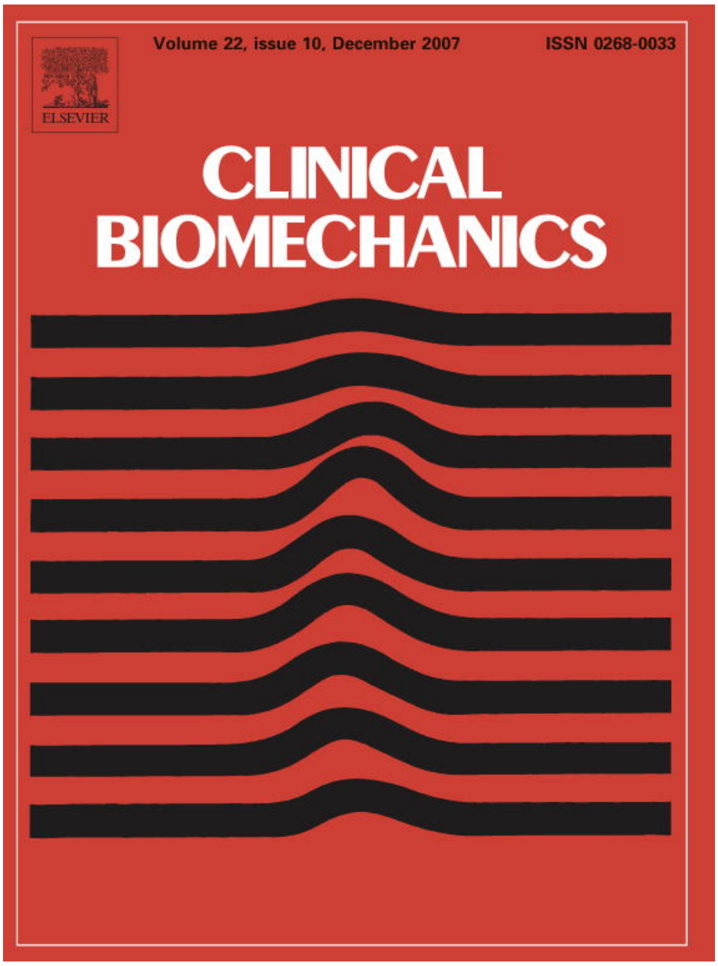
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Higher peak contact hip stress predetermines the side of hip involved in idiopathic osteoarthritis

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Abstract

Background. Biomechanical parameters of the hip have been suggested to have an important influence on the development of osteoarthritis. We aimed to find out whether higher stress is generated in a hip that subsequently results in earlier hip arthroplasty compared to the contralateral hip in the same subject.

Methods. Standard anterior–posterior pelvic radiographs with no or subtle radiological signs of hip osteoarthritis, of 59 female patients, who underwent hip arthroplasty for primary osteoarthritis years later, were selected from the archives. For each subject peak contact hip stress of the hip with earlier arthroplasty and of the contralateral hip (pair of hips), was calculated from the radiographically obtained geometrical parameters with the HIPSTRESS program, which is based on a three-dimensional biomechanical model of the resultant hip force in the one-legged stance and a three-dimensional mathematical model of the contact hip stress distribution. Differences in peak contact hip stress within pairs of hips were determined for subjects with unilateral (22 pairs of hips) and bilateral disease (37 pairs of hips) by using paired-samples *T*-test.

Findings. In the population of subjects with unilateral osteoarthritis, average peak contact hip stress was significantly higher ($P = 0.007$) in hips with arthroplasty (2.44 kPa/N) than in contralateral hips (2.32 kPa/N). In the population of subjects with bilateral osteoarthritis, average peak contact hip stress was significantly higher ($P < 0.001$) in hips with earlier arthroplasty (2.54 kPa/N) than in contralateral hips (2.35 kPa/N).

Interpretation. Results are consistent with the hypothesis that higher peak contact hip stress results in earlier hip arthroplasty due to faster development of idiopathic osteoarthritis.

