# Osteoarthritis and Cartilage

I C R S **International Cartilage Repair Society**

OARSI **OSTEOARTHRITIS RESEARCH SOCIETY INTERNATIONAL**

# The role of obesity, biomechanical constitution of the pelvis and contact joint stress in progression of hip osteoarthritis

G. Recnik†*, V. Kralj-Iglič‡, A. Iglič§, V. Antolič||, S. Kramberger†, I. Rigler||, B. Pompe||
and R. Vengust||
† Department of Orthopedic Surgery, University Hospital Maribor, Maribor, Slovenia
‡ Laboratory of Clinical Biophysics, Faculty of Medicine, University of Ljubljana, Ljubljana, Slovenia
§ Laboratory of Physics, Faculty of Electrical Engineering, University of Ljubljana, Ljubljana, Slovenia
|| Department of Orthopedic Surgery, University Medical Centre Ljubljana, Ljubljana, Slovenia

## Summary

*Objective*: The aim of our study was to explore whether earlier hip arthroplasty for idiopathic osteoarthritis (OA) might be explained by enlarged contact stress in the hip joint, and to what amount can that be attributed to obesity and biomechanical constitution of the pelvis.

*Method*: Fifty subjects were selected from a list of consecutive recipients of hip endoprosthesis due to idiopathic OA; standard pelvic radiographs made years prior to surgery were the main selection criteria. For 65 hips resultant hip force and peak contact hip stress normalized to the body weight ($R$/Wb and $p_{max}$/Wb) were determined from the radiographs with the HIPSTRESS method. Body weight and body mass index (BMI) were obtained with an interview. Regression analysis was used to correlate parameters of obesity (body weight, BMI), biomechanical constitution of the pelvis ($R$/Wb, $p_{max}$/Wb) and mechanical loading within the hip joint ($R$, $p_{max}$) with age at hip arthroplasty.

*Results*: Younger age at hip arthroplasty was associated with higher body weight ($P = 0.009$), higher peak contact hip stress normalized to the body weight − $p_{max}$/Wb ($P = 0.019$), higher resultant hip force − $R$ ($P = 0.027$) and larger peak contact hip stress − $p_{max}$ ($P < 0.001$), but not with BMI ($P = 0.121$) or $R$/Wb ($P = 0.614$).

*Conclusion*: Our results suggest that enlarged contact stress ($p_{max}$) plays an important role in rapid progression of hip OA with both obesity (increased body weight) and unfavorable biomechanical constitution of the pelvis (greater $p_{max}$/Wb) contributing.
© 2008 Osteoarthritis Research Society International. Published by Elsevier Ltd. All rights reserved.

*Key words*: Hip, Osteoarthritis, Contact stress, HIPSTRESS, Body weight, Arthroplasty.

## Introduction

The process of joint degeneration that leads to clinical symptoms of osteoarthritis (OA) remains poorly understood[1]. However, it has been pointed out that the evidence for the moderate influence of obesity on hip OA development is growing[2]. Higher relative risks were found for hip OA in subjects with increased body mass indices (BMI), with even higher risks attributed to increased body weight[3]. Most researchers nowadays believe the effect of overweight on OA occurrence in weight-bearing joints is predominantly a mechanical one. A similar mechanism has previously been suggested to be responsible for higher incidence of coxarthrosis in farmers involved in heavy lifting[4]. Since mechanical compressive stress was found to be related to chondrocyte senescence[5], increased loading forces within the hip joint due to excess body weight or heavy lifting with consequently enlarged peak contact hip stress could be responsible for accelerated loss of chondrocyte function and earlier degenerative thinning of articular cartilage.

Although previous studies on biomechanics of the hip failed to show major importance of compressive stress in the etiology of primary coxarthrosis[6], a new analytical method for calculating the resultant hip force and peak contact stress challenged the conclusions made above. In the last few years the HIPSTRESS method[7] has successfully provided mathematical evidence of reduction in contact hip stress after operative procedures for developmental dysplasia of the hip[8], avascular necrosis of the femoral head[9] and slipped capital femoral epiphysis[10], as well as indicated the importance of stress in idiopathic hip OA development.

