

# Contact Stress in Hips with Osteonecrosis of the Femoral Head

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**Contact stress distribution in the articular surface of the hip is considered a factor in the development of osteoarthritis, a common complication in hips with aseptic necrosis of the femoral head. We present evidence supporting the hypothesis that osteoarthritis in hips with aseptic necrosis of the femoral head can be caused by elevated contact stress related to the reduced load-bearing ability of the necrotic bone. By using a previously validated mathematical model, we observed that hip contact stress may increase considerably if the load-bearing capacity of the necrotic lesion is decreased, if the size of the necrotic segment is increased, and if the necrotic segment is located more laterally. These effects are affected by the intrinsic shape of the hip. As the estimated values of stress in hips with osteonecrosis are in the range obtained by the same method in dysplastic hips, osteoarthritis in hips with osteonecrosis can be caused by elevated contact stress.**

Osteonecrosis of the femoral head is a relatively common disorder of the human hip.<sup>24</sup> It is characterized by deterioration of the bone tissue ostensibly related to disruption of the blood supply to the diseased region of the bone.<sup>1</sup> The structural properties of the bone change as a result of repair and resorption,<sup>15</sup> so its ability to bear a load is reduced with respect to the healthy hip.<sup>8,41</sup> Fractures in the

necrotic part may occur, and ultimately, the bone collapses.<sup>20</sup> Following osteonecrosis, osteoarthritis (OA) of the hip is likely to develop.<sup>20,21</sup>

Because the mechanical properties of the hip are affected by osteonecrosis of the femoral head,<sup>8</sup> they have been the subject of numerous theoretical studies. These include two-dimensional linear, three-dimensional linear, and nonlinear finite element methods.<sup>7,8,11,39,41</sup> The results of the finite element method analyses indicate that the stress pattern in the femoral head with osteonecrosis is substantially different from that in the healthy femoral head, with particular sensitivity to the size and shape of the lesion and to its location in the load-bearing area,<sup>14,41</sup> and that the decreased strength of the necrotic bone segment may contribute to fractures in the necrotic bone.<sup>7,10,11,41</sup> Previous studies based on finite element methods focused mainly on the stress distribution in the femoral head bone, whereas stress distribution in the cartilage was neglected<sup>41</sup> or considered a fixed-input parameter at the boundary defined by the articular surface.<sup>7,10,12</sup>

Numerous authors have suggested that elevated articular contact stress acting on the cartilage is one of the major reasons for development of OA in various anomalies of the hip.<sup>2,4,5,13,22,32,34</sup> Because OA also is a common complication secondary to osteonecrosis of the femoral head,<sup>20,21</sup> we propose OA in hips with aseptic necrosis of the femoral head can be caused by the elevated contact stress arising from changes in the ability of necrotic bone to bear a load. Because changes in the load-bearing area of the hip may have a complex effect on the stress-distribution pattern,<sup>17,23</sup> a detailed analysis is necessary to estimate the importance of the change in the weightbearing area for stress distribution in the hip.

We tested the above hypothesis by performing such an analysis using a mathematical model. We studied how the load-bearing capacity, size and position, and the intrinsic shape of the hip and pelvis influence contact stress of the hip.

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**MATERIALS AND METHODS**

To determine the contact stress distribution in a hip with a necrotic segment, we used a previously developed three-dimensional mathematical model of the one-legged stance.<sup>26–28</sup> The model was verified in population studies involving clinical evaluation<sup>19,32,33,35</sup> and the effect of different operations on contact stress distribution.<sup>23,40,42</sup> In the model, one-legged stance is considered a representative body position for hip loading.<sup>16</sup> Assuming the equilibrium of forces and torques acting on the pelvis, it was found that the hip reaction force lies almost in the frontal plane of the body in the one-legged stance,<sup>26</sup> so that it can be expressed as  $\mathbf{R} = (R \sin\vartheta_R, 0, R \cos\vartheta_R)$  where  $R$  is the magnitude and  $\vartheta_R$  is the inclination of the force  $\mathbf{R}$  with respect to the superior direction (Fig 1).

We assumed the acetabulum and the femoral head are spherical, and separated by a cartilage layer of constant thickness. When unloaded, the femoral head and the acetabulum are concentric. During loading, the femoral head is moved toward the acetabulum and the cartilage is squeezed. The spherical surfaces of the acetabulum and the femoral head reach the minimum separation at a point on the articular surface that is called the stress pole (P).<sup>6</sup> It is assumed that the hip is well lubricated, so that tangential stress is negligible compared with radial stress. The cosine stress distribution function  $p = p_0 \cos \gamma$  was adopted from Brinckmann et al.<sup>6</sup> The value  $p_0$  is the stress at the pole, and

$\gamma$  is the angle between the vector from the center of the femoral head to the pole and the vector from the center of the femoral head to the given point.