Keywords: Contact stress; Hip; Osteoarthritis; Idiopathic; Coxarthrosis; Arthroplasty

1. Introduction

Over decades, causative factors for hip osteoarthritis development have been searched for, and it is known that several metabolic diseases and hip disorders lead to the development of secondary OA (Brinker and O'Connor, 2004). Research today is focused on the causes of idiopathic disease. Authors use questionnaires in large population surveys and case-control studies to study the influence of environmental and occupational exposure on osteoarthritis development (Rossignol, 2004).

Since it has been suggested that contact stress at various strenuous activities and its distribution on the articular surface might be important factors in cartilage degeneration and coxarthrosis occurrence, experimental and analytical approaches to study the effect of contact hip stress have

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been designed (Brand et al., 2001). Experimental studies have determined contact hip stresses at different physical activities by using an implanted partial endoprosthesis (Hodge et al., 1989) and provided us with the insight of stress distribution within the joint. In the last couple of years analytical approaches have gained popularity due to their non-invasiveness and cost-effectiveness. By evolving from two-dimensional to three-dimensional in both numerical (Ipavec et al., 1999) and discrete element analysis modelling (Yoshida et al., 2006), they became more reliable and relevant.

Previous studies failed to show a major importance of compressive stress in the etiology of an idiopathic hip osteoarthritis (Brinckmann et al., 1981). A new analytical method for calculating the resultant hip force, called HIP-STRESS, was introduced into clinical studies in the last few years (Ipavec et al., 1999). Studies have shown that peak contact hip stress, which could be an important contributor in idiopathic osteoarthritis development, depends on the geometry of the pelvis (Kersnič et al., 1997). It has been suggested that the development of coxarthrosis depends not only on the environmental factors but also on the intrinsic properties of the pelvic ring and hip. We based our study on the assumption that work and leisure time loading as well as body weight is symmetrical on both hips in a healthy subject and that additional factors determine the side of the hip, which would need earlier arthroplasty for idiopathic osteoarthritis compared to the contralateral hip.

Contact stresses in the hip joint have been associated with coxarthrosis development in various hip disorders. Higher hip stresses were observed in hips with developmental dysplasia compared to healthy hips, and it was proposed that hip stress might be responsible for earlier osteoarthritis occurrence (Mavčič et al., 2002). It has also been suggested that avascular necrosis decreases the weight-bearing surface of the hip, which results in an increment of the peak contact hip stress and faster degeneration of the cartilage (Daniel et al., 2006). Postoperative contact hip stress has been determined for various procedures used in the treatment of developmental dysplasia of the hip (Vengust et al., 2001; Herman et al., 2002; Kralj et al., 2005), avascular necrosis of the femoral head (Dolinar et al., 2003), and slipped capital femoral epiphysis (Zupanc et al., 2001) with the intention to provide mathematical evidence of reduction in hip stress, which was suggested to delay progression of the disease into osteoarthritis.

Based on these recent papers, peak contact hip stress was used in our study to reflect the geometrical intrinsic properties of the pelvis and hips that might be responsible for earlier hip replacement due to faster osteoarthritis development. The objective of the paper is to investigate whether hips, which needed earlier arthroplasty for coxarthrosis years later, have higher values of peak contact hip stress compared to the contralateral hips.

2. Methods

Fifty-nine subjects were selected from a group of 431 female patients who had had a total hip replacement for primary hip osteoarthritis at the Department of Orthopedic Surgery, University Clinical Hospital Maribor in the years 2004 and 2005. Exclusion criterion for participation in the study included secondary causes for osteoarthritis of the hip, which were established through medical records. Ninety patients with rheumatoid or psoriatic arthritis, avascular necrosis, slipped capital femoral epiphysis, dysplasia of the hip or lower extremity fracture, were excluded from the survey at this stage. We recorded no activities that would cause a long-term asymmetrical loading of hips.

