Impulse-forces during walking are not increased in patients with knee osteoarthritis

Impulse-forces during walking are not increased in patients with knee osteoarthritis

Authors: Marius Henriksen [a]; Erik B. Simonsen [b]; Thomas Graven-Nielsen [c]; Hans Lund [a]; Bente Danneskiold-Samsøe [a]; Henning Bliddal [a]
Affiliations:
[a] The Parker Institute, Frederiksberg Hospital, Frederiksberg, Denmark

[b] Institute of Medical Anatomy, Section C, The Panum Institute, University of Copenhagen, Copenhagen N, Denmark

[c] Center for Sensory-Motor Interaction, Laboratory for Experimental Pain Research, Aalborg University, Aalborg, Denmark
DOI: 10.1080/17453670610012747
Publication Frequency: 6 issues per year

Published in: Acta Orthopaedica, Volume 77, Issue 4 August 2006 , pages 650 - 656
Previously published as: Acta Orthopaedica Scandinavica (0001-6470, 1651-1964) until 2005

Abstract

Background Impulsive forces in the knee joint have been suspected to be a co-factor in the development and progression of knee osteoarthritis. We thus evaluated the impulsive sagittal ground reaction forces (iGRF), shock waves and lower extremity joint kinematics at heel strike during walking in knee osteoarthritis (OA) patients and compared them to those in healthy subjects.

Subjects and methods We studied 9 OA patients and 10 healthy subjects using three-dimensional gait analyses concentrated on the heel strike. Impulse GRF (iGRF) was measured together with peak accelerations (PA) at the tibial tuberosity and sacrum. Sagittal lower extremity joint angles at heel strike were extracted from the gait analyses.

As OA is painful and pain might alter movement strategies, the patient group was also evaluated following pain relief by intraarticular lidocaine injections.

Results The two groups showed similar iGRF, similar tibial and sacral PA, and similar joint angles at heel strike. Following pain relief, the OA patients struck the ground with more extended hip and knee joints and lower tibial PA compared to the painful condition. Although such changes occurred after pain relief, all parameters were within their normal ranges.

Interpretation OA patients and healthy subjects show similar impulse-forces and joint kinematics at heel strike. Following pain relief in the patient group, changes in tibial PA and in hip and knee joint angles were observed but these were still within the normal range. Our findings make us question the hypothesis that impulse-forces generated at heel strike during walking contribute to progression of OA.

Introduction

Excessive impulsive forces in the knee joint have been suspected to be a co-factor in the development and progression of knee joint osteoarthritis (OA) (Simon et al. 1972, Radin et al. 1973, Gill and O'Connor 2003). This has been demonstrated in animal studies (Radin et al. 1978, 1982) and a detrimental effect of impulsive forces on the subchondral plate due to microdamage, subsequent deep layer cartilage calcification, and narrowing of the cartilage has been presented (Burr and Radin, 2003). Theoretically, these morphological changes of the cartilage and subchondral plate imply relatively increased demands on the remaining healthy cartilage and subchondral zone to absorb energy, which could thus induce a vicious circle of degeneration if not counteracted by a reduction of tissue load.

During normal walking, impulsive forces are created in the foot-ground interface at heel strike. These forces travel up the lower limb as a shock wave, also known as the heel strike transient (Collins and Whittle 1989). The human locomotor system is capable of attenuating the impulsive forces on their way up the lower limb and spine (Ratcliffe and Holt 1997). The attenuation takes place in a passive and an active system. The passive system consists of structures such as the heel pad, ligaments, joint cartilage, menisci, intervertebral discs and bone. In the active system, the impulsive forces are absorbed by lower-extremity kinematics and muscular control strategies at heel strike. Thus, impairment of the sensory-motor system due to e.g. joint pathology, pain, reduced muscle function, limitations in joint motion etc. may have unfavorable effects on attenuation of the impulsive forces caused by heel strike.

OA has been associated with both reduced muscle strength (Slemenda et al. 1997) and muscle function (Fisher and Pendergast 1997), which may cause an excessive amount mechanical loading. There are very limited human data supporting the theory of a link between OA and impulsive loads. Although there was no clinical evidence of OA, higher axial tibial accelerations at heel strike were found in a patient group with painful knee joints than in healthy controls (Radin et al. 1991). Interestingly, the patient group was pain-free at the time of data collection. Presence of pain may cause compensatory

changes in the walking pattern, which may mask the movement strategy chosen in a pain-free condition.

A link between excessive impulsive forces and knee OA remains to be demonstrated. We therefore compared the impulsive ground reaction forces at heel strike during walking, the subsequent lower extremity shock wave, and the lower extremity joint kinematics in a group of OA patients to those in a healthy control group. We also assessed whether the observed forces, shock waves and joint kinematics in the OA patient group are affected by local pain relief.

Subjects and methods

Subjects

10 patients were included in the study. All patients had unilateral medial knee joint OA diagnosed radiographically and in line with the criteria defined by the American College of Rheumatology. Average age was 68 (59-74) years, average height 164 (156-170) cm and weight 74 (60-86) kg. To be included in the study, the patients had to experience pain during walking. The patients took paracetamol (n = 7) or non-steroid anti-inflammatory drugs (NSAID) (n = 4) regularly. Analgesics were not allowed 48 h before the collection of data. Patients were excluded if they had a diagnosis of OA or other rheumatological diseases in the hip or ankle joints, if they had a history of lower extremity joint trauma or neurological disorders.

On the test day, all patients assessed their OA status using the Western Ontario McMaster Universities osteoarthritis index (WOMAC) with pain subscale (5 items), stiffness subscale (2 items), and physical function subscale (17 items). The WOMAC index is designed to assess OA disease during the previous 48 hours - in this case during the medicine washout period. It is designed as a 5-point Likert scale for each item, with high scores indicating a high degree of impairment. The total score was normalized to a score from 0-100.

