

Theoretical relationship between maximum pore size and toughness in experimental inflammatory arthritis

M. W. PYSKLYWEC, E. R. BOGOCH¹

Orthopaedic Research Laboratory and ¹Department of Surgery, University of Toronto, St Michael's Hospital, 160 Wellesley St. E., Toronto, Ontario, Canada M4Y 1J3

Rheumatoid arthritis increases the risk of fracture. In an animal model of inflammatory arthritis, femoral diaphysis had a decreased toughness as well as increased cortical porosity, when compared to normal bone. Based on the hypothesis that stress concentration from the large porous defects reduces the ability of the cortical bone to resist failure, this work determined if the changes observed in porosity could explain the changes observed in toughness. Using theoretical relationships of the stress concentration and stress states, a model of the observed conditions was considered. A relationship was developed that indicated the relative difference in toughness between normal and arthritic specimens as a function of pore size. Results indicated that the increase in cortical pore size could theoretically reduce toughness by 55%. This decrease compares with the experimentally observed drop in toughness of 61%. Furthermore, the critical parameter for fracture in this situation is the ratio of pore diameter to cortical thickness. Efforts to reduce cortical porosity seen in inflammatory arthritis would be effective in enhancing the toughness of bone and may reduce morbidity in a human population.

© 2000 Kluwer Academic Publishers

Nomenclature

a = pore diameter
 β = geometry factor for stress intensity factor
 c = microcrack length emanating from pore
 G = shear modulus
 I = polar moment of inertia
 J = fracture energy per unit volume = toughness
 J_0 = fracture energy
 K = stress intensity factor
 K_{IC} = critical stress intensity factor
 L = length of specimen
 π = pi
 θ = angular deflection
 r = radial distance of pore from centroid
 σ = applied tensile stress
 τ = shear stress
 T = torque
 V = volume of specimen
 w = width of stressed body

1. Introduction

Rheumatoid arthritis is characterized by a rapid remodeling osteopenia [1–4] and by increased fracture risk in the femur as well as in cancellous bone [5–7].

In both bending and torsional testing, the elastic properties (shear modulus and elastic modulus) of experimental arthritic bone were not significantly different from that of normal bone [8]. However

toughness, the energy absorbed to fracture, is significantly reduced in arthritic bone. Studies in a rabbit model of experimental inflammatory arthritis have confirmed that the femoral shaft has a significantly decreased toughness compared to normal [9].

Histomorphometric analysis of diaphyseal sections of bone in the same model of experimental inflammatory arthritis demonstrated structural changes that may account for the decreased toughness [10]. In particular, the arthritis sections had a few large defects or pores that were approximately twice the size of those in the normal group. In addition, the analysis demonstrated that various geometrical features of diaphyseal bone, such as cross-sectional area, cortical thickness and moment of inertia, were altered in arthritis. The results of these studies are summarized in Table I.

Given that elastic properties of bone in arthritis were virtually unchanged, but toughness considerably reduced, failure was hypothesized to occur secondary to the stress concentrating effect of the large porous bodies. A material containing an imperfection such as a hole will generally have a reduced toughness [11, 12]. While external stresses may appear safe, stress concentration can enhance areas of internal forces above critical levels, causing fracture. A few large cortical defects could theoretically reduce toughness while material properties, such as modulus, remain unchanged. Using

Please address correspondence to: Dr E. R. Bogoch, Suite 434, E. K. Jones Building, Wellesley Central Site, St Michael's Hospital, 160 Wellesley St. Toronto, Ontario, Canada M4Y 1J3.