Pelvic radiographs of patients that were taken within a single facility years before the operation for various reasons, including back pain, discrete pain in the hips or minor injury to the pelvis were searched for in the central archives; 92 radiographs were recovered. The width of the hip joint space was measured at the lateral margin of the subchondral sclerotic line, at the apical transection of the weight-bearing surface by a vertical line through the centre of the femoral head, and at the medial margin of the weight-bearing surface bordering on the fovea (Fig. 1). The minimum joint space width (m.j.s.w.) was defined as the smallest of the three measurements. Only 65 complete anterior–posterior radiographs with both hips and pelvis clearly visible, with spherical femoral heads, which complied well with Mose circle templates (Mose, 1980), and without considerable joint space narrowing (m.j.s.w. less than 3 mm), large osteophytes, subchondral cysts or acetabular protrusion were considered appropriate. We were unable to locate three of these patients and one failed to give uniform consent to participate in the

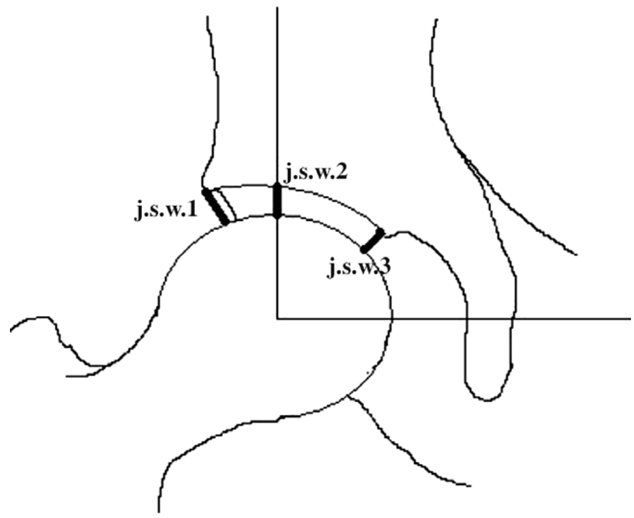

Fig. 1. Measurement of joint space width in the hip joint (j.s.w. – width of the hip joint space measured at the lateral margin of the subchondral sclerotic line (1), at the apical transection of the weight-bearing surface by a vertical line through the centre of the femoral head (2), and at the medial margin of the weight-bearing surface bordering on the fovea (3)).

study as the rest did. Two patients, when questioned about secondary causes of osteoarthritis, reported a fracture of the lower extremity during childhood. Hip stresses of the left and the right hip in 59 selected radiographs with no or only initial stage of coxarthrosis, which allowed reliable model implementation, were further evaluated.

The evaluation of peak contact hip stress was performed according to the HIPSTRESS method based on a three-dimensional biomechanical model of the resultant hip force in the one-legged stance (Iglič et al., 1993) and a three-dimensional mathematical model of the contact hip stress distribution (Ipavec et al., 1999). These models require as the input data body weight and geometrical parameters of the pelvis and proximal femora. The following parameters were measured from the pelvic contours and used to calculate the resultant hip force (Fig. 2): the pelvic height (H), the pelvic width laterally from the centre of the articular sphere (C), the interhip distance (l) and the coordinates of the insertion of the effective muscle on the greater trochanter (x, z) in its respect to the centre of the articular sphere. Accounting for the 10% magnification of the radiographs (except for the scale-independent centre-edge angle), the corrected values of all the parameters were used. The equations for the equilibrium of forces and torques were solved to yield the magnitude of the resultant hip force (**R**) and its inclination with respect to the vertical (ϑ_R). These parameters were used together with the centre-edge angle (ϑ_{CE}) and the radius of the femoral head (r) to calculate the peak contact hip stress (P_{max}). The resultant hip force and the corresponding peak stress were calculated as normalized to the body weight: R/W_B and P_{max}/W_B, respectively.

