Prevention and management of pressure injury to the heel

Authors: Joyce Black, Nick Santamaria, Amit Gefen, Tod Brindle, Jacqui Fletcher and Paulo Alves Pressure injuries on the heels often occur in immobile patients. The risk factors for these injuries stem from the anatomy of the calcaneus, impairments in blood flow to the foot and neuropathic disease. There are many clinical considerations in the prevention of heel pressure injury. This article addresses the epidemiology and economic impact of heel injury, identifies risk factors and differential diagnosis of their development, discusses challenges in prevention across the continuum of care, and provides guidance for selecting appropriate interventions in the prevention of heel pressure injury through the review of existing evidence.

ressure ulcers, now termed pressure injuries (PIs) in some countries, are defined by the National Pressure Ulcer Advisory Panel as "localized damage to the skin and/or underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue" (Edsberg et al, 2016). In adults, the heel is one of the most common areas of PI development, accounting for some of the most significant and severe PIs in both European and American clinical studies (Vanderwee et al, 2007).

Prevention of heel injury is paramount. This article addresses the epidemiology and economic impact of heel injury complications, identifies risk factors and the differential diagnosis of PI development, discusses challenges in prevention across the continuum of care, and provides guidance for selecting appropriate interventions in the prevention of heel PI through a review of the existing evidence.

Epidemiology and cost of heel pressure injuries

Heel PIs are commonly reported to be the first or second most prevalent hospital-acquired PIs (Kerstein, 2002; VanGilder et al, 2008; Jenkins and O'Neal, 2010; Salcido et al, 2010; Mulligan et al, 2011), ranging in prevalence from 2.0–41.0%. The wide range of international prevalence is due, in part, to the differing methodologies used in reporting the incidence and prevalence of heel PI and to differing health payment/ reimbursement systems (Berlowitz, 2012). There is some variation in the prevalence data based on the clinical area where the study was conducted. For example, rates of heel PI vary between settings such as intensive care units (Santamaria et al, 2015), operating rooms (Shen et al, 2015), general medical/surgical wards (Gunningberg et al, 2011) and elderly care settings (Rasero et al, 2015; Ahn et al, 2016).

Even though there are few data specifically reporting on the cost of heel PIs, from the prevalence in the literature it is clear that heel PIs make up a very large proportion of all hospital-acquired PIs, therefore, it is logical that they would also make up a large proportion of costs. It has been estimated that PI costs \$11bn (£7.8 bn) in the United States (Russo et al, 2008), AU\$3.5 billion (£1.9 billion) in Australia (Graves et al, 2005) and £531 million in the United Kingdom (Guest et al, 2017).

When considering the cost of heel Pls, clinicians should think beyond the direct costs of care, i.e. the financial cost to the hospital or facility. There are also costs associated with increased length of stay and decreased efficiency of the clinical unit due to decreased patient throughput as well as opportunity costs incurred through staff time spent caring for the injury rather than undertaking other activities. Importantly, there are personal costs to the patient in the form of pain, discomfort,

of Nursing, University of Nebraska Medical Center, Omaha, Nebraska, United States; Nick Santamaria is Professor of Nursing Research, University of Melbourne, Australia; Amit Gefen is Professor of Biomedical Engineering and the Herbert J. Berman Chair in Vascular Bioengineering, Department of Biomedical Engineering, Faculty of Engineering, Tel Aviv University, Tel Aviv, Israel; Tod Brindle is Doctoral Candidate, Virginia Commonwealth University, Richmond, Virginia, USA; Jacaui Fletcher is Independent Nurse Consultant, UK; Paulo Alves is Assistant Professor, Centre for Interdisciplinary Research in Health (CIIS), Universidade Católica Portuguesa, Porto, Portugal

Joyce Black is Professor, College

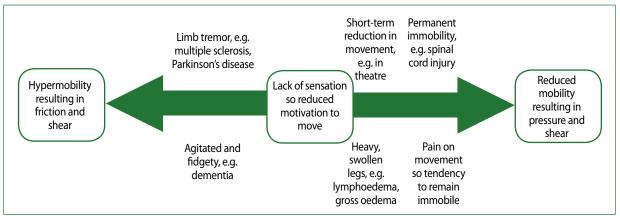


Figure 1. Factors that put heels at an increased risk of pressure injury.

limitations in mobility, decreased quality of life and — in the case of the high-risk patient with diabetes — the potential for osteomyelitis, amputation or even death (Kerstein, 2002; Orneholm et al, 2017).

