Hip International / Vol. 11 no. 3, 2001 / pp. 117-126

Invited article

Contact stresses in the human hip: implications for disease and treatment

R.A. BRAND[1], A. IGLIČ[2], V. KRALJ-IGLIČ[3]

[1] Department of Orthopaedic Surgery, The University of Iowa, Iowa City, IA - USA
[2] Faculty of Electrical Engineering, University of Ljubljana, Ljubljana - Slovenia
[3] Institute of Biophysics, Faculty of Medicine, University of Ljubljana, Ljubljana - Slovenia

ABSTRACT: Contact stresses in the hip articular surfaces relate in some way to normal maintenance as well as destruction of joints. In vivo *determinations of cartilage-on-cartilage contact pressure histories have never been reported, and current technology does not allow such measurements without the potential for artifact: all experimental methods require introducing some material between the surfaces, and all numerical methods have yet to be fully validated. Nonetheless, a variety of distinct experimental and numerical approaches lead to estimates of contact stresses and surprisingly, despite the choice of technique, values for peak contact stresses lie within a range of one order of magnitude (i.e. 0.5-5.0 MPa) and usually closer. Pathological conditions increase this to the range of over 5.0 MPa, while surgical procedures designed to reduce peak pressures theoretically can achieve reductions. Two critical unresolved issues are 1.) What aspect of the contact stress history (e.g. contact stress gradients over time) in fact cause the biological responses? 2.) What level of contact stress history is tolerated by the cartilage? Future research will need to address these critical issues. (Hip International 2001; 11: 117-26)*

KEY WORDS: Hip, Contact stresses, Treatment

INTRODUCTION

Researchers have long known pressure affects cartilage. Heuter recognized the effects of pressure on the cartilage of the developing joint (1). The effects on mature cartilage were not well recognized until well into the 20th century, however, when the role of loading on the developing joint or osteoarthrosis was clearly established. Lovett in 1891 mentions a mechanical role primarily to suggest it was not important (2). Pemberton and Osgood, in their classic textbook from 1934, allude to the role of mechanics in osteoarthrosis in suggesting the importance of "carriage of the body," but they do not explicitly mention overloading of cartilage as critical (3), while two other authors (4,5) writing about the same time explicitly noted repeated mild trauma as causative, although neither explored this concept in any detail. Thus, serious attention to the role of mechanics in joint degeneration primarily arose after the mid 20th century. We now recognize contact stresses on articular surfaces substantially affect the durability of joints and their responses to treatment (6-8). Based upon epidemiological evidence, the responses appear to vary from joint to joint (9).

The hip joint appears particularly vulnerable to osteoarthrosis, and evidence suggests conditions which increase contact stresses also substantially reduce its durability (6, 10, 11). On the other hand, procedures to reduce contact stresses may improve longevity (12-16).

We will review the literature with respect to contact

1120-7000/117-10$05.00/0

stresses in the hip, including methods to estimate these contact stresses, and the results. We will then discuss the aspects of contact aberrations which might affect cartilage, and why. Finally, we will review reports of conditions which alter contact stresses and discuss the implications of treatment.

REVIEW OF LITERATURE

Methods to estimate contact stresses

The earliest methods to study joint contact stresses were static and semi-qualitative (17-19). Such models took into account statically estimated loads, approximate joint surface areas, determined average stresses, then estimated stress distribution onto a planar projection of a joint. While these approaches illustrated important concepts, they did not contribute materially to a knowledge of actual stress levels or distribution.

Remarkably, an experimental approach was described (although not used to collect data in a systematic way) before most of the qualitative descriptions of contact stresses. A small transducer would record pressure in only one location in a joint where a small region was excavated to hold the transducer (20). Later approaches ascertain contact areas, but not stresses (7, 21). One of the earlier methods utilized a sophisticated multichannel transducer at the surfaces of a hemiarthroplasty implanted in a living patient (22-24). Although an immense achievement for the time, these efforts and later ones by the same group (25, 26) were subject to a number of limitations: they could record pressures only in a limited number of discrete areas, the data reflected the stresses of cartilage-on-metal (rather than cartilage-on-cartilage), and the resulting pressure maps were questionable in the sense that not all channels were working. They reported no "integration" of the individual readings to ensure they could "recover" the full loads applied (and in any case, they would not have known the hip loads in the patients during various activities). Using multiple individual transducers conceptually similar to the one described by Ingelmark, two groups mapped contact stresses using an *in vitro* experiment in which applied loads could be controlled (27-29). However, with *in vitro* experimental methods the molecular structure of the glycoprotein layers on both contact surfaces in the hip joint and the lubrication of the intermediate thin fluid film are not fully preserved, therefore the measured contact stresses may differ from the natural ones.