Higher hip stress was observed in a hip with subsequent earlier hip arthroplasty compared to the contralateral hip in the same subject with either unilateral or bilateral disease[11]. Furthermore, hip stress among the healthy was shown to decrease with age of the population, which was presumed to be caused by the gradual shifting of hips with higher stress into the population of OA hips[12]. Similar hip joint forces and peak contact stresses compared to those obtained by experimental studies constitute a sort of consensual validity for the HIPSTRESS method, even though no reciprocal validation was ever performed[13].

Since biomechanical constitution of the pelvic ring and hips affects peak contact hip stress distribution and magnitude[14], the importance of enlarged stress due to hip deformity has become appreciated in secondary OA etiology.

Most supporting data comes from studies on subjects with developmental dysplasia of the hip. Within the population of dysplastic hips, those with higher contact stress underwent joint degeneration earlier, which resulted in clinical and radiological unsatisfactory outcomes[15]. Comparing populations of healthy and dysplastic hips, higher cumulative hip stress was suggested to be responsible for faster OA occurrence in hips with developmental dysplasia[16].

The aim of our study was to further explore the role of compressive mechanical stress in progression of idiopathic hip OA. We pursued the idea by determining whether earlier hip arthroplasty might be related to enlarged contact stress within the hip joint. The question was addressed by studying the relationship between age at hip arthroplasty, and parameters of obesity, biomechanical constitution of the pelvis and mechanical loading within the hip joint.

### Methods

Subjects were recruited from a group of 431 consecutive female recipients of total hip endoprosthesis. Secondary causes for OA of the hip were used as clinical exclusion criteria in 92 patients. Archives were searched in order to recover 90 standard anterior−posterior pelvic radiographs made years prior to the operation as routine clinical images in the supine position. Acetabular protrusion, dysplasia, considerable joint space narrowing and large osteophytes were used as radiographic exclusion criteria in 27 patients. Mean age at pelvic radiographs taking was 63.1 ($\pm$8.1) years and at hip arthroplasty 68.5 ($\pm$7.8) years for 35 excluded hips. Radiographs with both hips and pelvis clearly visible, and with spherical femoral heads, which complied with Mose circle templates[17], were obtained for 63 patients.

We were unable to locate three of them and two patients have died by the time of the study. The rest were questioned by a single health care professional in a telephone interview to remember their most constant lifelong height and body weight. Four patients were unwilling to give informed consent and four were unable to report reliable data due to extreme fluctuations of body weight through life. BMI defined as body weight (kg) divided by height squared ($m^2$) was calculated for 50 female subjects (participation rate 79%) with performed 65 total hip arthroplasties (35 unilateral and 15 bilateral). Significantly larger contact stresses were observed in operated than in contralateral hips, as published in a previous study[11].

Pelvic radiographs were used to evaluate peak contact hip stress with the HIPSTRESS method, which is based on a three-dimensional biomechanical model of the resultant hip force in the one-legged stance[18] and a three-dimensional mathematical model of contact hip stress distribution[7]. It has shown good repeatability (intra-class correlation coefficient for $p_{max}$/Wb being 0.94) when adjusted for pelvic/femoral inclination[19]. Body weight and geometrical parameters of the pelvis and proximal femora are used as input data by these models. The following parameters were measured from the pelvic contours (Fig. 1) and used to calculate the resultant hip force: the pelvic height ($H$), the pelvic width laterally from the center of the articular sphere ($C$), the interhip distance ($l$) and the coordinates of the insertion of the effective muscle on the greater trochanter in its respect to the center of the articular sphere ($x$, $z$). Accounting for the 10% magnification of the radiographs (except for the scale-independent center-edge angle), the corrected values of all the parameters were used. The equations for the equilibrium of forces and torques were solved to provide the magnitude of the resultant hip force ($R$) and its inclination with respect to the vertical ($\vartheta_R$). They were used together with center-edge angle ($\vartheta_{CE}$) and radius of the femoral head ($r$) to calculate peak contact hip stress ($p_{max}$); the corresponding resultant hip force and peak stress normalized to the body weight ($R$/Wb, $p_{max}$/Wb), representing biomechanical constitution of the pelvis, were calculated.