10 healthy control subjects with a mean age of 61 (53-70) years, mean height of 163 (155-172) cm and weight 60 (47-79) kg were included. Subjects were excluded if they had clinical symptoms or signs of OA, knee joint pain, other rheumatological diseases in the joints of the lower extremities, previous lower extremity joint trauma, or neurological disorders.

Both patients and control subjects gave their informed consent to participate in the study, which was approved by the local ethics committee (J. No. 01-193/03).

Study design and data acquisition

We measured impulsive forces in the foot-ground interface at heel strike during walking and the subsequent shock wave in the lower extremities, together with a three-dimensional (3D) kinematic gait analysis in both the OA group and the healthy control group. The OA group underwent an additional series of identical measurements immediately after local knee joint analgesia.

The subjects were instructed to walk barefoot at a speed of 4.0 km/h (1.1 m/s). The speed was measured by photocells. The subjects practiced the desired walking speed in several tests before the actual measurements.

Impulsive forces at the foot-ground interface

Subjects walked across two force platforms (OR6-5-1; AMTI, Watertown, MA) that were mounted in a concrete floor in the basement of the building. The platforms measured 3D ground reaction forces (GRF) and had natural frequencies in vertical, anterior-posterior and mediolateral directions of 515 Hz, 350 Hz and 340 Hz, respectively. To ensure that the force platforms were sensitive to the impulsive forces generated at heel strike, a series of ground reaction forces during walking were analyzed for frequency power spectrum densities. The tests showed that 90% of the frequency spectrum was contained between 0 and 28 Hz and 99% between 0 and 42 Hz, and a sampling frequency of 1000 Hz was chosen (Gill and O'Connor 2003). The analog output signal from the force platforms was sampled using an analog-to-digital converter (Ariel Dynamics, Trabuco Canyon, CA) and specialized computer software (APAS XP Analogue software module, Ariel Dynamics). The force vectors from the anteroposterior and vertical directions were combined to yield the GRF magnitude in the sagittal plane. The impulsive ground reaction force (iGRF) at heel strike was defined as the heel strike transient peak, seen in the sagittal plane GRF between 10 and 20 m following heel strike (Figure 1). The iGRF was normalized to percentage of body mass and expressed in N/kg ×100. All calculations were performed in MATLAB 6.5 for Windows.

[Enlarge Image]

Figure 1. **Representative sample of sagittal plane ground reaction force in the first 45 ms of the stance phase of walking at 4 km/h. Impulsive ground reaction force (iGRF) was measured as the transient heel strike peak shown by the arrow.**

Assessment of the shock wave

We measured linear accelerations in units of gravity (*g*) at the tibial tuberosity and at the sacrum (S2 level) using lightweight miniature triaxial accelerometers (ADXL210E; Analogue Devices, MA, USA) that had a ± 10-*g* range. While a single uniaxial sensor has a mass below 1 gram and a natural frequency of 10 kHz, the encased triaxial accelerometric device has a mass of 12 grams and tests on between 75-100 Hz, which is similar to previous reports (Shorten and Winslow 1992). The tibial accelerometer was mounted with the vertical sensing axis aligned to the longitudinal axis of the tibia. The sacral accelerometer was mounted on the skin at S2 level, with the vertical sensing axis aligned to the craniocaudal axis of the sacrum. Elastic Velcro straps, tightened to the subjects' limit of tolerance, were used to reduce resonance vibrations from the skin and soft tissue. The acceleration signals were recorded at 250 Hz in a portable data logger (ME3000P8; Mega Electronics, Finland) with 14bit resolution. The acceleration data were downloaded to a computer by means of specialized software (MegaWin 2.3; Mega Electronics) and the digital acceleration signals were exported to MATLAB 6.5 for Windows and low-pass filtered digitally using a zero-phase lag fourth-order Butterworth

filter (second order forward and second order backward filtering, yielding a fourth-order zero-phase lag filter) with a cut-off frequency of 50 Hz. Previous investigations of skin-mounted accelerometers have shown that the heel strike during walking usually contains frequency components in the range 25-30 Hz (Wosk and Voloshin 1981) and skin-mounted accelerometers have proven to be a suitable tool to ensure accuracy in the study of mechanical signals resulting from heel strikes (Kim et al. 1993). The gravitational acceleration was removed from the filtered tibial acceleration signal by calculating the 3D inclination of the tibia with respect to the horizontal plane using Euler angles obtained from the 3D kinematic gait analysis. The filtered sacral acceleration signal was corrected for the gravitational acceleration component using an algorithm that has been described previously (Moe-Nilssen 1998). Thus, dynamic accelerations along the longitudinal axis of the tibia and the craniocaudal axis of the sacrum were obtained. To asses the propagation of the shock wave caused by heel strike, peak accelerations of the corrected tibia and sacrum acceleration signals in a 100-ms window following heel strike were extracted to represent the magnitude of the shock wave just distal to the knee and at the proximal end of the lower extremity, respectively. All filtering and post-processing of the acceleration signals were performed in MATLAB 6.5 for Windows.