TABLE I Experimental results from [9 and 10]

Reference	Normal or arthritic	Toughness (J) [J/m^3]	Shear modulus (G) [GPa]	Cross-sectional area (A) [$10E-6m^2$]	Moment of inertia (I) [$10E-12m^4$]	Pore diameter (a) [mm]	Cortical thickness (w) [mm]	a/w ratio
[9]	Normal	614 ± 59	4.4 ± 0.5	29.2 ± 1.7	363 ± 53			
	Arthritic	376 ± 25	3.9 ± 0.8	24.9 ± 3.2	339 ± 77			
[10]	Normal			27.2 ± 2.3	350.6 ± 14.8	0.292 ± 0.024	1.10 ± 0.02	0.459
	Arthritic			20.9 ± 0.7	292.7 ± 18.1	0.494 ± 0.160	0.86 ± 0.04	0.266

principles of fracture mechanics, a model may be constructed to simulate the stress concentrating effects of such pores. One may then determine the diminution in toughness expected for various sizes of a pore.

This study used a theoretical consideration to predict the loss of toughness of the femoral diaphysis that would result from the altered porosity observed in previous studies in a model of experimental inflammatory arthritis. The relative significance of the large porous bodies in the bone of arthritis could be estimated.

2. Methods

In order to simulate the experimentally observed changes, two conditions had to be reproduced: (a) the bone and defect configuration and (b) the loading conditions.

Femoral cortical bone was treated as a cylindrical tube of perfect dimensions. The bone material was assumed to be homogeneous and isotropic. As the three dimensional configuration of porosity in bone is unknown, estima-

tions of its geometry were made. Only one pore (the largest) was considered in the model, as the largest defect initiates crack propagation and subsequent fracture [13, 14]. This defect was taken to be a cylinder within the cortex, assuming that the long axis of the pore extends down the long axis of the bone (Fig. 1).

Previous experimental work had involved mechanical testing of diaphyseal sections in torsion [9]. Thus, this analysis considered torsion of a cylindrical body. In an effort to simulate the stress state surrounding the pore in the wall of a cylindrical body, a small volume of interest was considered (Fig. 1). The stress applied to this unit volume was representative of the gross action on the structure. Torsional loading of the diaphysis translates to maximum tensile and compressive forces at 45° from the long axis [14]. As brittle fracture mechanisms usually occur due to maximum tensile forces, the important fracture forces occur at 45° from the long axis. The long axis of the pore was oriented at 45° , normal to the tensile force. The unit volume was also oriented obliquely, such that it became a cube of material under tension with a