Univariate regression analysis was used to correlate age at hip arthroplasty with parameters of obesity (body weight, BMI), biomechanical constitution of the pelvis ($R$/Wb, $p_{max}$/Wb) and mechanical loading within the hip joint ($R$, $p_{max}$), all of which followed a normal distribution. In addition, a bivariate regression model was constructed in order to try to explain the variability in age at hip arthroplasty with the most prominent parameters of obesity (body weight) and biomechanical constitution of the pelvis ($p_{max}$/Wb). All analyses were performed using the SPSS 12.0 statistical package with significance determined at $P < 0.05$ level.

### Results

The characteristics of the study population, eligible hips and their radiographic parameters are presented in Table I. Pelvic radiographs were taken on average at 62.5 ($\pm$8.5) years of age, while hip arthroplasty was performed at 67.6 ($\pm$8.4) years of age.

The relationship between age at hip arthroplasty and parameters of obesity, biomechanical constitution of the pelvis
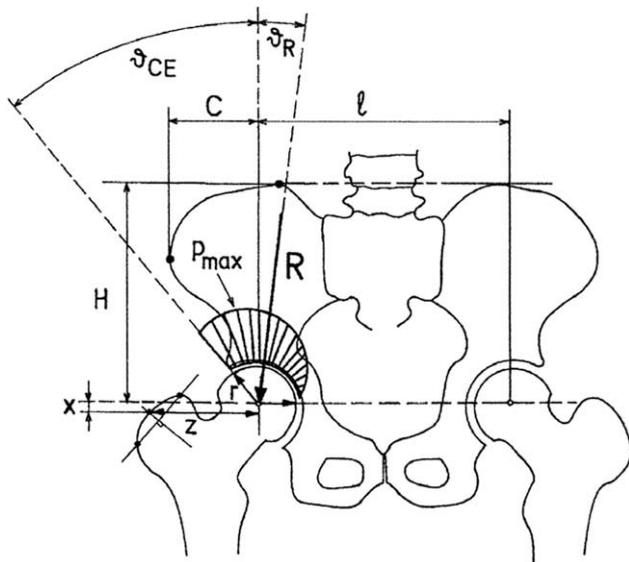


Fig. 1. The geometrical and biomechanical parameters of the pelvis and hips used to calculate the peak contact hip stress ($l$ − the interhip distance, $C$ − pelvic width laterally from the center of the articular sphere, $H$ − pelvic height, $r$ − radius of the femoral head, $x$, $z$ − vertical and horizontal coordinates of the insertion of the effective muscle on the greater trochanter with respect to the center of the articular sphere, $R$ − magnitude of the resultant hip force, $\vartheta_R$ − inclination of the resultant hip force with respect to the vertical axis, $\vartheta_{CE}$ − center-edge angle, $p_{max}$ − peak contact hip stress displayed with the gradient of contact stress inside hip joint).

Table I
*Demographic and exposure characteristics of the study population*

| Characteristics of subjects | $n = 50$ |
|---|---|
| Female gender | 100% |
| Body height (cm) | 164 (5) |
| Body weight (kg) | 71 (10) |
| Body mass index − BMI (kg/m$^2$) | 26.2 (3.5) |
| **Characteristics of eligible hips** | $n = 65$ |
| Age at hip arthroplasty (years) | 67.6 (8.4) |
| Age at pelvic radiographs' taking (years) | 62.5 (8.5) |
| Peak contact hip stress − $p_{max}$ (MPa)* | 1.69 (0.41) |
| Peak contact hip stress normalized to body weight − $p_{max}$/Wb (kPa/N)* | 2.45 (0.52) |
| Magnitude of the resultant hip force − $R$ (kN)* | 1.79 (0.30) |
| Resultant hip force normalized to body weight − $R$/Wb* | 2.57 (0.18) |
| Inclination of the resultant hip force − $\vartheta_R$ (°)* | 8 (1) |
| **Characteristics of radiographic parameters** | |
| Pelvic width laterally from the center of the articular sphere − $C$ (mm)* | 65 (9) |
| Pelvic height − $H$ (mm) * | 155 (8) |
| Radius of the femoral head − $r$ (mm)* | 26 (1.5) |
| Interhip distance − $l$ (mm)* | 198 (12) |
| Vertical offset of abductor muscle insertion − $x$ (mm)* | 12 (7) |
| Horizontal offset of abductor muscle insertion − $z$ (mm)* | 67 (5) |
| Center-edge angle − $\vartheta_{CE}$ (°)* | 35 (7) |

