A stochastic structural reliability model explains rotator cuff repair retears

Drew Michael S. Donnell, Jessica L. Seidelman, Christopher L. Mendias, Bruce S. Miller, James E. Carpenter and Richard E. Hughes

"Department of Orthopaedic Surgery, University of Michigan, 2017 Biomedical Research Building, 109 Zina Pitcher Place, Ann Arbor, MI, USA; bDepartment of Biomedical Engineering, University of Michigan, Ann Arbor, MI, USA; cDepartment of Industrial & Operations Engineering, University of Michigan, Ann Arbor, MI, USA

(Received 14 August 2014; accepted 20 October 2014)

High rates of tendon retear following surgical repair of large rotator cuff tears have been reported. This study developed a probabilistic structural reliability model of retear with potential to guide quality improvement interventions. A probabilistic biomechanical model of survivorship from a recurrent tear, based on structural reliability and Markov processes, treated the capacity of the surgical repair to withstand tensile loading and the load applied by the supraspinatus muscle as independent lognormally distributed random variables. For a repair to remain intact at the end of the $t$th day, it had to remain intact on days 0, …, $t$–1. After retear was predicted to occur, that repair remained torn in the model. The model predicted two-year survival of 75.7%, which is within the 95% confidence interval of the Kaplan–Meier for data reported by others. The model’s demonstrated prediction of retear can be used for improving repair survival: lowering the variance in both repair strength and in post-operative supraspinatus muscle loading is an effective method for lowering the retear rate. Variance reduction alone may be an effective way to improve surgical treatment of this disorder.

Keywords: failure analysis; probabilistic biomechanics; quality improvement; stochastic modeling; tendon

Introduction

Despite recent advances in the design of surgical components and innovation in techniques, the high rate of recurrent rotator cuff tear failures remains a significant clinical dilemma. Retear rates as high as 94% have been reported in large to massive rotator cuff tear arthroscopic repairs (Galatz et al. 2004), and studies of small- to medium-sized repairs have reported retear rates from 25 to 35% (Cummins & Murrell 2003). Understanding the process of surgical construct failure and reducing the rate of retears is an important part of improving the quality of care for future rotator cuff tear patients.

The surgically repaired tendon has mechanical properties that change over time as the tissues heal. Consequently, one approach to improving repair survival is to apply principles of structural reliability. Probabilistic engineering analysis methods have been used for the design and analysis of orthopedic devices (Browne et al. 1999; Laz et al. 2006; Nicollella et al. 2006; Pérez et al. 2006; Easley et al. 2007; Bah & Browne 2009; Dopico-González et al. 2009, 2010a, 2010b; Galbusera et al. 2010). However, such methods have not yet been applied to the analysis and design of soft-tissue reconstructive techniques and tissue healing.

The purpose of this study was to develop a probabilistic structural reliability model to explain the retear rate found in rotator cuff repair patients.

Methods

Structural reliability modeling

The analysis of retear was based on the application of structural reliability modeling (Thoft-Christensen & Baker 1982) to rotator cuff repair. For man-made structures, failure occurs when an incurred load ($L$) exceeds structural capacity ($C$). In the case of a steel bridge, for example, collapse occurs when an applied load exceeds the structural strength of the bridge. Analogously, the rotator cuff repair fails when the tensile load on the repair exceeds the load-to-failure of the surgical construct. We assumed failure occurs at the suture–tendon interface. Probabilistic structural reliability modeling extends this analysis by modeling both the load and capacity as random variables. We assumed that load and capacity could be reasonably modeled as lognormally distributed independent random variables. Suppose $\mu_{L,t}$ and $\mu_{C,t}$ are the mean values of $L$ and $C$, respectively, at time $t$, and $\sigma_{L,t}$ and $\sigma_{C,t}$ are the corresponding standard deviations. Then the probability of failure for a single set of realizations of load and capacity values is

$$P_t = \Phi \left( \frac{\ln \left( \frac{\mu_{L,t}}{\mu_{C,t}} \sqrt{\frac{\sigma_{L,t}^2 + 1}{\sigma_{C,t}^2 + 1}} \right)}{\sqrt{\ln((\gamma_C)^2 + 1)/(\ln((\gamma_L)^2 + 1))}} \right)$$