Joint kinematics

15 reflective markers were placed on the subjects according to an established marker set-up (Vaughan et al. 1999). 5 digital video cameras operating at 50 Hz were used to record the movements using the Ariel Performance Analysis System (APAS XP CapDV Software Module; Ariel Dynamics) and the video sequences were digitized (APAS XP Digitize software module; Ariel Dynamics), stored on a PC and the 3D coordinates were reconstructed by direct linear transformation (APAS XP Transform software module; Ariel Dynamics). Transient accelerations of the lower limbs can be evaluated from kinematic data, but there was significant low-frequency noise corrupting the marker position data, and therefore accelerometers were applied to evaluate the shock wave. Thus, the kinematic analysis was only used to describe joint angles at heel strike and tibial inclinations for use in the acceleration signal analysis. To reduce the noise in the marker position data, the 3D marker coordinates were low-pass filtered digitally by a zero-phase lag fourth-order Butterworth filter (second order forward and second order backward filter, yielding a fourth-order zero-phase lag filter) with a cut-off frequency of 6 Hz. The filtering was done using the Ariel Performance Analysis System (APAS XP Filter software module, Ariel Dynamics). From the filtered 3D marker coordinates, we calculated the angular positions of the ankle, knee and hip in the sagittal plane at the instant of heel strike joints of the lower extremities from the marker positions as previously described (Vaughan et al. 1999). As only single joint positions at a well-defined time (heel strike) were extracted, the filter properties would not affect the results, which could be expected if transient acceleration waves were to be calculated from a time-series of kinematic data. Kinematic analyses were performed using MATLAB 6.5 for Windows.

Synchronization and normalization

Ground reaction data, acceleration signals and video were synchronized when the subject interrupted a set of photocells. The photocells triggered a radio-transmitted signal that put a mark in the acceleration data recorded on the portable data logger. 5 acceptable trials were captured, with an acceptable trial being defined as a trial with a walking speed of 4.0 (± 0.05) km/h. Joint angles were time-normalized to a 500-point time scale representing 0-100% stance phase with 0.2% increments, averaged across trials for each subject, and joint angles at heel strike (first value) were extracted from the ensemble averages. iGRF and peak acceleration values were extracted from each accepted trial and averaged across trials for each subject.

Pain assessment and knee joint analgesia

Knee joint analgesia was induced by injecting 10 mL lidocaine (1%) into the suprapatellar bursa. The injections were guided by ultrasound. Control subjects did not receive knee joint analgesia.

The OA patients were asked to register their perceived knee joint pain during the tests on a 100mm visual analog scale (VAS) with endpoints of 0 mm ("no pain") and 100 mm ("worst imaginable pain"). Pain registration was done just after the initial measurement (before injection) and again after the last measurement (after injection).

Statistics

An unpaired t-test was used to compare patients to healthy controls. Paired t-tests were used to compare the pre- and post-injection conditions in the patient group. The level of significance was set to 5%.

Results

1 patient was excluded due to misplacement of the injection outside the joint, and the data from the patient group is therefore based on 9 patients.

Assessment of patient disease

In the patient group, the average normalized WOMAC scores were 40 (SD 13) for pain, 41 (SD 15) for stiffness, 39 (SD 13) for physical function and 39 (SD 13) for total WOMAC. The mean knee joint pain during walking in the patient group was 37 mm (SD 28). Following lidocaine injections, the mean knee joint pain decreased to 1.8 mm (SD 2.0) ($p = 0.005$).

Impulsive forces and the shock wave

Representative samples of iGRF and acceleration signals are shown in Figures 1 and 2. The iGRF was not significantly different between the OA and control groups, and the lidocaine injections did not affect the iGRF in the OA group (Table).

[Enlarge Image]

Figure 2. **Example of tibial (bottom) and sacral (top) accelerations during the initial 400 ms of stance. Peak values (indicated by arrows) were extracted as a measure of the acceleration wave magnitude at below-knee and pelvis levels, respectively.**

Average (SD) measures of pain, impulsive ground reaction forces (iGRF), shock waves and joint kinematics at heel strike in the healthy control group (n = 10) and in the OA group (n = 9) with and without knee-joint analgesia (intraarticular lidocaine injections)

Healthy

OA

No analgesia

Analgesia

[a] significant difference between pain conditions in the OA group ($p < 0.01$).

[b] significant difference between pain conditions in the OA group ($p < 0.05$).

Pain (mm)			
N/A	37 (28)	1.8 (2.0) [a]	
iGRF (N/kg × 100)			
514 (185)	485 (125)	464 (106)	
Shock wave			
Tibia (g)			
1.91 (0.64)	2.00 (0.59)	1.76 (0.59) [a]	
Sacrum (g)			
0.54 (0.14)	0.52 (0.13)	0.50 (0.11)	
Sagittal joint angles (degrees)			
Ankle (dorsiflexion)			
2.7 (4.0)	5.4 (4.6)	3.5 (4.4)	
Knee (flexion)			
5.6 (2.7)	8.4 (4.3)	4.0 (5.0) [a]	
Hip (flexion)			

27 (8.7)
27 (3.9)
24 (5.2) [b]
In the patient group, the mean tibial peak acceleration was 2.00 g (SD 0.59), while the mean tibial peak acceleration in the healthy control group was 1.91 (SD 0.64). The mean difference was not statistically significant (p = 0.7). Following pain relief in the patient group, the mean tibial peak acceleration decreased as compared to before joint analgesia (p = 0.003) (Table).

The group mean sacral peak acceleration was not significantly different between the patient group and the healthy controls. Following lidocaine injections, no changes in the mean sacral peak accelerations were observed.

Lower extremity joint angles at heel strike

The mean ankle joint angle at heel strike was 5.4° (SD 4.6) and 2.7° (SD 4.0) dorsiflexion for the patient group and healthy controls, respectively. The mean group difference was not statistically significant. After relief of knee pain, the patient group ankle joint angle at heel strike was not significantly different (Table).