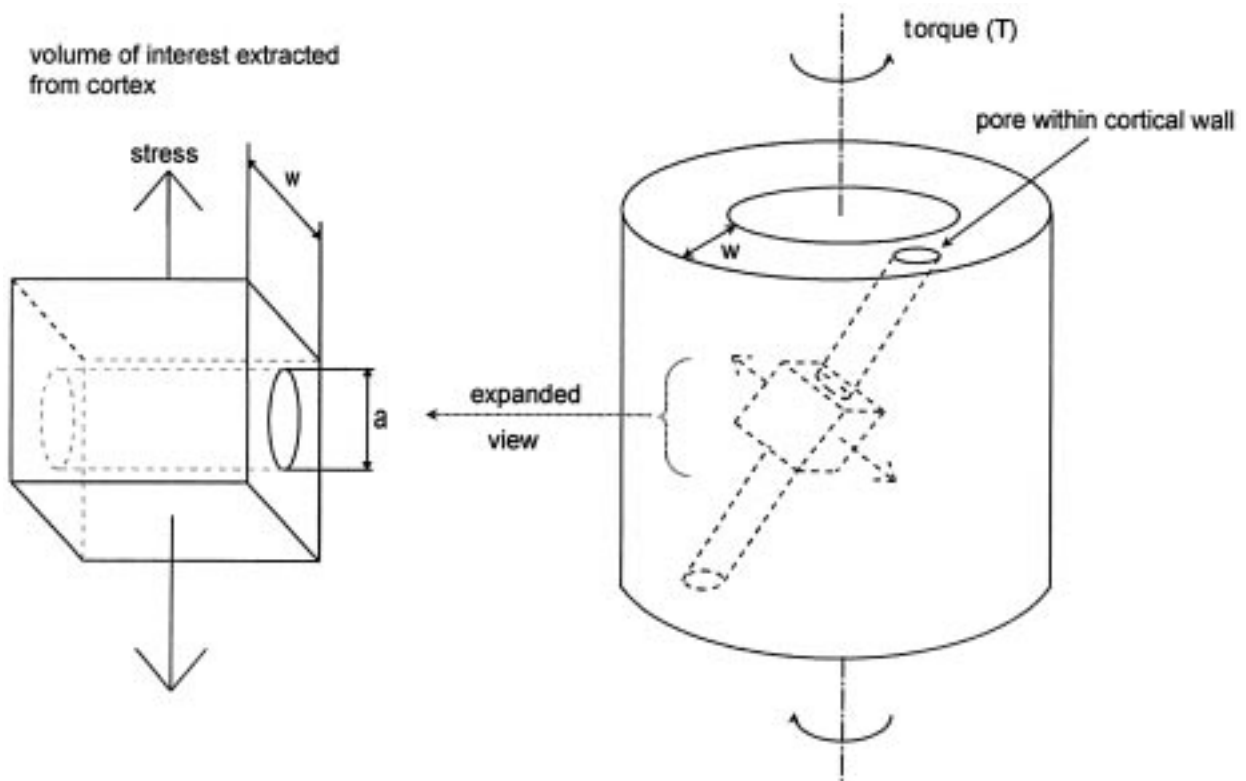


Figure 1 Cortical bone is modeled as a hollow cylinder. The porous defect is taken to be of cylindrical shape and contained within the cortical wall. A volume of interest was abstracted from around the defect. This area of interest was used to analyze the local stresses resulting from a gross torsion.

transverse cylindrical hole. The compressive stress that would act normal to the tensile force was ignored as it would have little effect on fracture mechanisms.

2.1. Calculations

In developing a relationship between porosity and toughness, two basic principles were used. One equality considered the relationship between applied torque and the energy absorbed to fracture (toughness). The other equality involved stress intensity factors which related torsion to pore size.

Assuming a linear elastic material, the energy absorbed by a body under torsion is the area under the torque-angular deformation curve [14].

$$J_o = \text{energy} = \text{area under } T - \theta \text{ curve}$$

$$J_o = \frac{1}{2} T\theta$$

$$\text{Knowing that } \theta = \frac{TL}{GI},$$

$$J_o = \frac{1}{2} \frac{T^2 L}{GI}$$

$$J = \frac{1}{2} \frac{T^2 L}{GI} \quad (1)$$

Stress concentration depends on the size and geometry of the imperfection [15–19]. For the particular stress-concentrating situation being analyzed, the stress becomes redistributed around a cylindrical hole. For a cylindrical hole in an *infinite* plate, the stress at the edges of the hole are three times greater than the stress being applied to the plate [13,18]. For a plate with *finite* dimensions (i.e. the hole becomes significant in size with respect to the width of the plate), the stress concentration around the hole becomes even greater [18], as demonstrated in Fig. 2.

The critical stress intensity factor (K_{IC}) is the value at which stress concentration causes failure [13]. The stress intensity factor depends on the applied stress, the geometry and location of the stress concentrator and the size of the microcrack emanating from the stress concentrator. The relevant stress concentration equality is [13, 18]:

$$K = \beta\sigma\sqrt{\pi c}.$$

The β value indicates the ratio of the maximum stress concentration to the general stress experienced by the body. For a cylindrical defect in a *finite* sheet:

$$\beta = \frac{\sigma_{\max}}{\sigma_{\text{gen}}}$$

$$\beta = \frac{2 + \left(1 - \frac{a}{w}\right)^3}{1 - \frac{a}{w}} \quad (2)$$

where σ_{\max} = maximum stress within the body due to stress concentration
 σ_{gen} = general stress computed from the external force

In torsion the maximum tension is oriented 45° to the horizontal [14]. By Mohr's circle analysis, this tensile