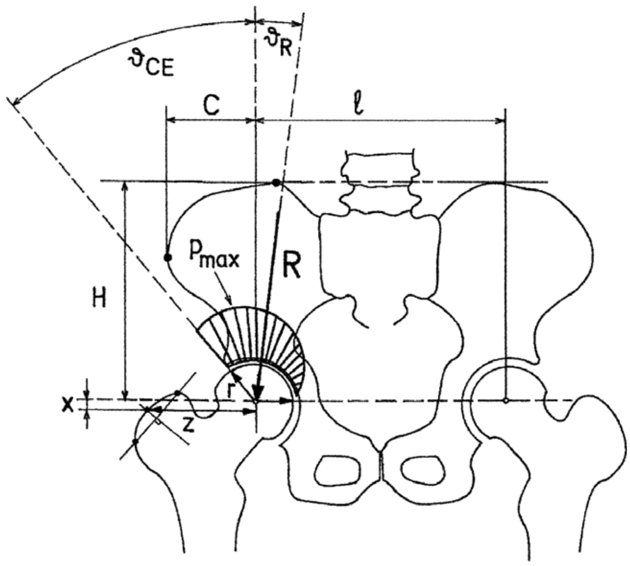

Fig. 2. The geometrical and biomechanical parameters of the hip and pelvis needed for determination of the peak stress on the weigh bearing area (l – the interhip distance, C – pelvic width laterally from the centre of the articular sphere, H – pelvic height, r – radius of the femoral head, x, z – vertical and horizontal coordinates of the insertion of the effective muscle on the greater trochanter with respect to the centre of the articular sphere, **R** – magnitude of the resultant hip force, ϑ_R – inclination of the resultant hip force with respect to the vertical axis, ϑ_{CE} – centre-edge angle, P_{max} – peak contact hip stress displayed with the gradient of contact stress inside hip joint).

The side of coxarthrosis was recorded for every subject based on the available medical records and side of the arthroplasty. Subjects were divided into two populations with unilateral and bilateral osteoarthritis. Accordingly, our samples consisted of 22 female patients with unilateral and 37 female patients with bilateral disease. The ages of the subjects in the former population ranged from 45 to 79, median 69 years, while in the latter, the subjects' ages ranged from 50 to 80, median 68 years. Pelvic radiographs were made on average 4.4 years prior to the hip replacement in the population with unilateral disease and 5.0 years prior to the first total hip replacement in the population with bilateral disease.

In the population with unilateral disease, peak contact hip stress in hips that had arthroplasty performed was compared to peak contact hip stress of the contralateral hips for which we found no sign of degenerative process (pairs of hips). In the population with bilateral disease, peak contact hip stress of hips with earlier implementation of total hip endoprosthesis was compared to peak contact hip stress of contralateral hips with later (or none yet) implementation of total hip endoprosthesis (pairs of hips). Also, values of resultant hip force normalized to the body weight (R/W_B) and its inclination with respect to the vertical (ϑ_R), geometrical parameters, used to calculate the peak contact hip stress ($\vartheta_{CE}, C, H, r, x, z$), and values of minimum joint space width were compared for pairs of hips within the above populations. Values of geometrical parameters were not influenced by the difference in minimum joint space width. Paired-samples T-test was used for statistical analysis. Statistical significance was determined at $P < 0.05$ level.

3. Results

In the population of subjects with unilateral diseases as presented in Table 1, statistically significant ($P = 0.007$) higher normalized peak contact hip stress was found in hips that needed arthroplasty due to primary osteoarthritis compared to those that did not (2.44 kPa/N compared to 2.32 kPa/N). Also, significantly lower centre-edge angle was observed in hips with performed arthroplasty.

Table 2 demonstrates statistically significant ($P < 0.001$) higher normalized peak contact hip stress in hips with earlier arthroplasty (2.54 kPa/N) compared to the contralateral hips (2.35 kPa/N) with corresponding standard deviation in the population of subjects with bilateral osteoarthritis. The resultant hip force was found to be higher in magnitude and more vertically oriented in subjects with earlier arthroplasty. Furthermore, significantly lower values of centre-edge angle and horizontal offset (z) as well as higher values of vertical offset (x) were measured in this group. We found no other statistically significant difference in measured parameters within populations.