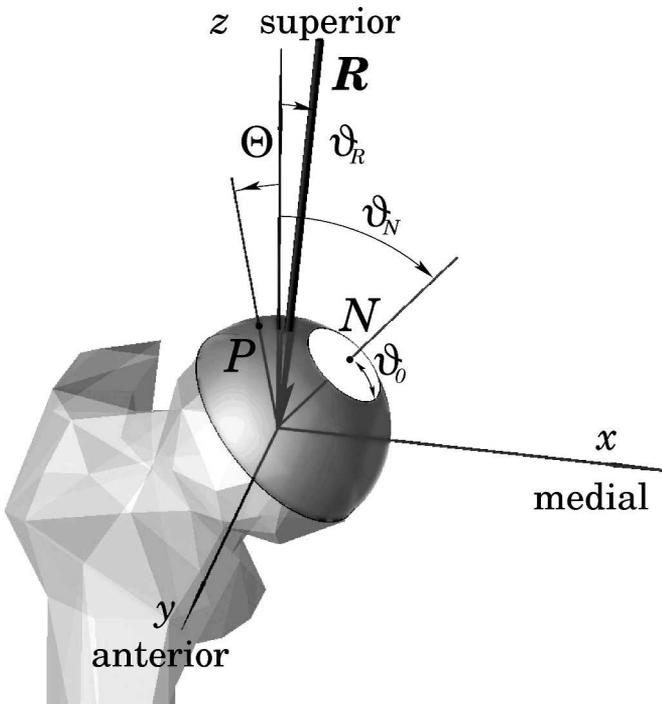
The contact stress was related to the resultant hip force by:

$$\mathbf{R} = \int_A p d\mathbf{S} \tag{1}$$

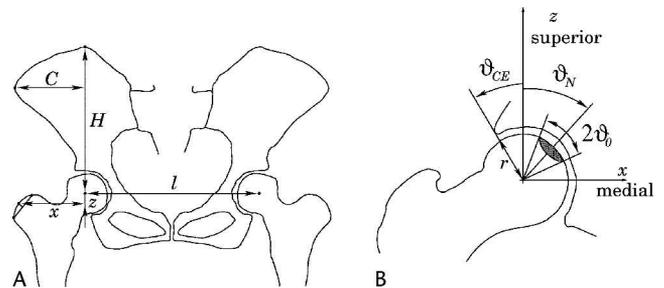
where  $A$  is the load-bearing area (ie, the part of the articular surface that bears the load and  $d\mathbf{S}$  is the area element). In an intact hip, the load-bearing area is bounded by the acetabular rim and by the condition of vanishing stress ( $\cos \gamma = 0$ ). If the frontal plane is the plane of symmetry of the articular surface (Fig 1), the pole must lie in this plane to fulfill Equation 1. The position of the stress pole (P) in the frontal plane was denoted by the angle  $\Theta$  which was taken to be positive in the lateral direction and negative in the medial direction with respect to the sagittal plane passing through the center of the femoral head.

We took the specific configuration of the load-bearing area attributable to necrosis into account. The necrotic lesion of the femoral head is represented by an area of bone with decreased stiffness.<sup>15,30,41</sup> We assumed the articular surface corresponding to the necrotic part had a decreased load-bearing capacity  $n$  ( $0\% \leq n \leq 100\%$ ), where  $n = 100\%$  refers to the full load-bearing capacity of an intact femoral head and  $n = 0\%$  corresponds to the nonload-bearing area. The parameter  $n$  reflected changes in the mechanical properties of the necrotic bone and the corresponding cartilage.

To maintain the symmetry of the articular surface with respect to the frontal plane, the shape of the necrotic segment was described by a cone with its axis lying in the frontal plane and defined by the angle  $\vartheta_N$  (Fig 1) that was denoted as positive if the center of the necrotic region is located medially from the sagittal plane passing through the center of the femoral head and negative if the center of the necrotic region is located laterally (Figs 1, 2). The size of the cone was defined by the angle  $\vartheta_0$ . The definition of the shape of the necrotic segment was the same as in previous works,<sup>11,14,41</sup> while  $\vartheta_0$  represents  $\frac{1}{2}$  of the arc angle corresponding to the necrotic segment.<sup>31</sup> The cone of necrotic bone intercepts the spherical articular surface, which defines an



**Fig 1.** A schematic representation of the articular surface with a necrotic lesion (white) is shown.  $\mathbf{R}$  denotes the resultant hip force, P denotes the position of the stress pole, and N denotes the position of the center of the articular surface area corresponding to the necrotic segment.