(1)
where $\Phi(z)$ is the cumulative distribution function of the standard normal distribution, $\gamma_{L,t}$ is the coefficient of variation of the load ($\sigma_{L,t}/\mu_{L,t}$), and $\gamma_{C,t}$ is the coefficient of variation of the capacity ($\sigma_{C,t}/\mu_{C,t}$) (Sundararajan & Witt 1995).

The parameters defining the lognormal load distribution were derived from published electromyographic data of the supraspinatus recorded during passive external rotation (McCann et al. 1993) which is a common exercise performed in the early post-operative period. These data were combined with a simple model of muscle force production to estimate the mean and standard deviation of the load applied to the surgical repair. The muscle model was the product of the fraction of maximum activation (McCann et al. 1993), physiological cross-sectional area (6.65 cm$^2$) (Ward et al. 2006), specific tension (22.5 N/cm$^2$) (Valero-Cuevas et al. 2003), and the cosine of the pennation angle (5.1°) (Ward et al. 2006). The resulting mean ($\mu_{L,0}$) and standard deviation ($\sigma_{L,0}$) of load at $t = 0$ were 40.1 and 78.7 N, respectively. We assumed the mean load increased linearly for six months post-op in a study conducted by Smith et al. (2003).

This re-

营业额 was assumed to match the values reported in the obtained from published studies. The initial repair capac-

ity both increase linearly up to six months post-operatively. This reflects the gradual increase in activity and healing that occur over time.

Figure 1. Mean load and capacity over time. Load and capacity both increase linearly up to six months post-operatively. This reflects the gradual increase in activity and healing that occur over time.

Survival function modeling
Conceptually, this model requires that the surgical repair must survive intact through days 0, … , $t - 1$ to be intact on the beginning of the $t$th day post-op. Once the surgical repair retears, it remains torn. A time-inhomogeneous two-state Markov chain model (Bhat 1972) was used to represent the survival of the surgical repair (Figure 2). A stochastic process was defined as $\{X_t | t = 0, 1, 2, 3, \ldots\}$, where $t$ indexed the beginning of the day following surgery ($t = 0$ represents the time of surgery). The two states in the model consist of (1) intact repair ($X_t = 1$) and (2) retear of repair ($X_t = 2$) at the beginning of post-op day $t$. The transition probability matrix for this stochastic process was

$$p(t+1) = \begin{pmatrix} (1 - p_1) & p_1 \\ 0 & 1 \end{pmatrix}. \quad (2)$$

In Markov process terminology, state 1 is transient and state 2 is absorbing. Over time, the probability of being in state 2 increases monotonically. The $t$-step transition probability matrix was

$$p(0:t) = \prod_{i=0}^{t-1} \begin{pmatrix} (1 - p_1) & p_1 \\ 0 & 1 \end{pmatrix}. \quad (3)$$

We modeled the day of surgery as having a success-

fully repaired rotator cuff at the beginning of the day, i.e. the row vector of state probabilities at time $t = 0$ was $\pi = [1 \ 0]$. The probability of the stochastic process being in state 2 at time $t + 1$ was

$$\text{Prob}[X_{t+1} = 2] = \pi P(0:t+1) = p_t \prod_{i=0}^{t-1} (1 - p_i). \quad (4)$$