All variables described as mean ($\pm$SD) unless otherwise stated.
*Please refer to Fig. 1 for further details.

and mechanical loading within the hip joint is presented in Table II. Using univariate regression analysis, a year earlier arthroplasty was found to be associated with 4.5 kg increase in body weight ($P = 0.016$) and statistically insignificant 2.5 kg/m$^2$ increase in BMI ($P = 0.121$). With regard to biomechanical constitution of the pelvis, 1 year sooner total hip replacement was related to 0.263 kPa/N increase in $p_{max}$/Wb ($P = 0.034$), but not $R$/Wb ($P = 0.614$). Younger age at hip arthroplasty correlated well with increased mechanical loading within the hip joint; 147 N greater $R$ ($P = 0.027$) and 139 kPa larger $p_{max}$ ($P = 0.001$) were associated with endoprosthesis implantation 1 year earlier. Furthermore, a satisfactory bivariate regression model with body weight and peak contact hip stress normalized to the body weight acting as independent variables was constructed explaining 14.0% of variability in age at hip arthroplasty (adjusted $R^2 = 0.140$; $P = 0.004$).

## Discussion

Our study has shown a significant negative correlation between age at hip arthroplasty and contact hip stress — $p_{max}$ — suggesting compressive stress plays an important role in progression of hip OA. Obesity — body weight — and unfavorable biomechanical constitution of the pelvis — $p_{max}$/Wb — were both associated with younger age at arthroplasty.

No previous study has examined the relationship between mechanical loading within the hip joint and age at hip arthroplasty. Our results support the thesis that body weight might be a more important risk factor in coxarthrosis occurrence than BMI[3], with its mechanical influence exerted through an increase in $R$ and the enlargement of $p_{max}$. While $R$/Wb is widely used in implant design as a determinant of hip joint biomechanics[20], our findings suggest that

$p_{max}$/Wb might be better in representing the biomechanical constitution of the pelvis responsible for hip cartilage pathology. As the regression model with $p_{max}$ serving as an independent variable provided best correlation with age at hip arthroplasty, enlarged contact stress in the standing position [MPa] is proposed to be the principal mechanical entity behind articular cartilage damage over ones' lifetime [years]. Occupational surveys on heavy lifting, in which both weight of the burdens (at least 10−20 kg) and the duration of lifting (at least 10−20 years) were associated with increased risk of hip OA[21], further support this claim. However, it appears that lifelong joint trauma in long-distance runners does not increase the risk of hip OA, presumably due to change in joint fluid composition and adaptive soft-tissue hypertrophy[22], making the association between mechanical stress and cartilage degeneration less straight-forward and meriting further research.

Since timing of the operation is influenced by clinical as well as social-economic factors[23], age at hip arthroplasty is not a validated, but rather improvised surrogate for hip OA onset, which poses a limitation of our study. Also, although self-reported weight was shown to strongly correlate with direct measurements[24], good mnemonic skills of the subjects were required and could have biased the reported data. Inability of the HIPSTRESS method to account for the size of the fovea and for the decrease in hip joint contact surface during walking leads to general underestimation of $p_{max}$ values[7], but without influence on their inter-relations. As our institution is the only referring facility for hip and back pain within the area, and as consecutively operated patients were selected for the study, the crude sample can be viewed as population-based. However, strict radiographic exclusion criteria could have biased the sample towards selection of younger subjects with fewer radiographic signs but more debilitating clinical signs of OA, which were unfortunately not assessed. In order to obtain sufficient sample size which is another limitation of our study, hips with early radiographic signs of OA were included. If we would include only healthy hips, we could have lengthened the time interval between pelvic radiographs taking and hip arthroplasty, but would have decreased the sample size even further.