The aetiology of pressure injury

There are three mechanisms at the cellular level that lead to PI:

- Direct deformation injury of soft tissue cells, which irreversibly damages the cell membrane and cytoskeleton (supporting structure of the cell)
- Pressure and shear applied to soft tissues over time resulting in ischaemia, inflammation and possibly cell death (Oomens et al, 2015). Tissue injury from pressure, shear and ischaemia leads to ischaemia-reperfusion injury over time. The effects of reperfusion worsen damage in the 2–5 days after circulation has returned (Xiao et al, 2014; Hammers et al, 2015; Wilson et al, 2015)
- Changes in the microclimate of the skin due to the accumulation of moisture and heat, which increases the metabolic demand on cells and weakens the intercellular connections reducing the tolerance of

the superficial skin structures to pressure and shear.

Various factors lead to PI of the heels [Figure 1]. PI of the heel is primarily due to external pressure. Regardless of the position of the leg on the bed, the angular calcaneus is exposed to pressure, so posterior, medial and lateral pressure injuries are possible. When the pressure is intense, such as when the heel is resting on a hard surface (e.g. the operating or radiology table) or when there is inadequate contact with the footrest of a wheelchair, strain causes the cells to deform and then rupture (Luboz et al, 2015), gradually losing their structural integrity and, in some cases, leading to deep tissue PI [Figure 2a]. When the pressure is less intense, but prolonged, as seen in bedbound patients, the tissues become obstructed or ischaemic due to the collapse of blood vessels that provide nutrients, often leading to classic stage 1 PI [Figure 2b]. When the patient slides down in bed, shear forces injure soft tissue by stretching the blood vessels, distorting cell structures and also creating ischaemia. Shear forces often occur in deeper soft tissue, in the fat pad of the heel, and can lead to blood blisters [Figure 2c]. In the heel, shear combined with



Figure 2. (a) Deep tissue pressure injury due to intense pressure; (b) stage 1 pressure injury due to moderate, prolonged pressure; (c) blood blister due to shear forces; and (d) fluid-filled blister due to shear and friction forces.

friction increases the temperature of the tissue, leading to blistering. Fluid-filled blisters are often the result of friction damage from rubbing and may later become infected. Agitated patients and those with spasticity of the limbs or muscle spasm often rub or dig their heels into the bed, which worsens the biomechanical conditions and exposes the tissue to friction and shear forces [*Figure 2d*].

All PIs are caused by pressure and shear. While the physics of soft tissue injury from pressure and shear is straightforward, not every patient exposed to the same amount of pressure develops a PI. The extent of damage is dependent on the tolerance of the soft tissue for pressure and shear. Factors that reduce the tolerance of the heel for these forces are the anatomy of the calcaneus, perfusion to the leg and changes in sensation in the leg from conditions such as diabetes or paralysis (Dobos et al, 2015).

The angular anatomy of the calcaneus and the thinness of the fat pad create areas of high pressure. Physiological factors, including reduced perfusion from underlying arterial inflow disease and neuropathic disease, decrease the ability to reperfuse ischaemic tissue. The calcaneal branches of the posterior tibial and peroneal arteries that supply the heel tissue with blood often cannot provide adequate perfusion when pressure is applied. When patients have peripheral vascular disease and diabetes, perfusion is further impaired due to changes in both the large and small vessels that cannot always be corrected through revascularisation (Thiruvoipati et al, 2015). In addition, smoking which causes vasoconstriction and accelerates vascular disease — is a red flag for patients at risk of developing heel PI.

Pain is a useful indicator of tissue damage. However, not all patients can report pain. In individuals with spinal injury, peripheral neuropathy or who are sedated, the patient either does not or cannot reposition his or her leg, thus increasing the risk of Pl. Any form of neuropathy halts sensory signals from the foot to the brain that indicate tissue ischaemia, which would normally promote movement. Motor neuropathy prevents the patient from voluntarily moving the foot to restore blood flow. Diseases that impair leg sensation, e.g. diabetic neuropathy, or movement, e.g. stroke, increase the risk of Pl.