Pressure sensitive films provided a conceptually new approach which had the advantage of providing spatially continuous measures of pressure, yet were subject to a number of problems (30-33). First, they inevitably require the introduction of some material between the articular surfaces, insuring some level of artifact; obviously, the thinner and more flexible the material the less potential artifact. Second, they were static in nature, so they only provided a "snapshot" of pressures. Third, they required tricky calibration procedures. In addition, the hemispherical nature of the hip results in the need for special treatment of what are essentially flat films. Semi-automated scanning of such films provides a more objective way to analyze the data (34). Furthermore, Brown et al developed special algorithms to remove crinkle artifact from scanned images of such pressure sensitive film used for the hip (35).

An even newer approach uses thin pressure sensitive mats which can be used for dynamic measurements (35-40). These have the distinct advantage of being able to record dynamic measurements over a period of time. While the devices have been largely used for external measurements, they have been applied to joints (41), although to the authors' knowledge, not reported for the hip joint. They are subject to some of the same problems as pressure sensitive films, but are typically thicker, and thus have greater potential to introduce measurement artifact. Further, the spherical nature of the hip would obviously create problems for flat sensors, while less so for essentially flat joints.

An entirely different approach is analytic, either numerical or using finite element models. Although complete credibility of these sorts of models demands validation against some independent standard (typically an experiment), such validation is rarely reported. However, the fact that many different sorts of models result in similar contact stresses constitutes a sort of consensual validation.

The numerical approaches were the first to be de-

scribed (42) and include a variety of approaches (14, 43-48). Such approaches typically involved a number of assumptions such as geometry (two dimensions in the earlier studies, or perfectly spherical femoral heads), a single loading condition, and frictionless and/or rigid surfaces. These are not, of course, inherent assumptions, for some (10, 15, 49-51) included three dimensions. Material properties of the cartilage can be incorporated (48) to eliminate the assumption of rigid surfaces, and of course multiple loading conditions can readily be simulated (6, 43, 45, 52, 53).

The earlier finite element models were two-dimensional (54, 55) but later reports included three-dimensional models (56, 57). These models can readily incorporate elements to account for the properties of both bone and cartilage, as well as varying the properties of each to simulate variations between patients. Furthermore, with contemporary computer hardware and software, solutions are quite tractable, even for very sophisticated meshes (geometry) and properties, not to mention loading (boundary) conditions.

While each approach has its own advantages and limitations, all of these have several common limitations. First, any contact stress distribution depends intimately upon loading conditions, which vary widely in the literature. Experimental approaches are usually limited to only a few loading conditions, and these may not be representative of the ranges observed *in vivo*. Furthermore, the contact stress recorded is highly dependent upon the constraints imposed by the loading apparatus on the joint and the configuration of the bones. Numerical approaches are perhaps not so limited in numbers of loading conditions, in that stresses in new conditions can be computed by changing a few input conditions in a program. However, even in that case, the numbers are limited. Second, the biological effects of stresses on or in tissues are likely related to the load history, not a given or even several loading conditions (58-63). Any load history is dependent not only upon peak magnitude, but also sub-peak magnitudes, numbers of cycles, timing of cycles, frequency, and duration. Which aspects of these characteristics are most important are unknown, but it is entirely possible that magnitude of stresses, that is the peak contact stresses we determine with the methods noted above, constitute a relatively unimportant aspect.

Estimates of contact stresses in activities of daily living

Not surprisingly, estimates of contact stresses in the literature vary considerably. One should not expect identical measures given distinct experimental or analytic methods, differing loading conditions, and even different reported parameters (e.g., most commonly spatially-averaged contact stresses or peak stresses). Nonetheless, it is rather amazing that the results fall with in an order of magnitude of each other (Tab. I). Most reports suggest average stresses in the range of 0.1 to 2.0 MPa while peak stresses range from about 2 to 10 MPa.

Patterns of stress distribution

Perhaps more important for cartilage longevity than the peak stresses in a given loading situation are the distributions of the stresses. Only a few authors report these patterns (27, 28, 64). These patterns are typically isometric “contour” maps with typically only one or perhaps two regions of high stress at a given instant. These high stress regions are limited to relatively small areas compared to the entire area, which means the spatially-averaged contact stresses are substantially less than the peaks. Furthermore, with a moving joint *in vivo* these high stress regions would not remain in the same region (43, 46). However, given that the resultant joint load varies its location considerably on the acetabulum with motion (65, 66), but remains in a relatively similar location on the femoral head with motion (67-72), it is obvious the location of the patterns of stress on the acetabulum would be much more variable than those on the femoral head. Normal hips have a more uniform contact stress distribution on the lateral side of the acetabular roof (45) reflected in equal thickness of bone condensation layer in this region of acetabular roof (42, 73). On the other hand, nonuniform contact stress distribution in dysplastic hips (45) leads to triangular shape of the bone condensation layer on the lateral side of the acetabular roof (42, 73). Several authors suggest, however, that high magnitude of the gradient of the contact stress distribution could be even more important than high magnitude of the contact stress (59, 74, 75).