Knee joint angles in the patient and healthy control groups were 8.4° (SD 4.3) and 5.6° (SD 2.7) flexion, respectively, and the difference between groups was not statistically significant. After lidocaine injections, the mean knee joint angle at heel strike for the OA group was 4.0° (SD 5.0) flexion, and the mean paired difference between pain conditions was 4.4° (SD 2.3), i.e. there was less knee joint flexion at heel strike. The difference between pain and pain-free conditions was statistically significant (p < 0.001).

Mean hip joint angles at heel strike were 27° (SD 3.9) flexion for the patient group and 27° (SD 8.7) flexion for the healthy controls. Knee joint pain relief in the patient group resulted in a mean hip joint angle at heel strike of 24° (SD 5.2) flexion, i.e. less hip joint flexion, which was statistically significant (p = 0.04).

Discussion

We found that impulsive ground reaction forces and peaks in the shock wave propagating up through the body were similar in OA patients and healthy controls. Analgesia in the OA group caused only subtle changes in the joint kinematics and shock wave, although iGRF was not affected.

The design of our study was cross-sectional, and therefore no conclusions about the role of impulsive forces during walking in the development of OA can be made. However, to the authors' knowledge, this hypothesis has not been tested on OA patients before; and based on the present results, the hypothesis needs to be revised.

A study of impact forces during stepping 4.5 cm down onto a force platform showed a positive correlation between age and impact force, indicating that the irreversible neurological decline with age affects motor strategies during locomotion (Robbins et al. 2001). Although the stepping task is dissimilar to walking, the hypothesis that a life-long

exposure to excessive heel strike forces can contribute to the development of OA cannot be rejected. However, as the patient group in our study did not differ from a healthy control group of similar age, the causal connection seems weak and longitudinal studies are needed.

The lack of differences between patients and healthy subjects in impulse-forces could be attributed to the presence of pain in the patient group, as pain can be considered a protective mechanism, causing adaptive changes in order to reduce potentially harmful and/or painful joint loads. In that case, iGRF and shock waves would be less than in a pain-free condition. However, based on our results, the impulsive ground reaction forces and shock waves seem to be of little significance in this respect. Secondly, our findings indicate that pain relief caused reduced peak tibial accelerations at heel strike. Finally, it is still not clear which structures contribute to the pain reported by OA patients and when in the gait cycle - or how - the pain is elicited while walking. Furthermore, pain relief resulted in a functional lengthening of the leg through increased extension at both hip and knee joints at heel strike. Change of knee joint angles at heel strike is effective in regulating shock wave magnitude, as higher degrees of knee joint flexion have been shown to result in higher tibial impact acceleration values (Lafortune et al. 1996). Our findings support this relationship, as the patients walked with more extended knee and hip joints - together with reduced tibial accelerations at heel strike following lidocaine injections. In a similar study of intraarticular injections of lidocaine and steroid (Shrader et al. 2004), no changes in joint kinematics were observed. This observation is probably due to an increase in walking speed following pain relief, which has been shown to affect knee joint angles at heel strike (Kirtley et al. 1985). In a study of long-term (4-week) osteoarthritic knee pain relief from a daily dose of 20 mg piroxicam, changes in knee joint angles at heel strike were similar to those seen in our results (Schnitzer et al. 1993). Although pain relief caused kinematic changes in the hip and knee joint at heel strike, the joint angles are similar to those considered to be normal values in both pain conditions. Similarly, the observed tibial peak accelerations are within previously reported normal range of 1.5-3 *g* (Folman et al. 1986, Voloshin 1988).

While we tested only small sample sizes, our results indicate that possible differences in iGRF and shock waves between OA and healthy controls are of no clinical relevance. To show a difference, based on the present results with a power of = 90% and α = 5%, requires a sample size of 547 in each group. As mentioned, the values reported here are well within the reference range (Folman et al. 1986, Voloshin 1988), which also supports the expectations of clinically insignificant differences between groups. On the other hand, it cannot be excluded that iGRF and shock waves are relevant in certain subgroups of OA patients that are still to be identified.

Concerns have been raised as to the possible adverse effects of training in OA patients, which might be expected to bring about further wear of the joint (Rogind et al. 1998). Our findings do not support limitation of everyday walking activities, and are in accordance with those of a recent meta-analysis suggesting that walking exercises may benefit the patient (Roddy et al. 2005).

Contributions of authors

All authors approved the final version of this article. MH, HL, BDS and HB were responsible for conception and design of the study, while MH, EBS and HB performed the data acquisition. Data analysis was done by MH and interpretation was done by MH, EBS and TGN. Drafting of the article was done by MH and EBS, and TGN, HL, BDS and HB assisted in critical revision of the article.