Risk factors for heel pressure injury Immobility

The primary risk factor for heel PI is leg

immobility. The patient should be assessed as to whether he/she can move his/her legs and determine whether he/she does move the legs. If the patient cannot voluntarily move the legs due to paralysis, weakness or unconsciousness, the heel is at risk of PI. Legs that do not move are subjected to intense pressures on the portion of the heel that is in contact with the bed.

A common patient with immobile legs seen by clinicians is the patient with a fractured hip. These patients are at increased risk for heel ulcers due to pain, spasms and leg immobility (Black, 2012). Exposure of the heel to pressure can begin in or during transport to the emergency room (Muntlin Athlin et al, 2016).

Impairments in perfusion

Patients with impairments in perfusion are at higher risk for heel PI due to delayed reperfusion following any ischaemia. These patients can be identified by a history of peripheral vascular disease, routine use of anticoagulant medications, prior surgery for revascularisation of the legs, thick toenails, hairless legs, shiny skin on the legs and weak to absent pedal pulses. If the pedal pulse cannot be palpated, assess for the posterior tibial pulse and/or assess perfusion with a Doppler.

The use of antiembolic and compression stockings can also result in constriction of the blood flow and damage to the heel. Sometimes the heel is injured because:

- The stocking is too tight or has become too tight since it was first applied due to oedema
- It was applied incorrectly
- It was rolled down by the patient.

Neuropathic disease

Neuropathic disease also increases the risk of heel PI. The presence of diabetic neuropathy should be evaluated in all patients with diabetes, especially those who have had the condition for over 5 years. Assess the foot for protective sensation, not sharp or dull pain (Delmore et al, 2015). Patients with diabetes also commonly have peripheral vascular disease.

Age-related, disuse-related and diabetes-related soft tissue changes

From a biomechanical perspective, the posterior heel is a small compartment with sharp transition between the rigid, highly-curved bony surface of the calcaneus to the thin layers of connective soft tissues. Under weight-bearing, these structural features cause severe internal distortions and shearing in the soft tissues. The skin over the posterior heel is typically relatively

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Figure 3. The heel pressure ulcer on this patient's foot is not visible when her leg is at rest (a). Only by examining the entire foot is the ulcer found (b).

thin, but in older people it may be particularly fragile due to age-related tissue deterioration. Likewise, capillary density in connective tissues is reduced with old age, and so is the overall mass of soft tissue at the posterior heel.

The anchoring between the skin layers, specifically, the micro-anatomical interlocking at the epidermal-dermal junctions, is compromised in older people, and such changes are also characteristic to disuse conditions, e.g. chronic bed confinement. Finally, in diabetes, there are pathological changes in mechanical behaviour of connective tissues that relate to the increase in diameters and fusion of collagen fibres, which ultimately make tissues stiffer and less able to distribute bodyweight forces through effective deformations at non-damaging levels. The result of progression of the disease to the stage where it influence connective tissue structures is that soft tissue deformations become very localised and intensive which, in engineering terms, is called 'stress concentrations' and would lead to earlier cell and tissue death (Gefen, 2017).

Examination and assessment

Any devices or stockings that are on the leg should be removed. The skin of the foot should then be examined visually, using a mirror if needed to see the posterior heel and insertion site of the Achilles tendon into the calcaneus. Clinicians should not assume that the skin of the heel is intact if it cannot be seen. If the patient's foot is externally rotated at rest, the medial and lateral aspects of the foot should also be inspected [*Figure 3a and 3b*]. The patient should be assessed for dry skin of the legs and conditions that increase shear and friction, such as agitation or leg spasms. Risk factors, such as cigarette smoking, peripheral vascular disease, arterial disease and diabetes, warrant heightened awareness.

If a formal risk assessment tool is being used to determine risk for PI, it should be borne in mind that risk factors that stem from impairments in perfusion or sensation may not be identified. Clinical judgement should be used to determine whether the patient is at risk for PI by considering all of the risk factors.