By focusing on most constant body weight we minimized a negative effect that direct measurements at one specific time might have. Also, distinctive radiological requirements were followed in order to enable reliable HIPSTRESS model implementation. While determining peak contact hip stress from readily available pelvic radiographs presents an affordable, reliable and harmless way of acquiring appropriate data, its use in further clinical research is expected. Further strength of our study is that only women with idiopathic disease were included.

In conclusion, this study demonstrates that among women with idiopathic hip OA, younger age at hip arthroplasty is associated with enlarged contact hip stress — $p_{max}$ — with both obesity and unfavorable biomechanical constitution of the pelvis contributing. These data suggest that increased mechanical stress may have a deleterious role in progression of hip OA but further longitudinal studies are required to elucidate the role of contact stress in hip OA development.

Table II
*The relationship between age at hip arthroplasty and parameters of obesity, biomechanical constitution of the pelvis and mechanical loading within the hip joint (BMI — body mass index, $R$/Wb — resultant hip force normalized to the body weight, $p_{max}$/Wb — peak contact hip stress normalized to the body weight, $R$ — magnitude of the resultant hip force, $p_{max}$ — peak contact hip stress)*

| Independent variable | Standardized correlation coefficient $R$† | Un-standardized correlation coefficient‡ (95% CI) | P-value |
|---|---|---|---|
| *Univariate regression analysis* | | | |
| Obesity | | | |
| Body weight (kg) | −0.297 | −0.22 (−0.40, −0.04) | **0.016** |
| BMI (kg/m$^2$) | −0.194 | −0.40 (−0.90, −0.11) | 0.121 |
| Biomechanical constitution of the pelvis | | | |
| $R$/Wb | −0.064 | −2.6 (−12.9, 7.7) | 0.614 |
| $p_{max}$/Wb (kPa/N) | −0.264 | −3.8 (−7.3, −0.3) | **0.034** |
| Mechanical loading within the hip joint | | | |
| $R$ (kN) | −0.275 | −6.8 (−12.8, −0.8) | **0.027** |
| $p_{max}$ (MPa) | −0.401 | −7.2 (−11.4, −3.1) | **0.001** |
| *Bivariate regression analysis*§ | | | |
| Body weight (kg) | −0.312 | −0.23 (−0.40, −0.06) | **0.009** |
| $p_{max}$/Wb (kPa/N) | −0.281 | −4.0 (−7.4, −0.7) | **0.019** |

The bold values signify statistically significant findings.
†Level of correlation of the independent variable with age at hip arthroplasty.
‡Change in age at hip arthroplasty (years) per unit increase in independent variable.
§Model summary: $R^2 = 0.166$; adjusted $R^2 = 0.140$; P-value = 0.004.

## Conflict of interest

No financial or personal relationships with other people or organizations that could inappropriately influence (bias) our

work were engaged into by the authors or affiliated institutions.