TABLE I - SPATIALLY-AVERAGED AND PEAK CONTACT STRESSES IN NORMAL HIPS

Author/Year	Spatially-averaged contact stress (MPa)	Peak contact stress (MPa)
Legal 1977 (97)	0.1	
Rushfeld 1979 (64)		6.8*
Brown 1980 (98)		10
Brinckmann 1981 (6)	1.4-1.6	2.4-3.2
Brown 1983 (28)	2.9	8.8
Adams 1985 (29)		4.9-9.6
Hodge 1989 (26)		5.5*
Maxian 1995 (52)	<2.0	6-10
Tackson 1997 (99)	5.6*	
Tsumura 1998 (77)		2.5
Hak 1998 (76)		7.5-9.0
von Eisenhart 1999 (33)		7.7
Hipp 1999 (53)		2.1
Ipavec 1999 (45)		1.6-2.7
Iglič 2001 (100)		2.2 (male) 2.4 (female)

The estimates from the various authors involve a number of differing methods (see descriptions in text) and assumptions which readily influence the results; these numbers often reflect only a representative figure from a number reported. Thus, one should not attempt to directly compare the results, but rather get a sense of the range of pressures. Remarkably, despite widely varying methods and assumptions, most of the estimates are within an order of magnitude of each other. The references marked with an asterisk (*) arise from studies using an instrumented hemiarthroplasty; thus, they reflect peak pressures of a metal-implant against cartilage, which may not be representative of a situation with cartilage against cartilage.

Clinical conditions altering contact stresses

A number of clinical conditions alter the contact stresses in joints, most often by reducing the contact areas (e.g. dysplasia, malreduced acetabular fractures), but also by creating non-congruent surfaces (e.g. Legg-Calvé-Perthes disease). The degree to which these conditions increase stress has not been studied in great detail, and only by a few groups. However, it does appear that dysplasia or malreduced acetabular fractures do increase contact stresses by a figure of 2-3 times (compared to the normals given by individual authors).

The experimental study of Hak et al (76) predicted quite large normal peak contact stresses compared to others, and their experimental acetabular fracture resulted in acute increases of peak contact stresses of 2-3 times. Using a very different analytical approach, Tsumura et al reported similar values (77). Increases above normal in this range are in contrast to those reported by others in the knee where malreductions of a tibial fracture resulted in increases of contact stresses by less than one times normal (78), and large cartilage defects in the femoral condyle resulted in increases of only about 30% (75). They suggested the failure to have such large increases in the knee contact stresses related to the compliance of the cartilage; that is, as cartilage in a high area is loaded its high compliance causes it to compress, thus resulting in the opposing surface contacting adjacent areas for load bearing. Furthermore, in an *in vivo* one-year follow-up study (79) of articular defects using the same defects and measuring techniques (75), the remodeling of cartilage (and likely subchondral bone) resulted in no elevations of contact stresses over time. Thus, the high contact stresses seen in acute models of defects or malreduced fractures are not likely to persist over time, although the degree to which they will be reduced is probably case-specific.

Treatments altering contact stresses

The literature contains thousands of references to various operations (e.g., pelvic or femoral osteotomies) os-

TABLE II - PEAK CONTACT STRESSES IN ABNORMAL HIPS

Author/Year	Normal hips Peak contact stress (MPa)	Dysplastic hips Peak contact stress (MPa)	Dysplastic hips after osteotomy (MPa)	Slipped capital femoral epiphysis after osteotomy (MPa)	Malreduced acetabular fractures Peak contact stress (MPa)
Iglič 1993 (14)	1.2-2.7	3-6	1.2-2.0		
Michaeli 1997 (101)	5-8*	1-2.5*			
Hak 1998 (76)	7.5-9.0				6.0-20.5
Tsumura 1998 (77)	2.5	5.3			
Hipp 1999 (53)	2.1-5.0	2.6-6.5			
Zupanc 2001 (102)				1.1-4.3	
Mavcic 2000 (46, 103)	2.3	4.6			

The reader should again note these values reflect the methods and assumptions of the study in question, and more emphasis should be placed on relative, rather than absolute values. The higher values reported by Michaeli et al (indicated by asterisk) came from pressure sensitive films in a cadaveric pelvis, while the lower values for a "dysplastic" hip came from a plastic model in which the lateral lip was resected to simulate dysplasia.

tensibly reducing articular contact stresses. There is little doubt many of these operations in fact achieve at least a redistribution of loads and contact stresses, if not reduction in loads and stresses. However, relatively few authors substantively address the reductions in a quantitative manner (15, 42, 51, 53, 73, 80, 81).

After triple pelvic osteotomy the estimated peak contact stress can be reduced from an initial 3.0 MPa to less than 2.0 MPa by acetabular rotation over the femoral head in lateral direction without any shift of the femoral head center (14). The main reason for the reduction of peak contact stress is the increase of the weight bearing area on the lateral and medial portions of the acetabulum. A triple pelvic osteotomy that additionally displaces the femoral head centre medially reduces the peak contact stress to less than 1.5 MPa (14) due to the decrease of the hip joint resultant force (15). The initial location of the peak contact stress at the lateral acetabular rim in dysplastic hips is moved after the triple pelvic osteotomy away from the lateral acetabular rim in the medial direction (14). This is favorable since most of the degenerative changes in dysplastic hips usually occur at the lateral acetabular rim (18). The reported reduction of the contact stress after the acetabular reorientation in triple pelvic osteotomy is consistent with the results of other studies (42, 53, 73) which also demonstrated the potential of acetabular reorientation osteotomies to reduce the contact stress in dysplastic hips. However, the change in the pattern of the contact stress distribution (i.e. contact stress gradients) after different osteotomies could be even more important than the reduction of the contact stress itself. After triple pelvic osteotomy the initial preoperative nonuniform contact stress distribution on the lateral side of the acetabular roof becomes much more uniform (i.e. the stress gradient is reduced in this region) (51) which may be considered as a positive effect (42, 73).