**Fig 2A–B.** (A) A diagram shows the characteristic parameters of the pelvis and proximal femur used to adjust the model for calculation of the reaction force of the hip. (B) This diagram shows the parameters of the shapes of the hip and of the osteonecrosis of the femoral head needed to determine the contact stress distribution.

area ( $A_N$ ) of the articular surface corresponding to the necrotic segment. We assumed the whole area corresponding to the necrotic lesion lies inside the load-bearing area.

The integration in Equation 1 was performed over the load-bearing area where the region corresponding to the necrotic segment was taken into account:

$$\mathbf{R} = \int_A p d\mathbf{S} - \int_{A_N} p d\mathbf{S} \quad (2)$$

The first integration was performed over the intact load-bearing area  $A$ , whereas the second integration was performed over the area  $A_N$  of the articular surface corresponding to the necrotic segment (Fig 1). The second term in Equation 2 is equal to the force that is not transmitted because of the decreased load-bearing capacity of the necrotic lesion.

Equation 2 was solved numerically by using the downhill simplex method<sup>36</sup> to determine the spherical coordinate of the stress pole ( $\Theta$ ) and the value of stress at the pole ( $p_0$ ). If the stress pole lay inside the load-bearing area, then the point of the peak contact stress coincides with the stress pole ( $p_{\max} = p_0$ ). If the pole lay outside the load-bearing area, then the point of maximum stress is the point of the load-bearing area closest to the stress pole, and  $p_{\max}$  at this point was calculated according to the cosine stress distribution function.<sup>6</sup>

Definition of the necrotic lesion by three parameters (load-bearing capacity ( $n$ ), mediolateral position in the frontal plane ( $\vartheta_N$ ), and size ( $\vartheta_0$ ) allowed us to study the individual effects of these parameters on the contact stress distribution. All results were calculated for the same magnitude and direction of the resultant hip force ( $R/W_B = 2.7$ ,  $\vartheta_R = 5^\circ$ ) and the same radius of the articular surface  $r = 2.7$  cm. These are values that commonly are found in healthy hips.<sup>29,32</sup>

Contact stress distribution of the hip also is influenced by the global shape of the hip.<sup>16,26,32</sup> The model was developed so that it could be adjusted for an individual patient by considering the characteristic shape of the pelvis, the proximal femur, and the patient's body weight ( $W_B$  [N]). The model for determining the force  $\mathbf{R}$  in a one-legged stance takes into account nine effective muscles.<sup>26</sup> The coordinates of their attachments were adjusted to each patient according to the shape of the pelvis and the proximal femur obtained from standard anteroposterior (AP) radiographs (Fig 2). The input parameters are the width of the pelvis ( $C$ ), the height of the pelvis ( $H$ ), the horizontal and vertical positions of the greater trochanter ( $x$  and  $z$ , respectively), and the interhip distance ( $l$ ) (Fig 2). After assessing the shape of the hip and pelvis, the reaction force of the hip was determined by assuming equilibrium of forces and torques acting on the pelvis in the one-legged stance.<sup>16,26</sup> To determine the stress distribution one must know the reaction force ( $\mathbf{R}$ ) of the hip, the radius of the articular surface ( $r$ ) defined as the mean of the radii of the acetabulum and of the femoral head, and the position of the lateral rim of the acetabulum obtained by intersection of the articular surface with a plane inclined by the center-edge angle of Wiberg ( $\vartheta_{CE}$ ) with respect to the sagittal plane (Fig 2B). According to the mathematical model, the shape of the necrotic segment was approximated by a spherical cone (Fig 1), whereas the size and location of the necrotic lesion was estimated from

the AP radiographs by two angles determining the most lateral and the most medial point of the necrotic part ( $\vartheta_L$  and  $\vartheta_M$ ) (Fig 2B). The position of the center of the necrosis ( $\vartheta_N$ ) is approximated by the mean of these two angles, and the size of the necrosis ( $\vartheta_0$ ) was defined as the angular distance between the center and  $\vartheta_L$  or  $\vartheta_M$  (Fig 2B). The values of  $R$ ,  $\vartheta_R$ ,  $p_{\max}$ , and  $\Theta$  were obtained by using the HIPSTRESS computer program<sup>26</sup> adjusted for the specific shape of the load-bearing area containing the necrotic segment. The peak contact stress and the magnitude of the resultant were normalized to the body weight ( $W_B$ ), ( $p_{\max}/W_B$  and  $R/W_B$ , respectively).