## Acknowledgement

## References

1. Buckwalter JA, Martin JA. Osteoarthritis. Adv Drug Deliv Rev 2006;58: 150−67.
2. Gelber CA. Obesity and hip osteoarthritis: the weight of the evidence is increasing. Am J Med 2003;114:158−9.
3. Oliveria SA, Felson DT, Cirillo PA, Reed JI, Walker AM. Body weight, body mass index, and incident symptomatic osteoarthritis of the hand, hip, and knee. Epidemiology 1999;10:161−6.
4. Croft P, Coggon D, Cruddas M, Cooper C. Osteoarthritis of the hip: an occupational disease in farmers. BMJ 1992;304:1269−72.
5. Martin JA, Brown T, Heiner A, Buckwalter JA. Post-traumatic osteoarthritis: the role of accelerated chondrocyte senescence. Biorheology 2004;41:479−91.
6. Brinckmann P, Frobin W, Hierholzer E. Stress on the articular surface of the hip joint in healthy adults and persons with idiopathic osteoarthrosis of the hip joint. J Biomech 1981;14:149−56.
7. Ipavec M, Brand RA, Pedersen DR, Mavčič B, Kralj-Iglič V, Iglič A. Mathematical modelling of stress in the hip during gait. J Biomech 1999;32: 1229−35.
8. Vengust R, Daniel M, Antolič V, Zupanc O, Iglič A, Kralj-Iglič V. Biomechanical evaluation of hip joint after Salter innominate osteotomy: a long-term follow-up study. Arch Orthop Trauma Surg 2001;121:511−6.
9. Dolinar D, Antolič V, Herman S, Iglič A, Kralj-Iglič V, Pavlovčič V. Influence of contact hip stress on the outcome of surgical treatment of hips affected by avascular necrosis. Arch Orthop Trauma Surg 2003;123: 509−13.
10. Zupanc O, Antolič V, Iglič A, Jaklič A, Kralj-Iglič V, Stare J, *et al.* Assessment of the contact stress in the hip joint after operative treatment of severe slipped capital femoral epiphysis. Int Orthop 2001;25:9−12.
11. Recnik G, Kralj-Iglič V, Iglič A, Antolič V, Kramberger S, Vengust R. Higher peak contact hip stress predetermines the side of hip involved in idiopathic osteoarthritis. Clin Biomech 2007;22:1119−24.
12. Mavčič B, Slivnik T, Antolič V, Iglič A, Kralj-Iglič V. High contact hip stress is related to the development of hip pathology with increasing age. Clin Biomech 2004;19:939−43.
13. Brand RA, Iglič A, Kralj-Iglič V. Contact stresses in the human hip: implications for disease and treatment. Hip Int 2001;11:117−26.
14. Kersnič B, Iglič A, Kralj-Iglič V, Srakar F, Antolič V. Increased incidence of arthrosis in women could be related to femoral and pelvic shape. Arch Orthop Trauma Surg 1997;116:345−7.
15. Hadley NA, Brown TD, Weinstein SL. The effects of contact pressure elevations and aseptic necrosis on the long-term outcome of congenital hip dislocation. J Orthop Res 1990;8:504−13.
16. Mavčič B, Iglič A, Kralj-Iglič V, Brand RA, Vengust R. Cumulative hip contact stress predicts osteoarthritis in DDH. Clin Orthop Relat Res 2008;466:884−91.
17. Mose K. Methods of measuring in Legg-Calv-Perthes disease with special regard to the prognosis. Clin Orthop 1980;150:103−9.
18. Iglič A, Srakar F, Antolič V. Influence of the pelvic shape on the biomechanical status of the hip. Clin Biomech 1993;8:223−4.
19. Mavčič B. Predictive value of hip stress for development of hip osteoarthritis. PhD thesis. University of Ljubljana, Ljubljana, Slovenia, 2007.
20. Bergmann G, Deuretzbacher G, Heller M, Graichen F, Rohlmann A, Strauss J, *et al.* Hip contact forces and gait patterns from routine activities. J Biomech 2001;34:859−71.
21. Jensen LK. Hip osteoarthritis. Influence of work with heavy lifting, climbing stairs or ladders, or combining kneeling/squatting with heavy lifting. Occup Environ Med 2008;65:6−19.
22. Cymet TC, Sinkov V. Does long-distance running cause osteoarthritis? J Am Osteopath Assoc 2006;106:342−5.
23. Dreinhöfer KE, Dieppe P, Stürmer T, Gröber-Grätz D, Flören M, Günther KP, *et al.* Indications for total hip replacement: comparison of assessments of orthopaedic surgeons and referring physicians. Ann Rheum Dis 2006;65:1346−50.
24. Troy LM, Hunter DJ, Manson JE, Colditz GA, Stampfer MJ, Willet WC. The validity of recalled weight among younger women. Int J Obes Relat Metab Disord 1995;19:570−2.