Offloading the heel

The geometry, anatomy and perfusion of the heel create challenges in preventing heel Pl. International guidelines suggest that in order to redistribute pressure from the heel, the heel should be floated from the bed (Haesler, 2014). However, floating the heel on pillows does not always work if the patient moves about in bed or the pillow collapses from the weight of the leg. In addition, floating the heels will always result in greater weight-bearing in other anatomical areas and may shift the risk for injury to elsewhere in the body.

When determining which mechanism to use to offload the heel, consider:

- How long the legs will be immobile
- How mobile the patient's legs are
- Whether the patient is agitated and moving a lot
- The presence of poor arterial flow
- The presence of neuropathy
- Whether the patient is ambulatory
- Whether the patient slides down the bed.

Duration of immobility

Use clinical judgment to determine whether the patient will need the heels to be "floated" for a few hours as he or she recovers from a procedure or whether the patient will need more advanced options that support the foot in a neutral position as well. If pillows are chosen for offloading, place them under the calf of the leg, allowing the heel to be suspended off the bed.

The legs will need frequent assessment because patients often kick the pillows off the bed and the filling in many pillows collapses



Figure 4. Powered bed frames cause patients to slide down the bed to the footboard, subjecting the heel to high pressure and causing occlusion of the vessels.

after a short time, allowing the heel to rest on the bed. If pillows need to be rolled up to support the neck for a patient, they are often too thin to offload the heel from the bed.

Using multilayer silicone foam dressings on the heel has been shown to significantly reduce the risk of heel PI. Santamaria et al (2015) placed heel dressings on 150 patients in the emergency department who were going to be transferred to the intensive care unit and compared the incidence of heel PI in this group to a prior group of 151 patients without the dressing. None of the patients with heel dressings developed a heel PI, whereas 14 (9.2%) of the control group did ulcerate.

Care should be taken to ensure the dressing is the correct size and shape and that it is appropriately placed to reduce the risk of wrinkling or lifting at the edges. When used prophylactically, the dressing should be peeled back at least once per day to examine the condition of the skin.

Leg mobility

A couple of quick questions should be asked by clinicians; can the patient lift and move their legs? Does the patient lift and move the legs? If not, dressings or a boot style heel-offloading device may be the best option for this patient.

Well-designed boots have been shown to reduce both heel PI and plantar foot contraction (Meyers, 2017). Heel boots vary in their constituent materials and construction. Some offer better visibility of the heel than others and some materials can be hot and uncomfortable.

Some boots come in different sizes. If only one size is available, it may be difficult to use on large or swollen limbs. Exercise caution when securing boots on oedematous limbs in order to prevent further skin damage.

Heel boots can be warm or cumbersome to wear. If the period of immobility is short, such as during surgery, silicone heel cups may work. However, they are not secured to the heels so they can come loose in the bed and may easily be discarded with the linen when the bed is changed.

In the authors' opinion, the existing evidence suggests that heel protection devices vary in their ability to protect against Pl. There is currently no guidance provided on which type of device may be appropriate for any given patient. When selecting a device, the factors discussed in the following subsections should be considered.

Hypermobility

Hypermobility must be considered during device selection because it can cause significant damage. Heel dressings that adhere to the skin to protect it from friction and shear are preferred. Friction-reducing bootees may be suitable for patients with hypermobility, but do not offer any pressure reduction, may not remain on the heel and — if the patient is ambulatory — also increase the risk of falls.

Poor arterial flow

A dressing should be considered if the patient cannot tolerate having the leg elevated. A dressing that can be removed without injury to the skin should be selected.

If a boot is used, one with additional padding to protect the limb and keep it warm should be chosen. The limb should be examined during each shift to identify any early signs of injury from the boot and, of course, examine the heel for possible PI.

Neuropathy

In patients with neuropathy, a heel-offloading device that does not contain hard plastic or metal components should be considered. These components can cause injury and the patient will not be aware of the damage.

Unfortunately, the very devices used to prevent heel Pl can lead to injury themselves. The heel-offloading device must fit correctly, and any tubes (e.g. sequential compression device inflation tubing) that are present should not be pinching the skin. The device

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Figure 5. Summary of risk factors for heel pressure injury (Welsh Wound Innovation and Direct Healthcare Services, 2018).



