue pressures at the heel, and thereby, on the risk for heel ulcers. The modeling demonstrated that atypical foot anatomies characterized by heavy-weight foot, sharp posterior calcaneus and thin soft tissue padding are theoretically more prone to heel ulcers. Diabetes and edema at the feet were also predicted to impose risks for heel ulcers, which agrees very well with clinical observations [12—14]. This paper therefore demonstrated that a biomechanical theory can be used to explain and interpret clinical and epidemiological findings related to heel ulcers.

Many devices are available commercially for preventing heel ulcers, and they all use the principle of dispersing pressures between the heel and the support over a larger contact area. The modeling described herein was limited to investigations of a foot supported by a horizontal surface, without considering the nature of this surface, e.g. its geometry, friction or stiffness properties. More sophisticated modeling is still needed for evaluating the benefits of heel pressure-reducing devices, in particular the effects of their shape and mechanical properties on achieving the desired reduction of tissue pressures. Such studies can be conducted using finite element analyses [5], which are commonly used in bioengineering research. A next step in studying the etiology of

heel ulcers would therefore be to develop more sophisticated biomechanical models of the heel, which represent the full complexity of the bones and surrounding soft tissues, in terms of geometry as well as mechanical properties of tissues, and the inter-subject variations in these parameters. These could then be employed for systematic evaluations of the performances of heel pressure-reducing device designs.

Conflict of interest

None.

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