Figure 2. Time-inhomogeneous Markov model. Circles represent states. State 1 at time $t$, i.e. $X_t = 1$, represents surgical repair being intact at the start of day $t$. Similarly, state 2 at time $t$ represents the repair being retorn at the beginning of day $t$. Intact states are in the top row of states; retorn states are below. A repair can remain intact from one day to the next (represented by horizontal arrows on top row of states). A repair can fail (diagonal arrows from top to bottom row of states). Once torn, a repair remains torn (represented by horizontal arrows on bottom row and the absence of arrows going from bottom to top row of states). Time moves from left to right. Transition probabilities are listed above the arrows.
The probability of being in state 1 at time \( t \), which corresponds to having an intact cuff at post-op day \( t \), was

\[
\text{Prob}[X_t = 1] = \pi P^{(0,t)} = \prod_{i=0}^{t-1} (1 - p_i)
\]

(5)

We sought to model the survivor function, \( S(t) = P[T \geq t] \) (Collett 1994). Having an intact cuff repair at the beginning of day \( t \) means the failure must occur on day \( t \) or later. Therefore,

\[
S(t) = \prod_{i=0}^{t-1} (1 - p_i).
\]

(6)

This is our model prediction of the survival function for rotator cuff retear.

**Clinical data**

Two sources of clinical data were used to evaluate the model. They were selected because they provide estimates of survival at multiple points in time. Primary data from Miller et al. (2011) were available for analysis. Retear data were prospectively collected on 22 consecutive patients who presented for arthroscopic repair of a large (>3 cm) or massive rotator cuff tear. Among the study population the mean age was 63.7 years, the mean duration of symptoms was 27 months, 50.0% were identified as female, and 4.5% were regular tobacco users. Tear shape varied among the patients: 63.6% were L-shaped, 27.3% were crescent-shaped (5.4 cm², mean), and 9.1% were U-shaped (6.0 cm², mean). All subjects had magnetic resonance imaging or ultrasound-confirmed full-thickness tears of the rotator cuff prior to surgery. Surgery was indicated if either 3 months of non-operative management proved unsuccessful or traumatic rupture of the rotator cuff was demonstrated in an otherwise asymptomatic shoulder (Miller et al. 2011).

Rotator cuff repairs were performed by one of two experienced shoulder surgeons (BSM or JEC) using a standardized arthroscopic technique. Ultrasound evaluations were performed at seven time points post-operatively: 2 days, 2 weeks, 6 weeks, 3 months, 6 months, 12 months, and 24 months. If a definite, full-thickness defect was visible, the tendon was considered torn; if continuous tendon fibers were visible over the humeral head attaching onto the greater tuberosity, the tendon was considered intact (Miller et al. 2011).

The second source of data was a published report of prospective study of 107 consecutive surgically treated rotator cuff tear patients having a median follow-up of eight years, which was used to validate our model’s survival prediction (Kluger et al. 2011). Primary data from this study were not available. The mean age was 59.5 years, the mean duration of symptoms was 7 months, 39.3% were identified as female, and 42.1% were regular tobacco users. Tear shape was classified as crescent-shaped, L-shaped, inverse L-shaped, V-shaped, or U-shaped, but not presented. Tear size was reported dichotomously: 49.5% were less than 5.0 cm² and 50.5% were greater than 5.0 cm².

The survival function for clinical data on rotator cuff retears can easily be estimated for these data using the Kaplan–Meier estimator, which uses the retear data to construct the fraction of patients having intact repairs up to the time immediately before the time of the next retear (Collett 1994). That fraction is recomputed when the next retear occurs. The fraction remains constant until the next retear is identified. As such, the Kaplan–Meier estimator provides a graphic description of the retear times in our data-set. The Kaplan–Meier estimate was constructed from the data using SAS 9.1.3 Service Pack 4 (SAS Institute, Cary, NC).

**Quality improvement simulation**

Our model was used to analyze the effect of changing the means and standard deviations of the load and capacity distributions on cuff repair survival at two years. The purpose of the simulation was to guide quality improvement. In the first simulation, the mean load and capacity were varied while maintaining constant variances. Mean load was reduced up to 50% and mean capacity was increased up to 50%.

In the second simulation, the variances of load and capacity were varied while maintaining constant means. The standard deviation of load and capacity were reduced by up to 50% each. Fifty percent changes in variance in one variable were also combined with 50% changes in the other variable’s mean.