Don't leave it until LATER

Look. Ask. Test. Educate. Refer

Prevention of heel ulcers



should be removed during each shift to examine the skin.

Using the paper test to ensure the heels are floated
Articulating the bed frame correctly
Ensuring the feet are not pressed against the bed end

Keeping the skin clean and well hydrated

Ambulation

In ambulant patients, a dressing that can remain on the foot when shoes are worn should be considered. If boots are used, they must be removed before ambulation to reduce trip hazards. If the patient sits in a chair, it should be remembered that the heels can be subjected to pressure while resting on the footstool.

Sliding down the bed

Powered bed frames (also called profiling beds) can cause patients to slide down the bed to the footboard. The heel is subjected to pressure and shear during these movements and the foot may be pressed against the footboard of the bed, resulting in high pressures and occlusion of the vessels (Hermans and Call, 2014; Fletcher, 2015) [Figure 4].

The multiple risk factors for heel PI have been summarised in a document for staff nurses called "Don't Leave it until LATER". This document, produced by Direct Healthcare Services and Welsh Wound Innovation (2018), urges nurses to Look, Ask, Test, Educate and Refer patients [Figure 5].

Cost-benefit of heel offloading devices The cost of offloading options varies considerably; however, unit cost can be misleading as a number of devices are reusable and some can even be used to treat multiple patients.

The cost of the device must also be weighed against the cost of developing a Pl. Santamaria et al (2015) demonstrated that the average net cost of their intervention with a foam dressing was lower than that of the control — AU\$70.82 (£38.76) versus AU\$144.56 (£79.13) — and concluded that the use of soft silicone multilayered foam dressings to prevent heel Pls among critically ill patients results in cost savings in the acute care hospital.

Conclusion

PI on the heel can be devastating. Clinicians should ensure they can recognise patients at risk of PI development and take appropriate actions to reduce or remove pressure on this vulnerable area.

References

- Ahn H, Cowan L, Garvan C et al (2016) Risk factors for pressure ulcers including suspected deep tissue injury in nursing home facility residents: analysis of national Minimum Data Set 3.0. *Adv Skin Wound Care* 29(4): 178–90
- Berlowitz D (2012) Prevalence, incidence and facility acquired rates. In: Pieper B, National Pressure Ulcer Advisory Panel, eds, *Pressure Ulcers: Prevalence, Incidence and Implications for the Future*. NPUAP, Washington

Black J (2012) Pressure ulcers in patients with hip

fractures. In: Pieper B, National Pressure Ulcer Advisory Panel, eds, *Pressure Ulcers: Prevalence, Incidence and Implications for the Future*. NPUAP, Washington: 129–40

- Delmore B, Lebovits S, Suggs B et al (2015) Risk factors associated with heel pressure ulcers in hospitalized patients. *J Wound Ostomy Continence Nurs* 42(3): 242–8
- Dobos G, Gefen A, Blume-Peytavi U, Kottner J (2015) Weight-bearing-induced changes in the microtopography and structural stiffness of human skin in vivo following immobility periods. *Wound Repair Regen* 23(1): 37–43
- Edsberg LE, Black JM, Goldberg M et al (2016) Revised National Pressure Ulcer Advisory Panel pressure injury staging system. *J Wound Ostomy Continence Nurs* 43(6): 585–97
- Fletcher J (2015) Articulated bed frames and heel ulcer prevalence. *Wound Essentials* 10(1): 8–13
- Graves N, Birrell F, Whitby M (2005) Modelling the economic losses from pressure ulcers among hospitalised Australians. *Wound Repair Regen* 13(5): 462–7
- Guest JF, Ayoub N, McIlwraith T et al (2017) Health economic burden that different wound types impose on the UK's National Health Service. *Int Wound J* 14(2): 322–30
- Gunningberg L, Stotts NA, Idvall E.(2011) Hospitalacquired pressure ulcers in two Swedish County Councils: cross-sectional data as the foundation for future quality improvement. *Int Wound J* 8(5): 465–73
- Haesler E (2014) National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel, Pan Pacific Pressure Injury Alliance: Guidelines for the Prevention and Treatment of Pressure Ulcers. Cambridge Media, Perth, Australia
- Hammers DW, Rybalko V, Merscham-Banda M et al (2015) Anti-inflammatory macrophages improve skeletal muscle recovery from ischemia-reperfusion. *J Appl Physiol* 118(8): 1067–74
- Hermans MH, Call E (2015) Failure to reposition after sliding down in bed increases pressure at the sacrum and heels. *Wounds* 27(7): 191–8
- Jenkins ML, O'Neal E (2010) Pressure ulcer prevalence and incidence in acute care. *Adv Skin Wound Care* 23(12): 556–9
- Kerstein MD (2002) Heel ulceration in the diabetic patient. *Wounds* 14(6): 212–6
- Luboz V, Perrier A, Bucki M et al (2015) Influence of the calcaneus shape on the risk of posterior heel ulcer using 3D patient-specific biomechanical modeling. *Annals Biomed Eng* 43(2): 325–35
- Meyers T (2017) Prevention of heel pressure injuries and plantar flexion contractures with use of a heel protector in high-risk neurotrauma, medical, and surgical intensive care units: a randomized controlled trial. J Wound Ostomy Continence Nurs 44(5): 429–33