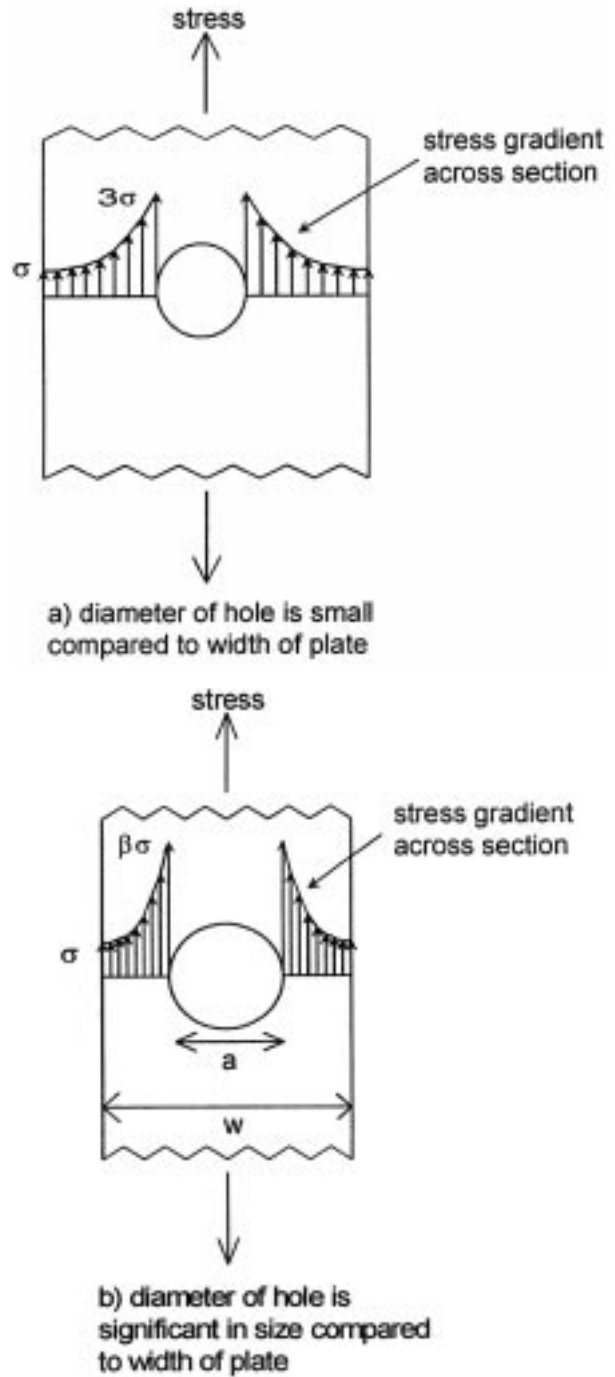


Figure 2 When stress σ is applied to an infinite (i.e. unlimited width) plate, the greatest stress concentration occurs adjacent to the defect, that being 3σ . However, when stress is applied to a finite plate, the stress concentration is greater than 3σ .

stress (σ) is equal to the overall shear stress (τ) caused by the torsion. Knowing that,

$$\tau = \frac{Tr}{I},$$

and that at failure, $K = K_{IC}$, then:

$$K_{IC} = \beta \frac{Tr}{I} \sqrt{\pi c}.$$

Rearranging and squaring:

$$T = \frac{K_{IC}I}{\beta r \sqrt{\pi c}},$$

$$T^2 = \frac{K_{IC}^2 I^2}{\beta^2 r^2 \pi c}.$$

Subbing into Equation 1 gives:

$$J = \frac{K_{IC}^2 I}{2GA\beta^2 r^2 \pi c}. \quad (3)$$

Thus, the amount of energy per unit volume absorbed to fracture depends on various structural properties of the bone, as well as material properties such as K_{IC} and G .