**Results**

The structural reliability model predicted a survival function lying almost entirely within the 95% confidence interval of the Kaplan–Meier estimate derived from our clinical data. The model prediction fell slightly outside the 95% confidence interval only during the very early post-operative period, specifically at day 38. The predicted survival curve declines rapidly before stabilizing between 2 and 6 months post-op (Figure 3). At two years, the survival estimate was 75.7%. For comparison, the Kaplan–Meier estimate of the survivor function from our clinical data at 2 years was 59.1% ([38.6, 79.6], 95% CI) (Miller et al. 2011).

Model predictions also agree well with the results published (Kluger et al. 2011). In the similar study performed outside of our group, the reported percentage of
repairs surviving intact at 3 and 6 months were 75.7% ([67.6, 83.8], 95% CI) and 72.0% ([63.5, 80.5], 95% CI), respectively. At the end of the study, 67.3% ([58.4, 76.2], 95% CI) were intact. For comparison, the model predicted survival of 75.8, 75.7, and 75.6% at 3, 6, and 60 months, respectively, well within the 95% confidence intervals previously reported.

According to our model predictions, if only one parameter is changed, reducing the variance of the load on the repair site is the most effective way to increase rotator cuff repair survival. Decreasing the standard deviation of the load increases the two-year probability of survival to 97.7% (Table 1). Decreasing the standard deviation of the capacity by 50% each gives a survival probability of 98.6%. Changing mean load and capacity is not nearly as effective as reducing their variability. Increasing capacity by 50% with every other factor remaining constant gives a probability of 88.0%; decreasing mean load 50% increases the probability to 79.6%. Changing both mean load and mean capacity gives a survival probability of 88.3%. Thus, the single most important parameter for increasing two-year survival is the standard deviation of load. If one mean and one standard deviation are allowed to be changed simultaneously, then a survival probability of 99.6% can be reached by reducing the standard deviation of load by 50% and increasing the mean capacity by 50%.

**Discussion**

The primary goal of our investigation was to develop a probabilistic model capable of explaining the high rate of rotator cuff repair failures. This study successfully demonstrates that structural and engineering reliability modeling effectively predicts the probability of rotator cuff repair retears. The model predicted that variance reduction could be an effective way to reduce retear rates. While no model is perfect and must rely on a number of assumptions, our model contributes to the understanding of retear rates by introducing a new modeling paradigm for explaining clinical retear data.

Probabilistic mechanical modeling of structural failure is more suitable for analyzing clinical survival data than deterministic modeling paradigms. Deterministic failure analysis makes dichotomous statements (i.e. the repair does or does not fail); probabilistic models are well suited for comparison with the statistical field of survival analysis.

| Capacity | Unchanged | Mean increased 50% | SD reduced 50%* |
|----------|-----------|--------------------|-----------------|
| Load     |           |                    |                 |
|          | Unchanged | 75.7               | 88.0            |
| Mean     |           | 79.6               | 88.3            |
| Capacity |           | 97.7               | 99.6            |

*Variance reduction in computer simulations was represented as a 50% reduction in the standard deviation (SD).
Our work adds to the literature on probabilistic modeling in biomechanics. Stochastic models have been developed for analyzing fatigue damage in bone (Pidaparti et al. 2001; Taddei et al. 2006; Laz et al. 2007), injury risk (Miller & Freivalds 1995; Davidson et al. 2006), muscle force estimating (Mirka & Marras 1993; Hughes & An 1997; Chang et al. 2000; Valero-Cuevas et al. 2003), kinematics (Santos & Valero-Cuevas 2006), and joint strength prediction (Langenderfer et al. 2005, 2006). In orthopedics, probabilistic engineering analysis methods have been used for the design and analysis of orthopedic devices (Browne et al. 1999; Laz et al. 2006; Nicolella et al. 2006; Pérez et al. 2006; Easley et al. 2007; Bah & Browne 2009; Dopico-González et al. 2009, 2010a, 2010b; Galbusera et al. 2010). Our study extends the stochastic modeling methods to the analysis of soft-tissue surgical repairs.