To show hip and pelvis shape influences the effects of the necrotic lesion on hip stress, we present two specific cases (Hips A and B). Hip B has a wider and larger pelvis and a smaller greater trochanter than Hip A; the lateral coverage  $\vartheta_{CE}$  and the radius of the femoral head are smaller in Hip B than in Hip A. The differences in geometric parameters were rather small so that in intact hips, the peak stress values were within the range of values found in healthy female hips<sup>23,32</sup> [ $p_{\max}/W_B < 3500$  m<sup>-2</sup>]. To compare the effect of the intrinsic shape, the peak stress was determined in both hips, when intact and after necrosis had developed (Table 1).

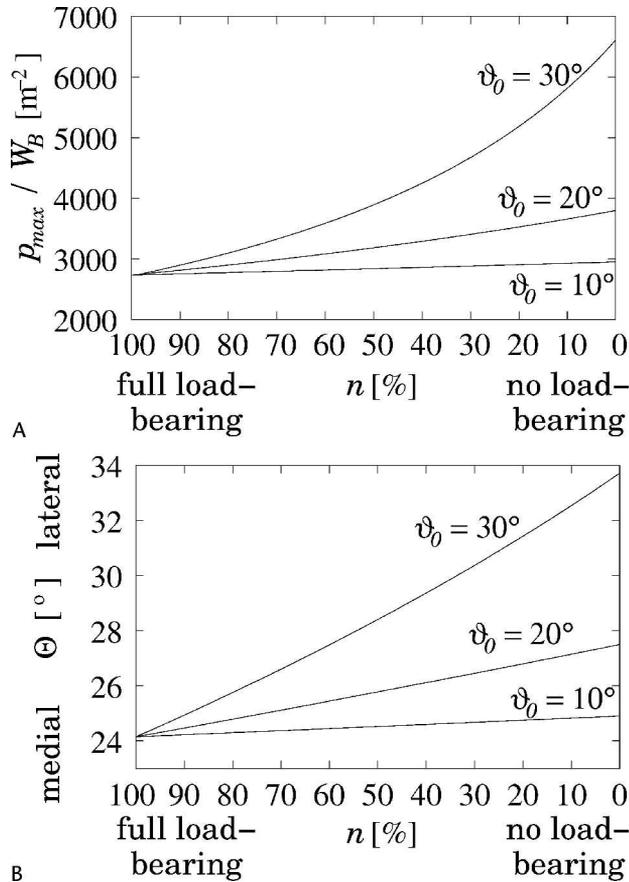
## RESULTS

Reducing the load-bearing capacity of the necrotic lesion increased the peak contact stress (Fig 3A) and shifted the stress pole laterally (Fig 3B). The effect was more pronounced for larger necrotic segments, which had as much as a 200% increase of stress compared with a normal hip. If the articular surface included a region that was unable to transmit load ( $n = 0\%$ ), the peak stress was greatest. For

**TABLE 1. Geometric Parameters and Calculated Biomechanical Parameters in Two Imaginary Hips (A and B)**

Category	Symbol (unit)	Hip A	Hip B
Position of the trochanter	$x$ (mm)	70.0	60.0
	$z$ (mm)	5.0	10.0
Shape of the pelvis	$l$ (mm)	180.00	190.0
	$C$ (mm)	40.0	60.0
	$H$ (mm)	140.0	160.0
Shape of the hip	$r$ (mm)	27.0	26.0
	$\vartheta_{CE}$ ( $^\circ$ )	35	30
Shape of the avascular necrosis	$\vartheta_N$ ( $^\circ$ )	10	10
	$\vartheta_0$ ( $^\circ$ )	20	20
Resultant hip force	$R/W_B$	2.37	2.73
	$\vartheta_R$ ( $^\circ$ )	11.8	6.8
Stress without necrosis ( $n = 100\%$ )	$p_{\max}/W_B$ (m <sup>-2</sup> )	1848	2826
	$\Theta$ ( $^\circ$ )	4.5	20.0
Stress with necrosis ( $n = 0\%$ )	$p_{\max}/W_B$ (m <sup>-2</sup> )	2845	4833
	$\Theta$ ( $^\circ$ )	11.1	30.8

\*The biomechanical parameters are calculated for a situation with and without avascular necrosis of the femoral head.