For a material under plane strain, K_{IC} is the same for all conditions, such as variable flaw [13, 15]. A normal bone and an arthritic bone stressed to failure will both achieve the same critical stress intensity factor assuming failure occurs due to crack propagation at the stress concentrator. $K_{IC(\text{arthritic})} = K_{IC(\text{normal})}$ at fracture.

A ratio for the toughness, may be derived.

$$\frac{2J_1 G_1 \beta_1^2 r_1^2 \pi c_1 A_1}{I_1} = \frac{2J_2 G_2 \beta_2^2 r_2^2 \pi c_2 A_2}{I_2}$$

3. Results

Considering basic mechanics of materials, theoretical relationships of fracture were used to derive an equation expressing the change in toughness due to a stress-concentrating flaw in a body under torsion. The relevant equation is:

$$\frac{J_1}{J_2} = \frac{G_2 A_2 I_1 r_2^2 \beta_2^2 c_2}{G_1 A_1 I_2 r_1^2 \beta_1^2 c_1} \quad (4)$$

$$\text{where: } \beta = \frac{2 + (1 - \frac{a}{w})^3}{1 - \frac{a}{w}}.$$

This equation indicates that a relative drop in toughness could occur due to various factors including: moment of inertia, shear modulus, cross-sectional area, distance of pore from the centroid, microcrack size and geometrical and size factor of stress concentrator.

3.1. Experimental correlates

The relative effect of porosity in affecting toughness may be established by considering experimental values in the theoretical context of the derived equation.

The porosity of normal and experimental arthritic bone was examined in a study [10] of rabbit cortical diaphysis. In a separate study, the toughness of arthritic and normal bone was examined [9]. The experimental results indicated changes in mechanical and structural properties in arthritis (Table I).

When the pore size values from the porosity study [10] were inserted into Equation 4, toughness of arthritic bone was predicted to be 55% that of normal. This substantially agrees with the experimental findings of 61% [9]. These results indicate that, in addition to the other geometric changes, experimentally observed changes in pore size could account for the observed reduction in toughness of the femoral diaphysis in this model.

4. Discussion

The toughness and pore size of diaphyseal bone have been linked by a theoretical mechanical relationship. When experimental values for observed porosity are considered in the theoretical equation, the predicted loss of toughness is similar in magnitude to that observed experimentally. This outcome suggests that the change in porosity, and in particular, the change in maximum pore size could be responsible for reducing toughness of bone in this model of arthritis.

4.1. Assumptions

Despite the close agreement between observed and predicted values, the results must be interpreted with caution because of numerous approximations and assumptions in the theoretical model. Bone was modeled as a homogeneous, isotropic material, which ignores microstructural features, some of which have significant mechanical effects. Due to the orthogonal nature of bone, stress is well tolerated along the long axis, but less so in the radial direction [20]. Cement lines surrounding osteons would probably act to arrest crack propagation, thereby increasing toughness [17]. In addition, the location and size of other flaws, such as osteons and other pores, will affect the fracture properties of the bone by further concentrating stresses and providing low-energy methods of crack propagation.

The choice of the stress intensity relationship is difficult. For a complex stress concentration such as that of porous bone under torsion, no published relationships were available. The stress intensity factor that was used in this work typically describes plates in pure tension. However, the torsional state causes a compressive stress normal to the tensile stress. This was ignored as this compressive stress was postulated to be much less significant than the maximum tensile stress, the critical factor in brittle fracture [18]. The dimensions of the involved body involved an infinite thickness, representing the length of the long bone. This assumption will introduce a situation of plane strain in the body. In plane strain critical stress intensity (K_{IC}) remains constant over a large range of thickness values [13] suggesting that the assumption of $K_{IC(\text{arthritic})} = K_{IC(\text{normal})}$ is reasonable in a theoretical context. Realistically, the K_{IC} likely does differ between the two groups as overall per cent porosity is increased throughout the cortex in arthritis. Not only does this introduce additional sites of stress concentration, it alters the material properties. However, the change in K_{IC} is speculated to be minimal at such low porosities, and large stress concentrators overwhelm the mechanism of weakening by per cent porosity.