This study has several limitations. First, the model is rather general in nature and does not explicitly account for specific factors associated with retear rates, such as age, atrophy, fatty degeneration, tear shape, and size beyond the inclusion criteria, tear thickness, and operative time (Le et al. 2014). Although our model predictions coincide well with both sources of data, the observed differences in the survival estimates may be explained by heterogeneity among the cohorts. Compared to the secondary data-set, the patients in the study at our institution were marginally older (63.7 years vs. 59.5 years), symptomatic for a longer period of time (27 months vs. 7 months), and diagnosed with larger rotator cuff tears.

From a mathematical perspective, it has all the inherent limitations of time-inhomogeneous Markov models. Specifically, Markov models are ‘memoryless’, i.e. the probability of making a transition between times t and t + 1 depends only on the state of the stochastic process at time t. Information before t is ignored. In our model, knowledge of the history of tissue healing and functional recovery from time 0 to t – 1 is incorporated into the capacity (C) and load (L) distributions. Analytically, the model required simplifying assumptions. The capacity–load interference model assumes the input variables are lognormally distributed and statistically independent. The lognormal distribution is characterized by a single peak of probability density, non-negative values, and a long tail on the positive side. The most popular distribution for stochastic modeling is the normal distribution. However, normal distributions were not used because they allow for negative load and capacity values, which are mechanically nonsensical.

While independent lognormal distributions for load and capacity were assumed, which implies that the two quantities are uncorrelated for each pair (L, C) of random variable realizations, the model did assume that both the mean load and mean capacity increase over time. Thus, at the time scale of months there is a positive, deterministic relationship between the means of load and capacity. This matches the intuition that as the shoulder heals the patient can engage in more frequent and, ultimately, more demanding activities. However, on the day-to-day time scale there is no correlation between load and capacity. We assessed the effect of the independence assumption by performing numerical copula simulations in which a multivariate lognormal distribution for load and capacity was used, thus correlation was introduced (Nelsen 2006). Generation of random variables from multivariate distributions with weak correlation coefficients of 0.1 and 0.2 yielded two-year survival estimates of 77.5 and 80.2%, respectively, the former of which fell within the empirical 95% confidence interval. The implications are not emphasized, because the correlation between in vivo repair strength and muscle loading is unknown. Any assumed correlation coefficient would be purely arbitrary.

The model is also limited by the input data, which were obtained from published studies of cadaver and animal tests. No human in vivo capacity values were available. Biomechanical testing was performed on cadaver shoulders that may not be representative of patient tissue. We assessed the effect of the maximum mean capacity following healing by varying the parameter estimate. The increase of this biomechanical parameter by 10 and 20% yielded two-year survival estimates of 79.1 and 81.9%, respectively, the former of which fell within the empirical 95% confidence interval. The decrease of this biomechanical parameter by 10 and 20% yielded two-year survival estimates of 71.5 and 66.3%, respectively, both of which fell within the empirical 95% confidence interval. A decrease in the maximum mean capacity, contrasted to an increase, may be of greater clinical relevance due to compromised tissue quality and biological healing processes within an aging population of patients with rotator cuff tears. Similarly, we selected one rehabilitation activity that is performed in the early preoperative period to estimate the mean and variance of the supraspinatus load. In reality a wider range of exercises are performed, but it was not possible to pool estimates of variances of different activities without some knowledge of their relative frequency.