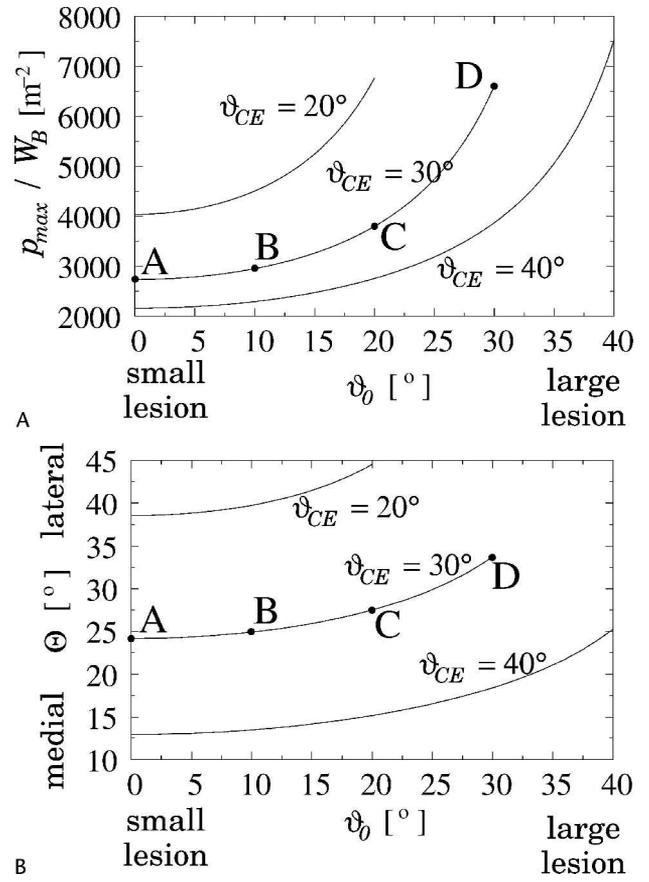


**Fig 3A–B.** The charts show the (A) relative peak contact stress  $p_{max}/W_B$  and (B) position of the stress pole  $\Theta$  as a function of the load-bearing capacity of the necrotic segment ( $n$ ) for different sizes of the necrotic segment ( $\vartheta_0$ ). The center of the articular area corresponding to the necrotic segment is located on top of the articular surface ( $\vartheta_N = 0^\circ$ ), the force  $\mathbf{R}$  lies in the frontal plane ( $R/W_B = 2.7$ ,  $\vartheta_R = 5^\circ$ ), and the radius of the articular sphere is 2.7 cm. The dependence presented simulates the development of the necrotic process by changing the load-bearing capacity of the articular surface area corresponding to the necrotic segment ( $n$ ).

clarity and simplicity, we consider a limiting case, where the necrotic part bears no load ( $n = 0\%$ ).

Increasing the size of the lesion increased the peak stress (Fig 4A) while the pole was shifted laterally (Fig 4B). The effect depended on the shape of the hip. If the hip had poor lateral coverage ( $\vartheta_{CE} = 20^\circ$ ),  $p_{max}/W_B$  reached the value of 6500 m<sup>-2</sup> for a necrotic lesion of 20° size, whereas if the lateral coverage was larger ( $\vartheta_{CE} = 40^\circ$ ), the necrotic lesion had to have a size of 38° to reach the same value of  $p_{max}/W_B$ .