Pauwels demonstrated certain varus-valgus positions of the osteoarthritic hip resulted in an increase in the radiographic joint shadow and presumed these increases would result in a reduced contact stress. He then advocated performing either a varus (PI) or valgus (PII) osteotomy that would result in "enlarging the area of the weight-bearing surface" (18). Using this qualitative approach, he reported many good results. In an effort to quantitatively explore that notion Miller et al, using a planar numerical model, demonstrated femoral osteotomies could increase the effective "joint space" and that the long term clinical outcome correlated with an increase in that joint space (81). While neither of these studies document actual reductions in hip contact pressures, they do indicate that methods designed to do so are clinically effective in many cases.

What aspects of joint contact affect durability?

As noted earlier, the load history and mechanical environment for all tissue consists in complex time- and spatially-varying loads (and contact stresses), dependent upon activity levels and choices of activities. We do not know which aspects of the load history result in tissue maintenance, formation, or destruction, although we do know some "physiological" loading is essential for normal tissue maintenance and other levels (below or above) result in tissue destruction or formation. Rubin and his colleagues in several papers have demonstrated that normal bone can be maintained or resorbed, or new bone can be formed depending upon the choice of load history under otherwise identical experimental conditions (82, 84). Robling et al have further shown that the amount of new bone formed in a different experimental model depends upon the "partitioning" of the loads given identical magnitudes and numbers of cycles (85). The analogous concept that dose, timing, and numbers of exposures to radiation – termed "dose-fractionation" – determine the tissue response has been well known in radiation since the mid 20th century (86). Thus, tissue responsiveness clearly relates to a variety of aspects of the mechanical environment over time.

Numerous studies document that the durability and tolerance of articular cartilage depends upon loading (87-93). Specific studies document the atrophy of cartilage under reduced loading (94), atrophy under excessive loading (95) and the maintenance under non-normal (moderate running) but presumably physiological conditions (89). Thus, there is probably some range of combinations of magnitude, frequency, duration, and partitioning ("duty cycle") of loads to which cartilage responds in one way or another, and some combination which defines "levels of tolerance." We do not know these levels. Until and unless we know which levels are not tolerable, it is impossible to ascertain whether manipulations which alter articular cartilage stresses are beneficial or deleterious.

Repo and Finlay suggested cartilage would tolerate only about 25 MPa of impact load, but most joints would never receive this level of loading except under conditions of an accident (96). We are aware of only one group that has addressed the tolerance of cartilage to elevated contact stresses over decades (10, 52). That group found the tolerance of cartilage under presumed high contact stress conditions (dysplasia) related not simply to some peak stress, but rather to a spatially-averaged stress over time (i.e., MPa-years). Specifically, they identified a level of spatially-averaged stress-time of 10 MPa-years as being likely to predict secondary hip degeneration. (The study implicitly assumed equal activity levels.) While at first glance that figure might be low (particularly given the estimates of static magnitude in Tab. I), one must keep in mind that during any activity, much of the cartilage experiences relatively low stresses (see section on Patterns of Loading), and thus a spatially-averaged stress accounts for both low and high regions. In other words, a spatial average will be much less than the peaks noted in the tables above. A more definitive identification of articular cartilage tolerance will need to await further studies, accounting for factors such as activity types and levels.

SUMMARY

Hip contact stresses have been studied using many experimental and numerical methods, and remarkably, the results are typically within an order of magnitude of each other, suggesting peak contact stresses are normally in the range of 2-10 MPa. Pathological conditions raise those levels to the range of 5-20 MPa. We do not know which specific aspect of the load or contact stress history affects the longevity of cartilage, nor do we have a clear idea of the tolerance of cartilage to stress over time. The only available estimates of tolerance are 10 MPa-years for spatially average (not peak) contact stresses. If these estimates are correct, and if a static contact stress measure (rather than some aspect of the contact stress history) is a surrogate for mechanical tolerance over time, then the difference between the anticipated and tolerated levels of stress and those which are deleterious is only 2-3 times, a relatively small "safety factor." However, it is premature to make that conclusion. We do not know which aspects of the mechanical history affect tissue responses and are uncertain as to the ranges of tolerable mechanical histories of the hip.