The simplifying assumptions of linear increases in the mean load and mean capacity for the first six months following surgery are another limitation. Variation among biological healing trajectories and rehabilitation protocols convolutes the estimation of these time-dependent profiles. We simultaneously assessed the effect of these assumptions by varying the time period of the linear increase. The reduction of this period of interest between four and five months (from the original six months) yielded two-year survival estimates of 83.1 and 79.4%, respectively, the latter of which fell within the empirical 95% confidence interval.
In summary, the most significant result of our work is the implication for future quality improvement efforts in rotator cuff tear repair and possibly other soft-tissue repairs. Clearly, the current rate of retears is higher than desired, and the quality of care can be enhanced by reducing the retear rate. Our model points to opportunity for significant quality improvement by reducing the variance of load and capacity. Variance in load may be reduced by standardizing rehabilitation protocols and improving patient compliance. Variance in capacity may be reduced in teaching hospitals such as ours by, for example, using surgical simulators to improve the proficiency of residents in arthroscopy techniques. Our results are consistent with manufacturing quality improvement theory, which holds that variance reduction and control are the keystones of quality improvement.

Conflict of interest
None.

Acknowledgements
The authors thank Brian Downie, PA-C, MS, and Tom Cichonski, BS, for their assistance.

Funding
University of Michigan’s Department of Orthopaedic Surgery and the Student Biomedical Research Program.