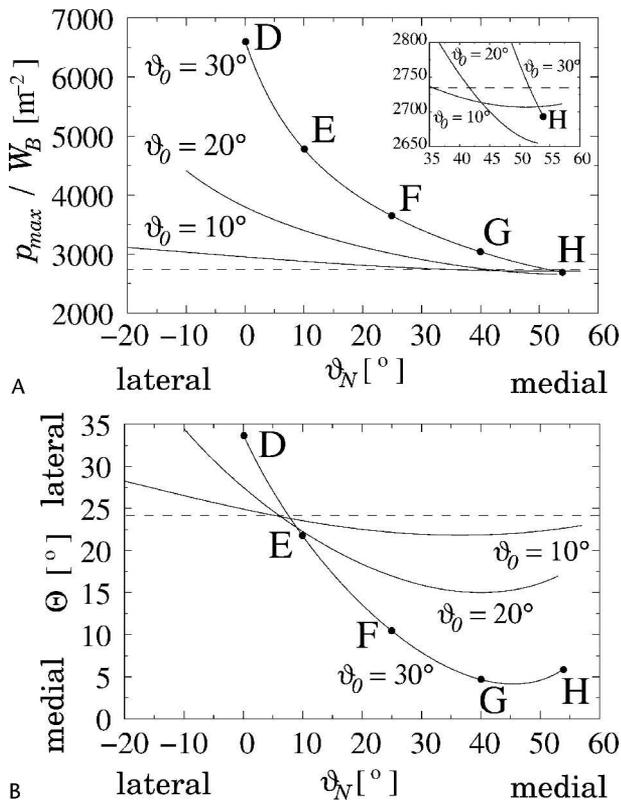
Moving the lesion medially reduced  $p_{max}/W_B$  (Fig 5A). The most medially located necrotic segment reduced the peak contact stress below the corresponding value ob-



**Fig 4A–B.** The charts show the effect of the size of the articular area corresponding to the necrotic segment ( $\vartheta_0$ ) (A) on the relative peak contact stress  $p_{max}/W_B$  and (B) on the position of the stress pole  $\Theta$  for different lateral coverage of the acetabulum  $\vartheta_{CE}$ . The center of the articular area corresponding to the necrotic segment is located on top of the articular surface ( $\vartheta_N = 0^\circ$ ), the resultant hip force  $\mathbf{R}$  lies in the frontal plane ( $R/W_B = 2.7$ ,  $\vartheta_R = 5^\circ$ ), and the radius of the articular sphere is 2.7 cm.

tained for an intact hip (Fig 5). However, the reduction was small. With medial shifting of the nonload-bearing area, the pole first was moved medially with respect to the position in the intact hip. On additional shifting of the nonload-bearing area medially, the position of the pole was moved to a slightly less medial position (Fig 5B). The influence of the size (Fig 6A–D) and position (Fig 6D–H) of the necrotic region on the distribution of stress over the load-bearing area for the selected sizes and positions was complex (Figs 4, 5).

If necrotic lesions of the same size and location developed in two hips which differed in the shape of the pelvis and the upper femur (Table 1), the increase in the peak stress differed considerably. The increase in the peak con-



**Fig 5A–B.** The charts show the (A) effect of the mediolateral position of the articular area corresponding to the necrotic segment ( $\vartheta_N$ ) (A) on the relative peak contact stress  $p_{max}/W_B$ , and (B) on the position of the stress pole  $\Theta$  for different sizes of the articular area corresponding to the necrotic segment  $\vartheta_0$ . The dashed lines show the corresponding values of  $p_{max}/W_B$  and  $\Theta$  in an intact hip. The resultant hip force  $R$  lies in the frontal plane ( $R/W_B = 2.7$ ,  $\vartheta_R = 5^\circ$ ), and the radius of the articular sphere is 2.7 cm.

tact stress in Hip B was almost twice the increase in Hip A. Considering the value of  $p_{max}/W_B = 3500 \text{ m}^{-2}$  as the stress level in healthy female hips,<sup>33</sup> the peak contact stress would stay normal in Hip A, whereas in Hip B it would be elevated. In Hip B, the additional increase from the presence of the nonload-bearing area might cause it to be at high risk for OA development. In Hip A, this risk would be considerably lower, owing to the more favorable shape of the hip and pelvis.