Reprint requests to:
Prof. Richard A. Brand, M.D.
Department of Orthopaedic Surgery
The University of Iowa Hospitals
1029 JPP
200 Hawkins Drive
Iowa City, IA 52242
USA
e-mail: dick-brand@uiowa.edu

REFERENCES

1. Heuter C. Klinik der gelenkkrankheiten mit einschluss der orthopädie: Auf anatomisch-physiologischen grundlagen nach klinischen beobachtungen für aerzte und studirende, studirende, 14- 16. Leipzig: Verlag von F.C. Wogel; 1876.
2. Lovett RW. The etiology, pathology, and treatment of diseases of the hip joint, 220. Boston: Geo. H. Ellis; 1891.
3. Pemberton R, Osgood RB. The medical and orthopaedic management of chronic arthritis, 403. New York: The Macmillan Company; 1934.
4. Hench PS. Acute and chronic arthritis. In: Ghormley RK, ed. Orthopaedic surgery. New York: William Wood and Co.; 1931.
5. Comroe BI. Arthritis and allied conditions. Philadelphia: Lea & Febiger; 1940.
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 (3): 149-56.
7. Bullough P, Goodfellow J, Greenwald AS, O'Connor J. Incongruent surfaces in the human hip joint. Nature 1968; 217: 1290.
8. Greenwald AS, Nelson CL, Jr. Relationship of degenerative arthritis to weight-bearing areas in the human hip joint. Surg Forum 1972; 23 (0): 463-4.
9. Felson DT. The epidemiology of osteoarthritis: Prevalence and risk factors. In: Kuettner KE, Goldberg VM, ed. Osteoarthritic disorders. Rosemont, IL: American Academy of Orthopaedic Surgeons; 1995; 13-24.
10. 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 (4): 504-13.
11. Olson SA, Bay BK, Hamel A. Biomechanics of the hip joint and the effects of fracture of the acetabulum. Clin Orthop 1997; 339: 92-104.
12. Siebenrock KA, Leunig M, Ganz R. Periacetabular osteotomy: the Bernese experience. Instr Course Lect 2001; 50: 239-45.
13. Leunig M, Siebenrock KA, Ganz R. Rationale of periacetabular osteotomy and background work. Instr Course Lect 2001; 50: 229-38.
14. Iglic A, Kralj-Iglic V, Antolic V, Srakar F, Stanic U. Effect of the periacetabular osteotomy on the stress on the human hip joint articular surface. IEEE Trans Rehab Eng 1993; 1 (4): 207-12.
15. Srakar F, Iglic A, Antolic V, Herman S. Computer simulation of periacetabular osteotomy. Acta Orthop Scand 1992; 63 (4): 411-2.
16. Vengust R, Daniel M, Antolic V, Zupanc O, Iglic A, Kralj-Iglic V. Biomechanical evaluation of hip joint after Salter innominate osteotomy: a long-term follow-up study. Arch Orthop Trauma Surg 2001; 121: 511-6.
17. Pauwels F. Die struktur der tangentialfaserschicht des gelenkknorpels der schulterpfanne als beispiel für ein verkörpertes spannungsfeld. Z Anat Entwicklungsgesch 1959; 121: 188-240.
18. Pauwels F. Biomechanics of the normal and diseased hip: Theoretical foundations, technique, and results of treatment, 276. Berlin: Springer-Verlag; 1976.
19. Maquet P. Le sourcil cotylidien, materialisation du diagramme des contraintes dans l'articulation de la hanche. Acta Orthop Belg 1974; 40: 150-65.
20. Ingelmark BE, Blomgren E. An apparatus for the measurement of pressure, especially in human joints. Ups J Med Sci 1948; 53: 53-75.
21. Greenwald AS, Haynes DW. Weight-bearing areas in the human hip joint. J Bone Joint Surg (Br) 1972; 54 (1): 157-63.
22. Carlson CE, Mann RW, Harris WH. A radio telemetry device for monitoring cartilage surface pressures in the human hip. IEEE Trans Biomed Eng 1974; 21 (4): 257-64.
23. Harris WH, Rushfeldt PD, Carlson CE, Scholler J-M, Mann RW. Pressure distribution in the hip and selection of hemiarthroplasty. In: Amstutz HC, ed. The Hip. St. Louis: The C.V. Mosby Company; 1975; 93-102.
24. Armstrong CG, Bahrani AS, Gardner DL. *In vitro* measurement of articular cartilage deformations in the intact human hip joint under load. J Bone Joint Surg (Am) 1979; 61 (5): 744-55.
25. Hodge WA, Fijan RS, Carlson KL, Burgess RG, Harris WH, Mann RW. Contact pressures in the human hip joint measured in vivo. Proc Natl Acad Sci USA 1986; 83 (9): 2879-83.
26. Hodge WA, Carlson KL, Fijan RS, Burgess RG, Riley PO, Harris WH, et al. Contact pressures from an instrumented hip endoprosthesis. J Bone Joint Surg (Am) 1989; 71 (9): 1378-86.