References

- 1. Burr, D. B. and Radin, E. L. (2003) Microfractures and microcracks in subchondral bone: are they relevant to osteoarthrosis?. *Rheum Dis Clin North Am* **29**:4 , pp. 675-685. [pubmed] [crossref]
- 2. Collins, J. J. and Whittle, M. W. (1989) Impulsive Forces During Walking and Their Clinical Implications. *Clin Biomech* **4**:3 , pp. 179-187. [crossref]
- 3. Fisher, N. M. and Pendergast, D. R. (1997) Reduced muscle function in patients with osteoarthritis. *Scand J Rehabil Med* **29**:4 , pp. 213-221. [pubmed]
- 4. Folman, Y., Wosk, J., Voloshin, A. and Liberty, S. (1986) Cyclic impacts on heel strike: a possible biomechanical factor in the etiology of degenerative disease of the human locomotor system. *Arch Orthop Trauma Surg* **104**:6 , pp. 363-365. [crossref]
- 5. Gill, H. S. and O'Connor, J. J. (2003) Heelstrike and the pathomechanics of osteoarthrosis: a pilot gait study. *J Biomech* **36**:11 , pp. 1625-1631. [pubmed] [crossref]
- 6. Kim, W., Voloshin, A. S., Johnson, S. H. and Simkin, A. (1993) Measurement of the impulsive bone motion by skin-mounted accelerometers. *J Biomech Eng* **115**:1 , pp. 47-52. [pubmed]
- 7. Kirtley, C., Whittle, M. W. and Jefferson, R. J. (1985) Influence of walking speed on gait parameters. *J Biomed Eng* **7**:4 , pp. 282-288. [pubmed] [crossref]
- 8. Lafortune, M. A., Lake, M. J. and Hennig, E. M. (1996) Differential shock transmission response of the human body to impact severity and lower limb posture. *J Biomech* **29**:12 , pp. 1531-1537. [crossref] [pubmed]
- 9. Moe-Nilssen, R. (1998) A new method for evaluating motor control in gait under real-life environmental conditions. Part 2: Gait analysis. *Clin Biomech* **13** , pp. 328-335. - (Bristol, Avon) [crossref]
- 10. Radin, E. L., Parker, H. G., Pugh, J. W., Steinberg, R. S., Paul, I. L. and Rose, R. M. (1973) Response of joints to impact loading. 3. Relationship between trabecular microfractures and cartilage degeneration. *J Biomech* **6**:1 , pp. 51-57. [crossref] [pubmed]

- 11. Radin, E. L., Ehrlich, M. G., Chernack, R., Abernethy, P., Paul, I. L. and Rose, R. M. (1978) Effect of repetitive impulsive loading on the knee joints of rabbits. *Clin Orthop* pp. 288-293. [pubmed]
- 12. Radin, E. L., Orr, R. B., Kelman, J. L., Paul, I. L. and Rose, R. M. (1982) Effect of prolonged walking on concrete on the knees of sheep. *J Biomech* **15**:7 , pp. 487-492. [crossref] [pubmed]
- 13. Radin, E. L., Yang, K. H., Riegger, C., Kish, V. L. and O'Connor, J. J. (1991) Relationship between lower limb dynamics and knee joint pain. *J Orthop Res* **9**:3 , pp. 398-405. [crossref] [pubmed]
- 14. Ratcliffe, R. J. and Holt, K. G. (1997) Low frequency shock absorption in human walking. *Gait Posture* **5**:2 , pp. 93-100. [crossref]
- 15. Robbins, S., Waked, E. and Krouglicof, N. (2001) Vertical impact increase in middle age may explain idiopathic weight-bearing joint osteoarthritis. *Arch Phys Med Rehabil* **82**:12 , pp. 1673-1677. [crossref] [pubmed]
- 16. Roddy, E., Zhang, W. and Doherty, M. (2005) Aerobic walking or strengthening exercise for osteoarthritis of the knee? A systematic review. *Ann Rheum Dis* **64**:4 , pp. 544-548. [crossref] [pubmed]
- 17. Rogind, H., Bibow-Nielsen, B., Jensen, B., Moller, H. C., Fri-modt-Moller, H. and Bliddal, H. (1998) The effects of a physical training program on patients with osteoarthritis of the knees. *Arch Phys Med Rehabil* **79**:11 , pp. 1421-1427. [crossref] [pubmed]
- 18. Schnitzer, T. J., Popovich, J. M., Andersson, G. B. and Andriacchi, T. P. (1993) Effect of piroxicam on gait in patients with osteoarthritis of the knee. *Arthritis Rheum* **36**:9 , pp. 1207-1213. [crossref] [pubmed]
- 19. Shorten, M. R. and Winslow, D. S. (1992) Spectral Analysis of Impact Shock During Running. *Int J Sport Biomech* **8** , pp. 288-304.
- 20. Shrader, M. W., Draganich, L. F., Pottenger, L. A. and Piotrowski, G. A. (2004) Effects of knee pain relief in osteoarthritis on gait and stair-stepping. *Clin Orthop* pp. 188-193. [pubmed]
- 21. Simon, S. R., Radin, E. L., Paul, I. L. and Rose, R. M. (1972) The response of joints to impact loading. II. In vivo behavior of subchondral bone. *J Biomech* **5**:3 , pp. 267-272. [crossref] [pubmed]
- 22. Slemenda, C., Brandt, K. D., Heilman, D. K., Mazzuca, S., Braunstein, E. M., Katz, B. P. and Wolinsky, F. D. (1997) Quadriceps weakness and osteoarthritis of the knee. *Ann Intern Med* **127**:2 , pp. 97-104. [pubmed]
- 23. Vaughan, C. L., Davis, B. L. and O'Connor, J. C. (1999) Dynamics of human gait. Kiboho Publishers , Cape Town

- 24. Voloshin, A. S. (1988) Shock absorption during running and walking. *J Am Podiatr Med Assoc* **78**:6 , pp. 295-299. [pubmed]
- 25. Wosk, J. and Voloshin, A. (1981) Wave attenuation in skeletons of young healthy persons. *J Biomech* **14**:4 , pp. 261-267. [crossref] [pubmed]

List of Figures

[Enlarge Image]

Figure 1. **Representative sample of sagittal plane ground reaction force in the first 45 ms of the stance phase of walking at 4 km/h. Impulsive ground reaction force (iGRF) was measured as the transient heel strike peak shown by the arrow.**

[Enlarge Image]

Figure 2. **Example of tibial (bottom) and sacral (top) accelerations during the initial 400 ms of stance. Peak values (indicated by arrows) were extracted as a measure of the acceleration wave magnitude at below-knee and pelvis levels, respectively.**

List of Tables

Average (SD) measures of pain, impulsive ground reaction forces (iGRF), shock waves and joint kinematics at heel strike in the healthy control group (n = 10) and in the OA group (n = 9) with and without knee-joint analgesia (intraarticular lidocaine injections)