- Mulligan S, Prentice J, Scott L (2011) WoundsWest Wound Prevalence Survey. 2011 State-wide Overview Report. Ambulatory Care Services, Department of Health. Perth, Australia
- Muntlin Athlin Å, Engström M, Gunningberg L, Bååth C (2016) Heel pressure ulcer, prevention and predictors during the care delivery chain – when and where to take action? A descriptive and explorative study. *Scand J Trauma Resusc Emerg Med* 24(1): 134
- Oomens CWJ, Bader DL, Loerakker S, Baaijens F (2015) Pressure induced deep tissue injury explained. *Ann Biomed Eng* 43(2): 297–305
- Örneholm H, Apelqvist J, Larsson L, Eneroth M (2016) Heel ulcers do heal in patients with diabetes. *Int Wound J* 14(4): 629–35
- Rasero L, Simonetti M, Falcian F et al (2015) Pressure ulcers in older adults: a prevalence study. *Adv Skin Wound Care* 28(10): 461–4
- Russo CA, Steiner C, Spector W (2008) Hospitalizations Related to Pressure Ulcers Among Adults 18 Years and Older, 2006: Statistical Brief #64. Healthcare Cost and Utilization Project (HCUP) Statistical Briefs [Internet]. Agency for Healthcare Research and Quality, Rockville (MD), US
- Salcideo R, Lee A, Ahn C (2010) Purple heel and deep tissue injury. Adv Skin Wound Care 24(8): 374–80
- Santamaria N, Gerdtz M, Liu W et al (2015) Clinical effectiveness of a silicone foam dressing for the prevention of heel pressure ulcers in critically ill patients: Border II Trial. *J Wound Care* 24(8): 340–5
- Shen WQ, Chen HL, Xu YH et al (2015) The relationship between length of surgery and the incidence of pressure ulcers in cardiovascular surgical patients: a retrospective study. *Adv Skin Wound Care* 28(10): 444–50
- Thiruvoipati T, Kielhorn CE, Armstrong EJ (2015) Peripheral artery disease in patients with diabetes: Epidemiology, mechanisms, and outcomes. *World J Diabetes* 6(7): 961–9
- VanGilder C, Macfarlane GD, Meyer S (2008) Results of nine international pressure ulcer prevalence surveys: 1989 to 2005. Ostomy Wound Manage 54(2): 40–54
- Vanderwee K, Clark M, Dealey C et al (2007) Pressure ulcer prevalence in Europe: a pilot study. *J Eval Clin Pract* 13(2): 227–35
- Wilson HMP, Welikson RE, Luo J et al (2015) Can cytoprotective cobalt protoporphyrin protect skeletal muscle and muscle-derived stem cells from ischemic injury? *Clin Orthopaed Related Res* 473(9): 2908–19
- Welsh Wound Innovation, Direct Healthcare Services (2018) *Don't leave it until LATER*. Available at: https:// bit.ly/2uLJkVG (accessed 19.04.2018)
- Xiao DZT, Wu SYQ, Mak AFT (2014) Accumulation of loading damage and unloading reperfusion injury – modeling of the propagation of deep tissue ulcers. J Biomech 47(7): 1658–64