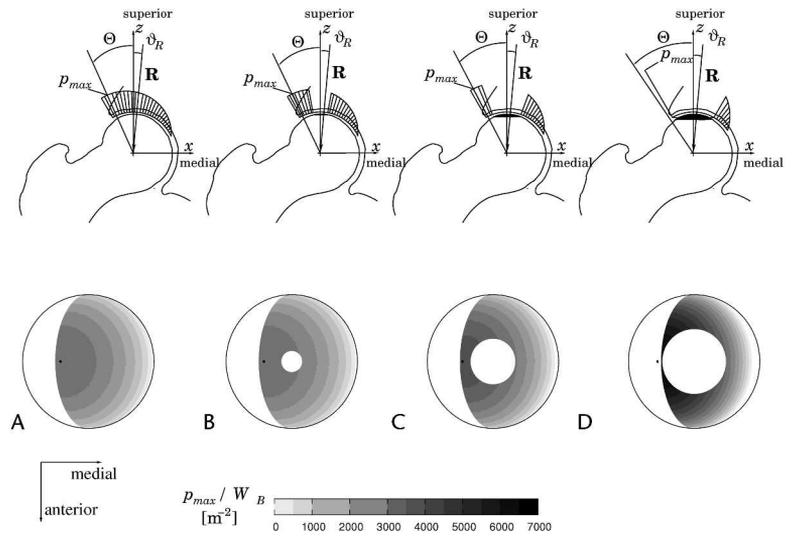
## DISCUSSION

Contact stress distribution in the articular surface of the hip is considered an important factor in the development of OA.<sup>22</sup> We have postulated that elevated hip stress is also the cause of OA in hips with necrosis of the femoral head.

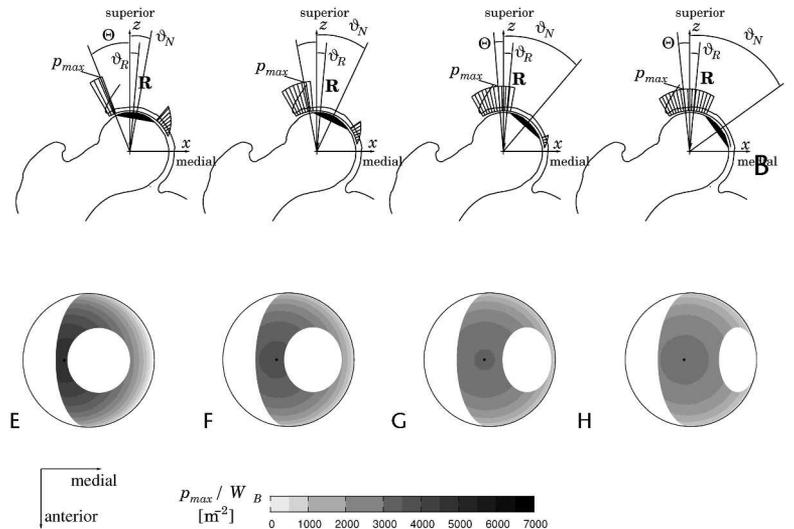
To test this hypothesis, we performed an analysis of possible clinical cases using a three-dimensional mathematical model of the contact stress distribution. The parametric definition of the model allowed study of the effect of load-bearing capacity, size, and position of the necrotic lesion, and shape of the hip and pelvis on contact stress distribution. It was found that formation of the necrotic lesion may considerably increase the peak contact stress in the hip (Figs 3–5).

Our model has certain limits because of its simplicity. First, the stress distribution function is based on the assumption that the cartilage ideally is elastic, uniform in structure, and that its thickness is constant.<sup>6</sup> However, articular cartilage is a complex permeable viscoelastic structure that has a site and depth dependence of its biomechanical properties.<sup>38</sup> This is especially notable in hips with osteonecrosis in which the necrotic segment has collapsed. However, the intraindividual variations of the thickness of the cartilage (1.4–2.4 mm)<sup>38</sup> and of the compressive modulus of the hip cartilage (3.8–16 MPa)<sup>37</sup> are rather large, and it is not possible to assess them in an intact hip. A more sophisticated model that included these variations would require additional experimental data. Second, the shapes of the articular surfaces and the necrotic region were described by simple geometric structures (spheres and a cone, respectively). The cartilage above the necrotic region was taken to attain the shape of the spherical shell. We studied only necrotic lesions located entirely in the load-bearing area. Our model could be upgraded to include larger regions. Third, the decrease of the load-bearing capacity was assumed to be uniform over the entire necrotic segment, whereas experiments on the hip with a necrotic segment showed that the mechanical properties of the necrotic part of the bone vary in the necrotic segment.<sup>15</sup> Necrotic lesions often have boundary irregularities.<sup>14</sup> Hips with advanced osteonecrosis may become incongruent.<sup>20,39</sup> Variations in the shape and material properties of the necrotic lesion, the cartilage, and shape of the femoral head could lead to local stress concentrations.