The shear modulus of bone in arthritis was assumed to be insignificantly different from normal. The porosity difference between normal and arthritis is thought to be largely due to a few large pores in arthritis, with a relatively smaller contribution from overall generalized osteopenia. While this is a simplification, it is again likely that stress concentration from the large defects overwhelms any weakening by changes in per cent porosity.

In the context of this study, analysis assumed that the critical long axis dimension was greater than the

magnitudes of the pore diameter (on the x - y plane) and the cortical thickness. That is, for this assumption to be valid, the length of the pore should extend along the long axis to a greater extent than the cortex is wide. This dimension of the pore is unknown as analysis has only considered cross-sections of the cortex. Previous work [21] indicates that sectional area of such a flaw is critical, with less dependence on its length in the long axis of the bone.

By assuming that the pore is oriented at 45° , the dimensions of the pore are only slightly affected. Any change in dimension will be seen in the circumferential dimension, not the radial direction. The latter dimension is critical in this model. Had the pore been oriented at 0° , a 45° unit volume would have formed a porous body with an elliptical cross-section rather than a circular one, with maintenance of the radial dimension ("a" value) of the defect.

4.2. Relative contribution of factors

Examination of the final equation demonstrates the values that would be significant in affecting toughness. The relative contributions of each of these factors may be examined. The observed decrease in shear modulus would actually predict an increase in toughness for arthritis. Similarly, the decrease in cross-sectional area would predict increased toughness. Given that toughness decreased in arthritis, these variables are being overwhelmed by another factor.

Polar moment of inertia is minimally changed and one study [9] found no significant difference between the two groups. The radial distance of the pore from the centroid (r) would appear to be significant as it is a squared term. This parameter was not measured experimentally. However, the radial distance of the pore in arthritis must be greater than that for normal if it is to decrease toughness in arthritis. The internal radius of arthritic diaphyseal cross-sections (from centroid to endosteum) is slightly decreased making a significant increase in radial distance unlikely.

In his review on fracture mechanics, Bonfield [15] discussed the relative unimportance of radius of curvature compared to crack length. Similarly, when considering the stress concentrating complex, the large defect (i.e. pore) influences the fracture mechanics while the microcrack (c) emanating from the pore does minimally so. In disregarding the microcrack, it is unlikely that a major contributor to loss of toughness has been ignored.

4.3. Significance of pore size

The theoretical drop in toughness (55%) was substantially similar to the experimentally observed diminution (61%). As most of the other factors are relatively unimportant or demonstrate a negative influence, one may then infer the significance of the porous bodies in reducing toughness. This analysis considers the ability of these cortical defects to concentrate stress, leading to weakening in brittle fracture. The experimentally observed pores are large enough to produce a change in toughness similar to that observed. Methods to prevent

the occurrence of these defects could be effective in improving toughness in bone in arthritis.

In this model, the β ratio, or geometric term, appears to be critical in affecting toughness. As a squared term, it becomes numerically significant even at low β values. The β term becomes exquisitely sensitive to changes in a/w (pore size/cortical thickness) when $a/w > 0.5$. That is, once pore size increases above half of the cortical thickness, the toughness decreases significantly. Above $a/w = 0.6$, the accuracy for the β relationship becomes questionable as β increases exponentially. The critical parameter for fracture of such a material is the ratio of pore diameter to cortical thickness. Thus pore size is to be most significant dimension in reducing toughness.

4.4. Future work

Numerous improvements to the model could be made. A more complex, heterogeneous model of bone structure might be considered to further refine calculations. The interaction of stress intensity from numerous pores would have additional effects in concentrating forces. The compressive stress component of the torsional stress state could be included in the analysis. Overall porosity may reach a level that affects the material properties of the bone, making some of the assumptions somewhat liberal.