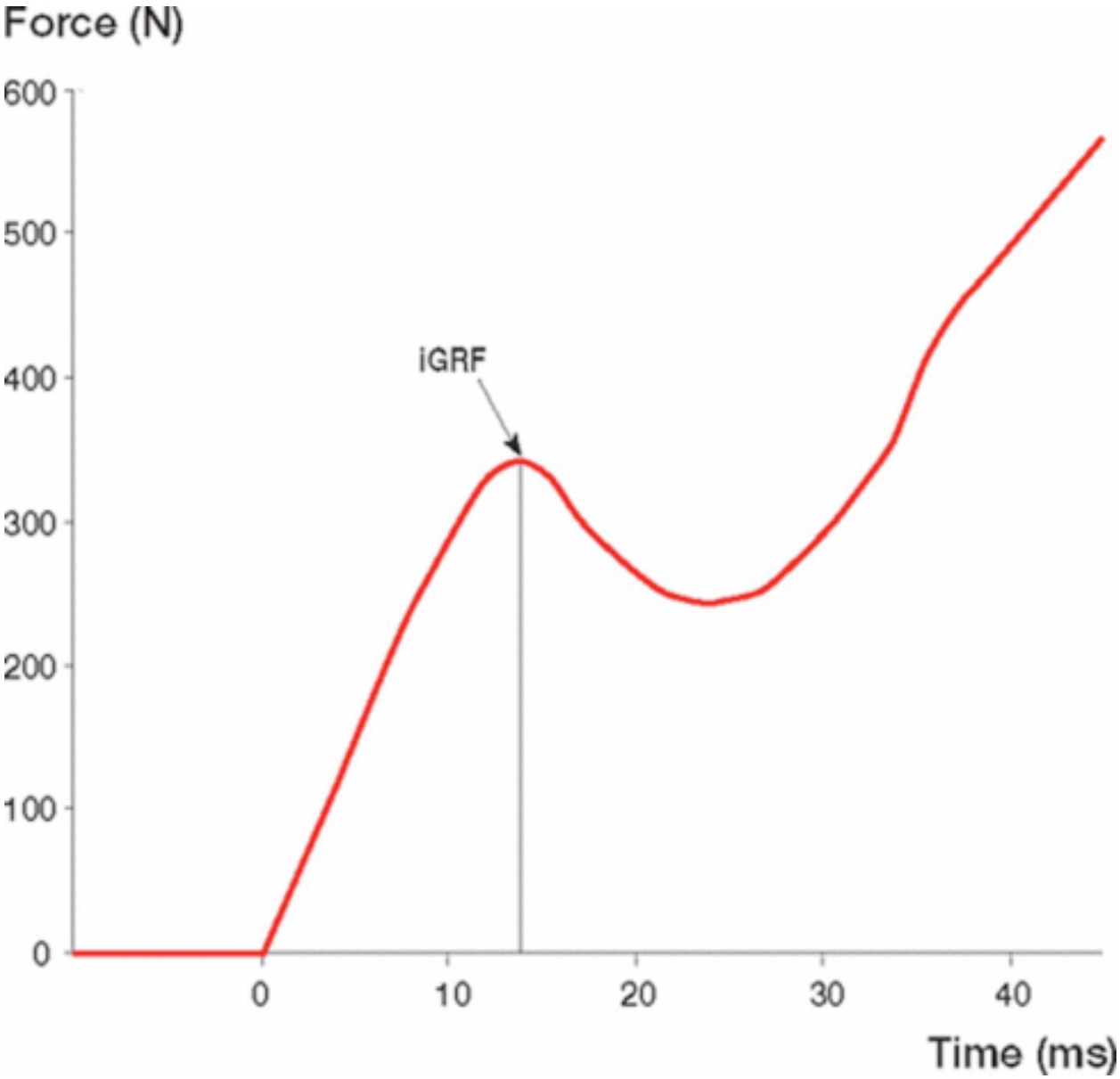
Healthy

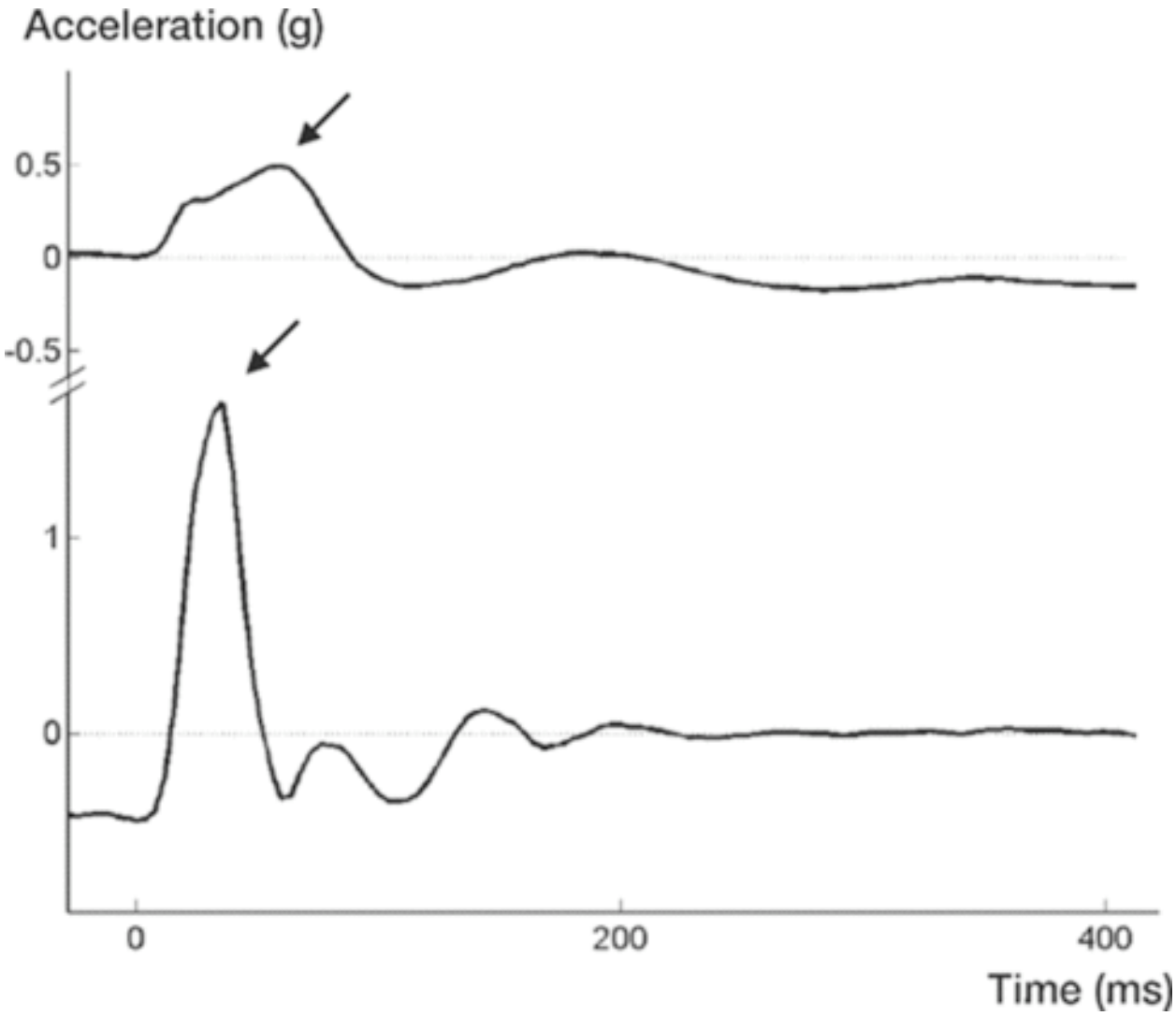
OA

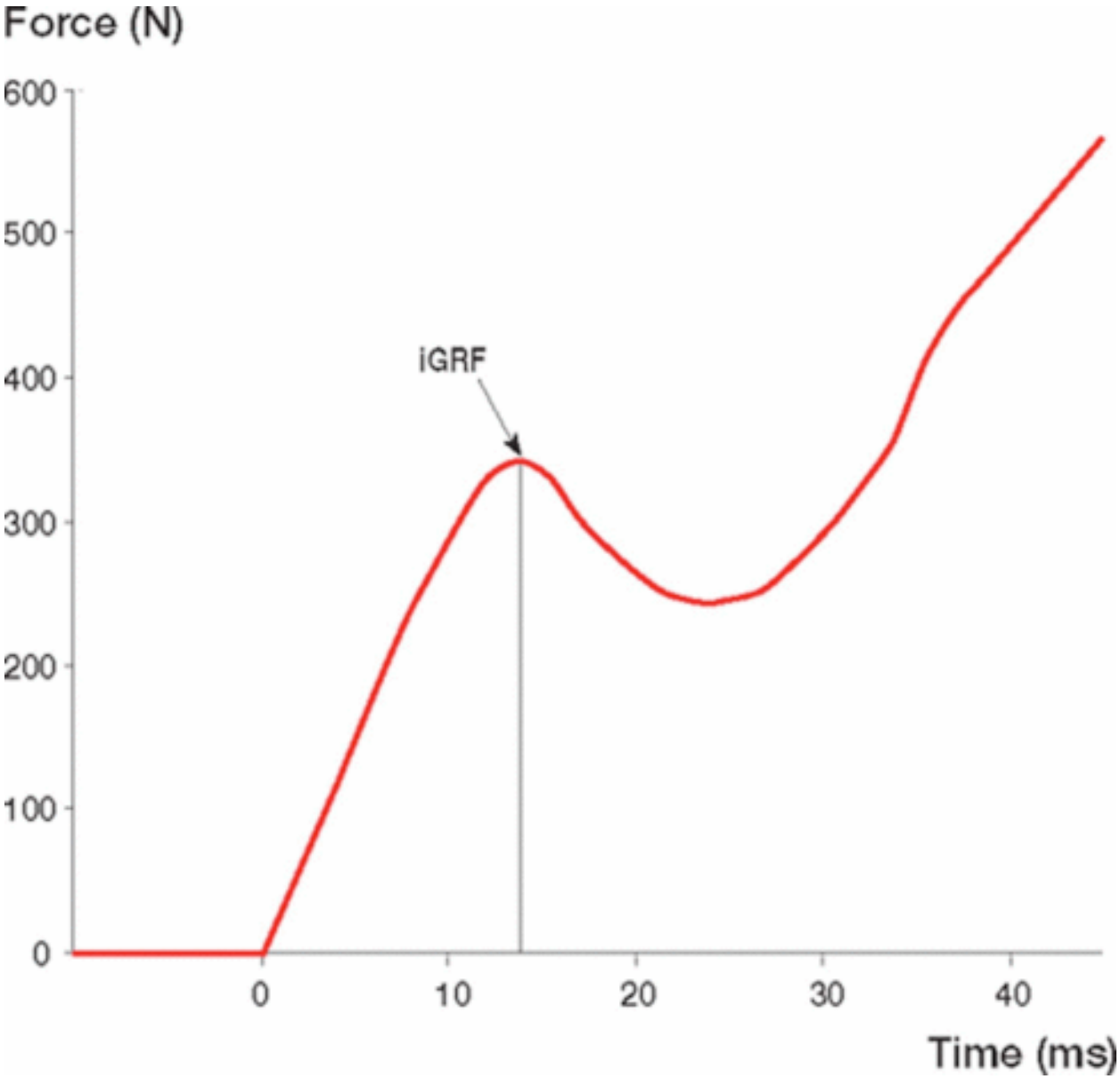
No analgesia
Analgesia

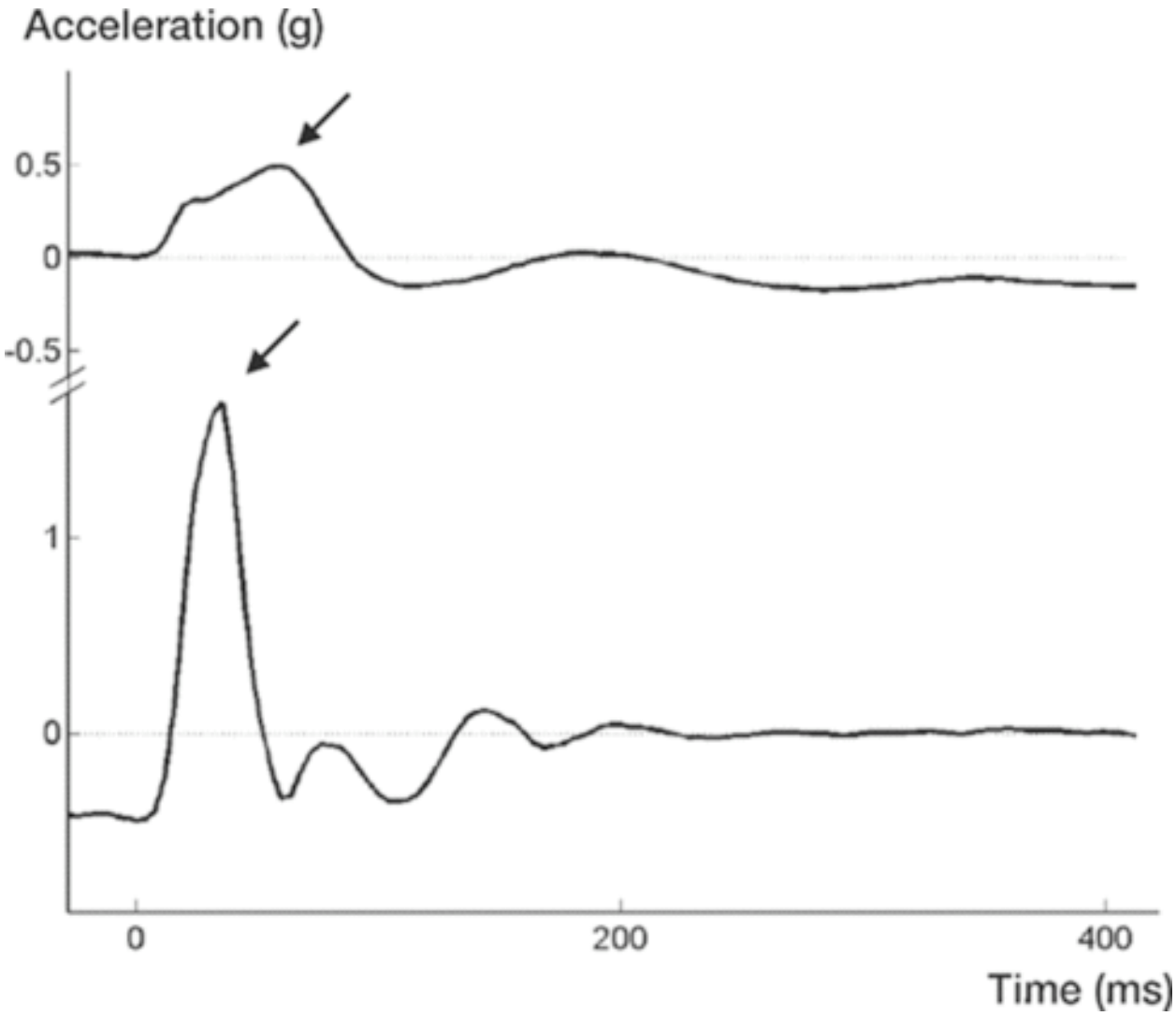
[a] significant difference between pain conditions in the OA group ($p < 0.01$).
[b] significant difference between pain conditions in the OA group ($p < 0.05$).
Pain (mm)
N/A
37 (28)
1.8 (2.0) [a]
iGRF (N/kg × 100)

514 (185)
485 (125)
464 (106)
Shock wave
Tibia (g)
1.91 (0.64)
2.00 (0.59)
1.76 (0.59) [a]
Sacrum (g)
0.54 (0.14)
0.52 (0.13)
0.50 (0.11)
Sagittal joint angles (degrees)
Ankle (dorsiflexion)
2.7 (4.0)
5.4 (4.6)
3.5 (4.4)
Knee (flexion)
5.6 (2.7)
8.4 (4.3)
4.0 (5.0) [a]
Hip (flexion)
27 (8.7)
27 (3.9)
24 (5.2) [b]


Force (N)
600
500
400
300
200
100
0
iGRF
0
10
20
30
40
Time (ms)


Acceleration (g)
0.5
0
-0.5
1
0
0
200
400
Time (ms)


Force (N)
600
500
400
300
200
100
0
iGRF
0
10
20
30
40
Time (ms)


Acceleration (g)
0.5
0
-0.5
1
0
0
200
400
Time (ms)