27. Brown TD, Shaw DT. A technique for measuring instantaneous *in vitro* contact stress distributions in articular joints. J Biomech 1982; 15 (4): 329-33.
28. Brown TD, Shaw DT. *In vitro* contact stress distributions in the natural human hip. J Biomech 1983; 16 (6): 373-84.
29. Adams D, Swanson SA. Direct measurement of local pressures in the cadaveric human hip joint during simulated level walking. Ann Rheum Dis 1985; 44 (10): 658-66.
30. Olson SA, Bay BK, Chapman MW, Sharkey NA. Biomechanical consequences of fracture and repair of the posterior wall of the acetabulum. J Bone Joint Surg (Am) 1995; 77 (8): 1184-92.
31. Afoke NY, Byers PD, Hutton WC. Contact pressures in the human hip joint. J Bone Joint Surg (Br) 1987; 69 (4): 536-41.
32. Bay BK, Hamel AJ, Olson SA, Sharkey NA. Statistically equivalent load and support conditions produce different hip joint contact pressures and periacetabular strains. J Biomech 1997; 30 (2): 193-6.
33. von Eisenhart R, Adam C, Steinlechner M, Muller-Gerbl M, Eckstein F. Quantitative determination of joint incongruity and pressure distribution during simulated gait and cartilage thickness in the human hip joint. J Orthop Res 1999; 17 (4): 532-9.
34. Caldwell NJ, Hale JE, Rudert MJ, Brown TD. An algorithm for approximate crinkle artifact compensation in pressure-sensitive film recordings. J Biomech 1993; 26 (8): 1001-9.
35. Garbalosa JC, Cavanagh PR, Wu G, Ulbrecht JS, Becker MB, Alexander IJ, et al. Foot function in diabetic patients after partial amputation. Foot Ankle Int 1996; 17 (1): 43-8.
36. Burgess S, Jordan C, Bartlett R. The influence of a small insert, in the footbed of a shoe, upon plantar pressure distribution. Clin Biomech 1997; 12 (3): S5-6.
37. Hayes A, Seitz P. Reproducibility test on a children's insole for measuring the dynamic plantar pressure distribution. Clin Biomech 1997; 12 (3): S4-5.
38. Hayes A, Seitz P. The average pressure distribution of the diabetic foot: can it be used as a clinical diagnostic aid? Clin Biomech 1997; 12 (3): S3-4.
39. Koch M. [Measuring plantar pressure in conventional shoes with the TEKSCAN sensory system]. Biomed Tech (Berl) 1993; 38 (10): 243-8.
40. Polliack AA, Sieh RC, Craig DD, Landsberger S, McNeil DR, Ayyappa E. Scientific validation of two commercial pressure sensor systems for prosthetic socket fit. Prosthet Orthot Int 2000; 24 (1): 63-73.
41. Cooper PS, Nowak MD, Shaer J. Calcaneocuboid joint pressures with lateral column lengthening (Evans) procedure. Foot Ankle Int 1997; 18 (4): 199-205.
42. Kummer B. Die Beanspruchung des menschlichek hüftgelenks I. Allgemeine Problematik. Z Anat Entwicklungsgesch 1968; 127 (4): 277-85.
43. Iglic A, Kralj-Iglic V, Antolic V. Reducing the stress in the articular surface of the hip joint after shifting the upper part of the body towards the painful hip. Acta Chir Orthop Traumatol Czech 1994; 61: 268-70.
44. Daniel M, Antolic V, Iglic A, Kralj-Iglic V. Determination of contact hip stress from nomograms based on mathematical model. Med Eng Phys 2001; 23 (5): 347-57.
45. Ipavec M, Brand RA, Pedersen DR, Mavcic B, Kralj-Iglic V, Iglic A. Mathematical modelling of stress in the hip during gait. J Biomech 1999; 32 (11): 1229-35.
46. Mavcic B, Antolic V, Brand R, Iglic A, Kralj-Iglic V, Pedersen DR. Peak contact stress in human hip during gait. Pflugers Arch 2000; 440 (5): R177-8.
47. Legal H, Reinecke M, Ruder H. Zur biostatischen analyse des hüftgelenks III. Z Orthop Ihre Grenzgeb 1980; 118 (5): 804-15.
48. Legal H. Introduction to the biomechanics of the hip. In: Tonnis D, ed. Congenital dysplasia and dislocation of the hip. Berlin: Springer-Verlag; 1987; 26-57.
49. Brinckmann P, Frobin W, Hierholzer E. [Pressure on the bearing surface of the hip joint (author's transl)]. Z Orthop Ihre Grenzgeb 1980; 118 (1): 107-15.
50. Ipavec M, Iglic A, Iglic VK, Srakar F. Stress distribution on the hip joint articular surface during gait. Pflugers Arch 1996; 431 (6): R275-6.
51. Iglic A, Antolic V, Srakar F. Biomechanical analysis of various operative hip joint rotation center shifts. Arch Orthop Trauma Surg 1993; 112 (3): 124-6.
52. Maxian TA, Brown TD, Weinstein SL. Chronic stress tolerance levels for human articular cartilage: two nonuniform contact models applied to long-term follow-up of CDH. J Biomech 1995; 28 (2): 159-66.
53. Hipp JA, Sugano N, Millis MB, Murphy SB. Planning acetabular redirection osteotomies based on joint contact pressures. Clin Orthop 1999; 364: 134-43.
54. Brown TD, DiGioia AM, 3rd. A contact-coupled finite element analysis of the natural adult hip. J Biomech 1984; 17 (6): 437-48.
55. Rapperport DJ, Carter DR, Schurman DJ. Contact finite element stress analysis of the hip joint. J Orthop Res 1985; 3 (4): 435-46.
56. Burton DS, Skinner HB, Eng M. Stress analysis of a total hip acetabular component: an FEM study. Biomater Artif Cells Artif Organs 1989; 17 (4): 371-83.
57. Maxian TA, Brown TD, Pedersen DR, Callaghan JJ. The Frank Stinchfield Award. 3-dimensional sliding/contact computational simulation of total hip wear. Clin Orthop 1996; 333: 41-50.
58. Brand RA. Autonomous informational stability in connective tissues. Med Hypotheses 1992; 37 (2): 107-14.