However, in pursuing the model further, the desired objectives of this work and the subsequent results must be considered. It was shown that porosity may affect the toughness, and this could occur to a degree seen experimentally, suggesting the important role of porosity. It is unlikely that further refinements to the model will disprove the results. Similarly, it is doubtful that refinements will further improve accuracy, as too many parameters are unknown or unmeasurable. The complexity of bone structure is difficult to model. Finite element analysis, although worthy of consideration, might not improve the results obtained.

Acknowledgments

This work was supported by the Arthritis Society of Canada. The authors thank Erica Moran of the Wellesley Orthopaedic Research Laboratory.

References

1. A. K. S. GOUGH, J. LILLEY, S. EYRE, R. L. HOLDER and P. EMERY, *Lancet* **344** (1994) 23.
2. M. MAGARO, A. TRICERRI, D. PIANE, A. ZOLI, F. SERRA, L. ALTOMONTE and L. MIRONE, *Rheumatol. Int.* **11** (1991) 73.
3. S. SHIMUZU, S. SHIOZAWA, K. SHIOZAWA, S. IMURA and T. FUJITA, *Arthritis Rheum.* **28** (1985) 25.
4. R. M. VAN SOESBERGEN, P. LIPS, A. VAN DAN ENDE and J. K. VAN DER KORST, *Ann. Rheum. Dis.* **45** (1986) 149.
5. C. COOPER and C. WICKHAM, in "Osteoporosis 1990", edited by C. Christiansen and K. Overgaard (Osteopress, Copenhagen, 1990) p. 1578.
6. J. R. HOOYMAN, I. MELTON, L. I. A. M. NELSON, W. M. O'FALLON and B. L. RIGGS, *Arthritis Rheum* **27** (1984) 1353.
7. T. K. SPECTOR, G. M. HALL, E. V. MCCLOSKEY and J. A. KANIS, *Br. Med. J.* **306** (1993) 558.
8. E. L. MORAN, J. M. LEE, S. REICHELDT and E. R. BOGOCH, *Orthopaedic Transactions* **17** (1992) 364.
9. C. M. BELLINGHAM, J. M. LEE, E. L. MORAN and E. R. BOGOCH, *J. Orthop. Res.* **13** (1996) 876.

10. M. W. PYSKLYWEC, E. L. MORAN, V. L. FORNASIER and E. R. BOGOCH, *J. Orthop. Rheum.* **9** (1996) 150.
11. D. B. BROOKS, A. H. BURSTEIN and W. H. FRANKELL, *J. Bone Joint Surg. Am.* **52A** (1970) 507.
12. R. B. MARTIN, *Crit. Rev. Biomed. Eng.* **10** (1984) 179.
13. D. BROEK, "The Practical Use of Fracture Mechanics" (Kluwer Academic Publishers, Norwell, MA, 1989) p. 56.
14. B. B. MUVDI and J. W. MCNABB, in "Engineering Mechanics of Materials 3" (Springer-Verlag, New York, 1991) p. 162.
15. W. BONFIELD, *J. Biomech.* **20** (1987) 1071.
16. S. D. BROWN, R. B. BIDDULPH and P. D. WILCOX, *J. Am. Ceram. Soc.* **47** (1964) 320.
17. J. D. CURREY, *Q. Jl. Microsc. Sc.* **103** (1962) 111.
18. R. E. PETERSON, "Stress Concentration Factors", (John Wiley and Sons, Toronto, 1974) p. 108.
19. R. W. RICE, *Mater. Sci. Eng* **A112** (1989) 215.
20. Y. C. FUNG, "Biomechanics: mechanical properties of living tissues" (Springer-Verlag, New York, 1981) p. 383.
21. J. A. HIPPEL, R. J. MCBROOM, E. J. CHEAL and W. C. HAYES, *J. Orthop. Res.* **7** (1989) 828.

*Received 18 June 1998
and accepted 29 April 1999*