Table 1
Comparison of mean values of peak contact hip stress and resultant hip force normalized to the body weight (P_{max}/W_B; R/W_B), inclination of the force with respect to the vertical (ϑ_R), geometrical parameters used to calculate peak contact hip stress and minimum joint space width, between hips with and without arthroplasty in a population of 22 female subjects with unilateral osteoarthritis and the statistical significance of the difference determined with paired-samples *T*-test

Parameter	Hips with arthroplasty		Hips without arthroplasty		*P*
	Mean	Standard deviation	Mean	Standard deviation	
P_{max}/W_B (kPa/N)	2.44	0.49	2.32	0.47	0.007
R/W_B	2.61	0.21	2.58	0.20	0.21
ϑ_R (degrees)	7.8	1.3	7.9	1.1	0.47
ϑ_{CE} (degrees)	33.5	7.1	35.2	7.3	0.03
r (mm)	26.5	1.5	26.5	1.4	0.84
C (mm)	64.0	10.6	64.8	9.5	0.50
H (mm)	157.1	8.3	157.8	8.3	0.18
x (mm)	10.8	8.7	11.8	8.7	0.41
z (mm)	66.9	5.6	68.3	4.8	0.07
m.j.s.w. (mm)	4.0	0.9	4.2	0.9	0.53

Table 2
Comparison of mean values of peak contact hip stress and resultant hip force normalized to the body weight (P_{max}/W_B; R/W_B), inclination of the force with respect to the vertical (ϑ_R), geometrical parameters used to calculate peak contact hip stress and minimum joint space width, between hips with earlier arthroplasty and contralateral hips in the population of 37 female subjects with bilateral osteoarthritis and the statistical significance of the difference determined with paired-samples *T*-test

Parameter	Hips with earlier arthroplasty		Contralateral hips		*P*
	Mean	Standard deviation	Mean	Standard deviation	
P_{max}/W_B (kPa/N)	2.54	0.55	2.35	0.49	< 0.001
R/W_B	2.59	0.17	2.54	0.17	0.005
ϑ_R (degrees)	7.5	0.9	7.8	1.0	0.016
ϑ_{CE} (degrees)	35.1	8.3	36.7	8.5	0.005
r (mm)	25.8	1.8	26.0	1.7	0.09
C (mm)	65.1	9.3	65.5	8.6	0.65
H (mm)	153.7	8.3	154.1	8.6	0.22
x (mm)	13.6	7.4	11.2	7.8	0.05
z (mm)	65.3	5.1	66.7	4.9	0.01
m.j.s.w. (mm)	3.8	1.0	3.9	0.9	0.81

4. Discussion

In this work we showed that the peak contact hip stress was on average higher on that side of the pelvis, where earlier arthroplasty (due to faster development of osteoarthritis) was performed. It has been suggested that temporal and spatial aspects of loading history rather than static contact stresses are responsible for the deleterious biological responses in articular cartilage (Brand, 2005). By comparing two hips in the same subject, assumption was made that the effect of work and leisure time loading was the same on both hips. We believe that static contact stresses do influence cartilage degeneration but the relative importance in respect to the ‘‘dynamic’’ contact stresses remains to be evaluated.

Although statistically highly significant difference in peak contact hip stress was found between groups of subjects within both populations, only small absolute difference in its average value was observed. By recruiting patients with developmental dysplasia of the hip or progressive obliteration of hip joint space the difference in average value of peak contact hip stress would be greater but the basic concept of our study would be flawed. Good selection of subjects might, therefore, be responsible for narrowing the peak contact hip stress gap, but leaving enough to make reliable conclusions.

A classical study, that compared a population of healthy hips and hips with idiopathic osteoarthritis (Brinckmann et al., 1981), failed to show any significant difference in peak contact hip stress between the two populations. We believe the results were biased due to the difference in the age of the populations – healthy hips being younger. It has been shown that peak contact hip stress decreases with age in the healthy population (Mavčič et al., 2004), presumably due to the progress of arthrosis and elimination of hips with higher peak contact hip stress from the population of healthy hips. One would expect that if populations of Brinckmann et al. were age-controlled, lower peak contact hip stress would be observed in the population of healthy hips.