59. Brand RA, Stanford CM. How connective tissues temporally process mechanical stimuli. Med Hypotheses 1994; 42 (2): 99-104.
60. Weinans H, Huiskes R, Grootenboer HJ. The behavior of adaptive bone-remodeling simulation models. J Biomech 1992; 25 (12): 1425-41.
61. Beaupre GS, Orr TE, Carter DR. An approach for time-dependent bone modeling and remodeling- application: a preliminary remodeling simulation. J Orthop Res 1990; 8 (5): 662-70.
62. Carter DR. Mechanical loading histories and cortical bone remodeling. Calcif Tissue Int 1984; 36 (Suppl 1): S19-24.
63. Carter DR, Rapperport DJ, Fyhrie DP, Schurman DJ. Relation of coxarthrosis to stresses and morphogenesis. A finite element analysis. Acta Orthop Scand 1987; 58 (6): 611-9.
64. Rushfeld PD, Mann RW, Harris WH. Influence of cartilage geometry on the pressure distribution in the human hip joint. Science 1979; 204 (4391): 413-5.
65. Pedersen DR, Brand RA, Davy DT. Pelvic muscle and acetabular contact forces during gait. J Biomech 1997; 30 (9): 959-65.
66. Witte H, Eckstein F, Recknagel S. A calculation of the forces acting on the human acetabulum during walking. Based on *in vivo* force measurements, kinematic analysis and morphometry. Acta Anat 1997; 160 (4): 269-80.
67. Davy DT, Kotzar GM, Brown RH, Heiple KG, Goldberg VM, Heiple KG, Jr., et al. Telemetric force measurements across the hip after total arthroplasty. J Bone Joint Surg Am 1988; 70 (1): 45-50.
68. Kotzar GM, Davy DT, Goldberg VM, Heiple KG, Berilla J, Heiple KG, Jr., et al. Telemeterized *in vivo* hip joint force data: a report on two patients after total hip surgery. J Orthop Res 1991; 9 (5): 621-33.
69. Kotzar GM, Davy DT, Berilla J, Goldberg VM. Torsional loads in the early postoperative period following total hip replacement. J Orthop Res 1995; 13 (6): 945-55.
70. Bergmann G, Graichen F, Rohlmann A. Is staircase walking a risk for the fixation of hip implants? J Biomech 1995; 28(5): 535-53.
71. Bergmann G, Graichen F, Rohlmann A, Linke H. Hip joint forces during load carrying. Clin Orthop 1997; 335: 190-201.
72. 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 (7): 859-71.
73. Kummer B. Is the Pauwels' theory of hip biomechanics still valid? A critical analysis, based on modern methods. Anat Anz 1993; 175 (3): 203-10.
74. Brand RA. Hip osteotomies: A biomechanical consideration. J Am Acad Orthop Surg 1997; 5 (5): 282-91.
75. Brown TD, Pope DF, Hale JE, Buckwalter JA, Brand RA. Effects of osteochondral defect size on cartilage contact stress. J Orthop Res 1991; 9 (4): 559-67.
76. Hak DJ, Hamel AJ, Bay BK, Sharkey NA, Olson SA. Consequences of transverse acetabular fracture malreduction on load transmission across the hip joint. J Orthop Trauma 1998; 12 (2): 90-100.
77. Tsumura H, Miura H, Iwamoto Y. Three-dimensional pressure distribution of the human hip joint - comparison between normal hips and dysplastic hips. Fukuoka Igaku Zasshi 1998; 89 (4): 109-18.
78. Brown TD, Anderson DD, Nepola JV, Singerman RJ, Pedersen DR, Brand RA. Contact stress aberrations following imprecise reduction of simple tibial plateau fractures. J Orthop Res 1988; 6 (6): 851-62.
79. Nelson BH, Anderson DD, Brand RA, Brown TD. Effect of osteochondral defects on articular cartilage. Contact pressures studied in dog knees. Acta Orthop Scand 1988; 59 (5): 574-9.
80. Kummer B. Die klinische relevanz biomechanischer analysen der huftregion. Z Orthop Ihre Grenzgeb 1991; 129 (4): 285-94.
81. Miller JA, Jr., Brand RA, Andrews JG. Quantifying osteoarthrotic hip incongruence. An approach to optimizing osteotomies. Clin Orthop 1997; 343: 124-34.
82. Rubin CT, Lanyon LE. Regulation of bone formation by applied dynamic loads. J Bone Joint Surg (Am) 1984; 66 (3): 397-402.
83. Rubin CT, McLeod KJ. Promotion of bony ingrowth by frequency-specific, low-amplitude mechanical strain. Clin Orthop 1994; 298: 165-74.
84. Rubin C, Gross T, Qin YX, Fritton S, Guilak F, McLeod K. Differentiation of the bone-tissue remodeling response to axial and torsional loading in the Turkey ulna. J Bone Joint Surg Am 1996; 78 (10): 1523-33.
85. Robling AG, Burr DB, Turner CH. Partitioning a daily mechanical stimulus into discrete loading bouts improves the osteogenic response to loading. J Bone Miner Res 2000; 15 (8): 1596-602.
86. Hall EJ. Radiobiology for the radiologist. Philadelphia: J.B. Lippincott Company; 1994.
87. Jurvelin J, Kiviranta I, Tammi M, Helminen HJ. Effect of physical exercise on indentation stiffness of articular cartilage in the canine knee. Int J Sports Med 1986; 7 (2): 106-10.
88. Jurvelin J, Kiviranta I, Tammi M, Helminen JH. Softening of canine articular cartilage after immobilization of the knee joint. Clin Orthop 1986; 207: 246-52.
89. Kiviranta I, Tammi M, Jurvelin J, Saamanen AM, Helminen HJ. Moderate running exercise augments glycosaminoglycans and thickness of articular cartilage