The theory that OA is induced by elevated contact stress is widely accepted for dysplastic hips that have poor lateral coverage of the femoral head.<sup>22,32,34,35</sup> It was determined by the same model as used in our work that the normalized peak stress in dysplastic hips is  $7100 \text{ m}^{-2} \pm 3700 \text{ m}^{-2}$  (compared with  $3500 \text{ m}^{-2} \pm 900 \text{ m}^{-2}$  found in normal hips).<sup>32</sup> Here we showed (Figs 3–5) that such values (which are known to be associated with OA) can be attained in hips with necrosis of the femoral head. The results support our hypothesis that OA in hips with necrosis of the femoral head is connected with elevated contact hip stress. Our results also are in agreement with clinical



**Fig 6A–H.** The diagrams show the contact stress distribution for the same position ( $\vartheta_N = 0^\circ$ ) and different size of the articular area corresponding to the necrotic segment: (A)  $\vartheta_0 = 0^\circ$ ; (B)  $\vartheta_0 = 10^\circ$ ; (C)  $\vartheta_0 = 20^\circ$ ; (D)  $\vartheta_0 = 30^\circ$ , and for the same size ( $\vartheta_0 = 30^\circ$ ) and different position of the articular area corresponding to necrotic segment: (E)  $\vartheta_N = 10^\circ$ ; (F)  $\vartheta_N = 25^\circ$ ; (G)  $\vartheta_N = 40^\circ$ ; (H)  $\vartheta_N = 54^\circ$ . The lateral coverage of the femoral head by the acetabulum is  $\vartheta_{CE} = 30^\circ$ . The examples shown in this figure correspond to points in Figures 4 and 5 that are marked by the same letters. The upper figures show the stress distribution in the frontal plane, the position of the peak contact stress  $p_{max}$ , the position of the stress pole  $\Theta$ , and the direction of the resultant hip force  $\vartheta_R$ . The lower figures show the corresponding projections of the stress distribution on the xy plane. The position of the stress pole is marked by the black dot.



observations that there was a high incidence of OA in hips with advanced necrosis of the femoral head.<sup>20,21</sup>

The presence of the necrotic lesion influences the value of the peak contact stress and it affects the stress distribution pattern on the articular surface. Compared with a normal hip (Fig 6A), a hip with necrosis is only moderately affected by a small necrotic segment (Fig 6B–C). However, if the necrotic segment becomes large, the contact stress becomes highly nonuniform (Fig 6D). Certain positions of the necrotic segment considerably increase the peak contact stress. If the necrotic segment is located laterally, then the value of the peak contact stress is high and stress distribution is highly nonuniform (Fig 6D). If the necrotic segment is moved from this region, the contact stress distribution is more favorable (Fig 6E–F). More-

over, if the necrotic segment lies in the region that in the normal hip does not bear much load (eg, close to the medial border of the load-bearing area), its effect on the value of the peak stress is negligible (Figs 5A, 6G–H).

To provide a complex biomechanical analysis of the individual patient, we must consider the shape of the osteonecrotic region and that of the hip and pelvis. We showed that two different normal shapes of the hip can have substantially different effects on contact stress in a hip with a substantially developed osteonecrosis. It is especially unfavorable if the effects that increase the peak contact stress in the hip (eg, unfavorable hip and pelvis shape) and a large necrotic segment act together to increase the peak contact stress (Fig 4A) because this can accelerate OA development.

An unfavorable biomechanical situation of the hip can be corrected by an operation that changes the shape of the hip, and therefore, the contact stress distribution. In hips with a necrotic segment, the operation should provide mechanical support for the subchondral bone (cortical bone graft) or shift the necrotic segment away from the area that would be subject to high load (osteotomy).<sup>18,25</sup> The change in the load-bearing capacity of the necrotic segment and the change of its position may modify the peak contact stress distribution (Figs 3, 5). As a result, contact stress of the hip would decrease, the cartilage would be relieved of stress, and the development of OA presumably would be retarded.<sup>1-4,18,24</sup> Understanding the complex interdependence of the size and position of the necrotic lesion and the shape of the hip and pelvis is important in making decisions regarding optimal treatment. In a patient, stress distribution can be estimated by using the above-described method. If stress is considerably greater than in the intact hip, surgical removal of the necrotic part from the region of greatest stress would be indicated. If, however, the values of stress in a necrotic hip remain in the normal range, the operation would not improve the loading condition in the hip.

Despite the limitations of our modeling procedure, we think taking into account the above irregularities would not change the general effects of the necrotic lesion on the stress distribution described in this work. Our model is especially suitable for retrospective studies as the geometric parameters needed to compute the stress distribution can be obtained from standard AP radiographs from the archives. The more realistic contact hip stress distribution can be incorporated into the finite element analysis as an improved boundary loading condition, which would improve the accuracy of the calculated stress patterns in the bony parts of the hip.<sup>9</sup>

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