Studies based on mathematical models have shown that lack of lateral coverage of the hip joint in hip dysplasia results in lower values of centre-edge angle (less than 20° for diagnosis) with consequently higher peak contact hip stress and faster osteoarthritis development (Mavčič et al., 2002). Our study found that the above association between lateral coverage, hip stress and coxarthrosis might also be applicable to the representative population of female hips with centre-edge angle averaging 35° (Jacobsen et al., 2005), making healthy hips with less lateral coverage more prone to developing earlier osteoarthritis. Furthermore, more caudally (higher *x* value) and medially (lower *z* value) located insertion of effective abductor muscle on greater trochanter, as observed by us in hips with earlier arthroplasty (within bilateral osteoarthritis group) and by Mavčič et al., 2002, in dysplastic hips, results in a more vertical orientation (lower ϑ_R) and higher magnitude of the resultant hip force, which subsequently produces higher peak stress (Daniel et al., 2001). Although mainly occupational demands have been proposed to contribute to hip osteoarthritis development (Rossignol, 2004), results of our study suggest that unfavourable biomechanical and geometrical properties of an otherwise healthy hip play an important etiological role in coxarthrosis.

The mathematical model used allows us to evaluate static parameters in one-legged stance only. It was assumed that this is the representative position for walking and, therefore, the most frequent activity in everyday life (McLeish and Charnley, 1970). It has been proposed that contact hip stresses in one-legged stance are reliable in predicting contact hip stresses during slow walking (Ipavec et al., 1999). A study by Hodge et al, which measured *in vivo* contact hip stresses in a single individual after a partial hip implant, recorded maximum contact hip stress 4,0 MPa during normal level walking 3 years postoperatively (after extensive rehabilitation and with improved muscle coordination). Its value normalized to the body weight ($P_{max}/W_B = 6.0$ kPa/N) is higher than an average value for peak contact hip stress obtained in our study, which could be attributed to the uneven distribution of stress on the metal–cartilage interface.

Our average values of resultant hip force (2.54–2.61 W_B) are in line with the *in vivo* measurements of the joint force in four patients with total hip implants (2.39–2.55 Wf_B) as observed by Bergmann et al., 2001. However, higher values of peak contact hip stress during slow walking ($P_{max}/W_B = 4.1$ kPa/N) were obtained with discrete element analysis of the contact area and pressure distribution in one of the tested subjects (Yoshida et al., 2006). Such a discrepancy might be caused by the introduction of compressive and shear spring of the deformable articular cartilage into the model and by a smaller contact area modeled. In addition to not considering fovea as part of the joint surface from the start, further reductions in contact area during the gait cycle were calculated (up to 20%). Weakness of our three-dimensional mathematical model (Ipavec et al., 1999) is, that it represents one specific point in the gait cycle, not accounting for the size of the fovea or contact area changes, and fails to account for the true elastic properties of the articular cartilage. Lower absolute values of peak contact hip stress nevertheless do not undermine the statistical significance observed, since proper model adjustments would influence both hips in the same pelvis equally.

In hips with considerable joint space narrowing, the shift of the centre of the femoral head in regard to the pelvic girdle could, depending on the direction of the shift, influence values of geometrical parameters and subsequently, the calculations of the resultant hip force and peak contact stress (Daniel et al., 2001). Since no significant difference in the average minimal joint space width within pairs of hips was observed, we believe no such influence was exerted. Design of our study does not allow us to speculate on the level of normalized peak contact hip stress able to produce osteoarthritis. By acquiring body weight of our subjects, absolute values of peak contact hip stress could be calculated and further analysed. Even more, if detailed data on occupational demands and leisure time loading would be recovered, then determining the relative importance of "static versus dynamic" hip stress in coxarthrosis development might become possible.

5. Conclusions

Average normalized peak contact hip stress was significantly higher in hips that needed earlier arthroplasty due to faster development of idiopathic osteoarthritis. Our findings indicate that higher contact hip stress could be a risk factor for the development of osteoarthritis in healthy hips and should, therefore, be incorporated in further study designs regarding etiology of coxarthrosis.

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