References
Bah MT, Browne M. 2009. Effect of geometrical uncertainty on cemented hip implant structural integrity. J Biomech Eng. 131:054501-1–054501-5.
Bhat UN. 1972. Elements of applied stochastic processes. New York (NY): Wiley.
Browne M, Langlely RS, Gregson PJ. 1999. Reliability theory for load bearing biomedical implants. Biomaterials. 20:1285–1292.
Chang Y-W, Hughes RE, Su F-C, Itoi E, An K-N. 2000. Prediction of muscle force involved in shoulder internal rotation. J Shoulder Elbow Surg. 9:188–195.
Collett D. 1994. Modelling survival data in medical research. New York (NY): Chapman and Hall.
Cummins CA, Murrell GA. 2003. Mode of failure for rotator cuff repair with suture anchors identifying factors. J Shoulder Elbow Surg. 12:128–133.
Davidson PL, Chalmers DJ, Stephenson SC. 2006. Prediction of distal radius fracture in children, using a biomechanical impact model and case-control data on playground free falls. J Biomech. 39:503–509.
Dopico-González C, New AM, Browne M. 2009. Probabilistic analysis of an uncremented total hip replacement. Med Eng Phys. 31:470–476.
Dopico-González C, New AM, Browne M. 2010a. A computational tool for the probabilistic finite element analysis of an uncremented total hip replacement considering variability in bone-implant version angle. Comput Methods Biomech Biomed Eng. 13:1–9.
Dopico-González C, New AM, Browne M. 2010b. Probabilistic finite element analysis of the uncremented hip replacement – effect of femur characteristics and implant design geometry. J Biomech. 43:512–520.
Easley SK, Pal S, Tomaszewski PR, Petrella AJ, Rullkoetter PJ, Laz PJ. 2007. Finite element-based probabilistic analysis tool for orthopaedic applications. Comput Methods Programs Biomed. 85:32–40.
Galatz LM, Ball CM, Tefsey SA Middleton WD, Yamaguchi K. 2004. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 86-A:219–224.
Galibusa F, Anasetti F, Bellini CM, Costa F, Fornari M. 2010. The influence of the axial, antero-posterior and lateral positions of the center of rotation of a ball-and-socket disc prosthesis on the cervical spine biomechanics. Clin Biomech (Bristol, Avon). 25:397–401.
Hughes RE, An KN. 1997. Monte Carlo simulation of a planar shoulder model. Med Biol Eng Comput. 35:544–548.
Klinger HM, Koelling S, Baums MH, Kahl E, Steckel H, Smith MM, Schultz W, Miosge N. 2009. Cell biological and biomechanical evaluation of two different fixation techniques for rotator cuff repair. Scand J Med Sci Sports. 19:329–337.
Langenderfer JE, Carpenter JE, Johnson ME, An KN, Hughes RE. 2006. A probabilistic model of glenohumeral external rotation strength for healthy normals and rotator cuff tear cases. Ann Biomed Eng. 34:465–476.
Langenderfer JE, Hughes RE, Carpenter JE. 2005. A stochastic model of elbow flexion strength for subjects with and without long head biceps tear. Comput Methods Biomech Biomed Eng. 8:315–322.
Laz PJ, Pal S, Halloran JP, Petrella AJ, Rullkoetter PJ. 2006. Probabilistic finite element prediction of knee wear simulator mechanics. J Biomech. 39:2303–2310.
Laz PJ, Stowe QJ, Baldwin MA, Petrella AJ, Rullkoetter PJ. 2007. Incorporating uncertainty in mechanical properties for finite element-based evaluation of bone mechanics. J Biomech. 40:2831–2836.
Le BT, Wu XL, Lam PH, Murrell GA. 2014. Factors predicting rotator cuff retears: an analysis of 1000 consecutive rotator cuff repairs. Am J Sports Med. 42:1134–1142.
McCann PD, Wootten ME, Kadaba MP, Bigliani LU. 1993. A kinematic and electromyographic study of shoulder rehabilitation exercises. Clin Orthop Relat Res. 288:179–188.
Miller BS, Downie BK, Kohan RB, Kijek T, Lesniak B, Jacobson JA, Hughes RE, Carpenter JE. 2011. When do rotator cuff repairs fail? Serial ultrasound examination after arthroscopic repair of large and massive rotator cuff tears. Am J Sports Med. 39:2064–2070.
Miller SA, Freivalds A. 1995. A stress–strength interference model for predicting CTD probabilities. Int J Ind Ergon. 15:447–457.
Mirka GA, Marras WS. 1993. A stochastic model of trunk muscle coactivation during trunk bending. Spine. 18:1396–1409.
Nelsen RB. 2006. An introduction to copulas. New York (NY): Springer Science+Business Media.
Nicolella DP, Thacker BH, Katoozian H, Davy DT. 2006. The effect of three-dimensional shape optimization on the probabilistic response of a cemented femoral hip prosthesis. J Biomech. 39:1265–1278.
Pérez MA, Grasa J, García-Aznar JM, Bea JA, Doblaré M. 2006. Probabilistic analysis of the influence of the bonding degree of the stem-cement interface in the performance of cemented hip prostheses. J Biomech. 39:1859–1872.
Pidaparti RM, Wang QY, Burr DB. 2001. Modeling fatigue damage evolution in bone. Biomed Mater Eng. 11:69–78.
Santos VJ, Valero-Cuevas FJ. 2006. Reported anatomical variability naturally leads to multimodal distributions of Denavit-Hartenberg parameters for the human thumb. IEEE Trans Biomed Eng. 53:155–163.
Smith CD, Alexander S, Hill AM, Huijsmans PE, Bull AM, Amis AA, De Beer JF, Wallace AL. 2006. A biomechanical comparison of single and double-row fixation in arthroscopic rotator cuff repair. J Bone Joint Surg Am. 88:2425–2431.
Sundararajan C, Witt FJ. 1995. Stress–strength interference method. In: Sundararajan C, editor. Probabilistic structural mechanics handbook. New York (NY): Chapman and Hall; p. 8–26.
Taddei F, Martelli S, Reggiani B, Cristofolini L, Viceconti M. 2006. Finite-element modeling of bones from CT data: sensitivity to geometry and material uncertainties. IEEE Trans Biomed Eng. 53:2194–2200.
Thoft-Christensen P, Baker MJ. 1982. Structural reliability theory and its applications. New York (NY): Springer-Verlag.
Valero-Cuevas FJ, Johanson ME, Towles JD. 2003. Towards a realistic biomechanical model of the thumb: the choice of kinematic description may be more critical than the solution method or the variability/uncertainty of musculoskeletal parameters. J Biomech. 36:1019–1030.
Ward SR, Hentzen ER, Smallwood LH, Eastlack RK, Burns KA, Fithian DC, Friden J, Lieber RL. 2006. Rotator cuff muscle architecture: implications for glenohumeral stability. Clin Orthop Relat Res. 448:157–163.