in the knee joint of young beagle dogs. J Orthop Res 1988; 6 (2): 188-95.
90. Kiraly K, Hyttinen MM, Parkkinen JJ, Arokoski JA, Lapvetelainen T, Torronen K, et al. Articular cartilage collagen birefringence is altered concurrent with changes in proteoglycan synthesis during dynamic *in vitro* loading. Anat Rec 1998; 251 (1): 28-36.
91. Haapala J, Arokoski JP, Ronkko S, Agren U, Kosma VM, Lohmander LS, et al. Decline after immobilisation and recovery after remobilisation of synovial fluid IL1, TIMP, and chondroitin sulphate levels in young beagle dogs. Ann Rheum Dis 2001; 60 (1): 55-60.
92. Haapala J, Arokoski J, Pirttimaki J, Lyyra T, Jurvelin J, Tammi M, et al. Incomplete restoration of immobilization induced softening of young beagle knee articular cartilage after 50-week remobilization. Int J Sports Med 2000; 21 (1): 76-81.
93. Haapala J, Arokoski JP, Hyttinen MM, Lammi M, Tammi M, Kovanen V, et al. Remobilization does not fully restore immobilization induced articular cartilage atrophy. Clin Orthop 1999; 362: 218-29.
94. Benichou C, Wirotius JM. Articular cartilage atrophy in lower limb amputees. Arthritis Rheum 1982; 25 (1): 80-2.
95. Arokoski J, Kiviranta I, Jurvelin J, Tammi M, Helminen HJ. Long-distance running causes site-dependent decrease of cartilage glycosaminoglycan content in the knee joints of beagle dogs. Arthritis Rheum 1993; 36 (10): 1451-9.
96. Repo RU, Finlay JB. Survival of articular cartilage after controlled impact. J Bone Joint Surg (Am) 1977; 59 (8): 1068-76.
97. Legal H, Ruder H. Zur biostatischen Analyse des hüftgelenks I. Z Orthop Ihre Grenzgeb 1977; 115 (2): 215-34.
98. Brown TD, Ferguson AB, Jr. The effects of hip contact aberrations on stress patterns within the human femoral head. Ann Biomed Eng 1980; 8 (1): 75-92.
99. Tackson SJ, Krebs DE, Harris BA. Acetabular pressures during hip arthritis exercises. Arthritis Care Res 1997; 10 (5): 308-19.
100. Iglic A, Daniel M, Kralj-Iglic V, Antolic V, Jaklic A. Peak hip-joint contact stress in male and female populations. J Musculoskel Res 2001; 5 (1): 17-21.
101. Michaeli DA, Murphy SB, Hipp JA. Comparison of predicted and measured contact pressures in normal and dysplastic hips. Med Eng Phys 1997; 19 (2): 180-6.
102. Zupanc O, Antolic V, Iglic A, Jaklic A, Kralj-Iglic V, Stare J, et al. The assessment of contact stress in the hip joint after operative treatment for severe slipped capital femoral epiphysis. Int Orthop 2001; 25 (1): 9-12.
103. Mavcic B, Antolic V, Brand R, Iglic A, Ipavec M, Kralj-Iglic V, et al. Weight bearing area during gait in normal and dysplastic hips. Pflugers Arch 2000; 439 (3): R213-4.