

Analysis of Intervertebral Disc Degeneration with Nondestructive Indentation Test: Analyses in Time and Frequency Domains

Kun-Yi Lin, Shih-Youeng Chuang, Shyu-Jye Wang, Shing-Sheng Wu, and Leou-Chyr Lin*

*Department of Orthopaedic Surgery,
Tri-Service General Hospital, National Defense Medical Center,
Taipei, Taiwan, Republic of China*

Background: The objective of this study was to develop a nondestructive indentation technique to quantify the degradation of the material properties of the annulus fibrosus (AF) resulting from nontraumatic cyclic fatigue and to analyze the data in both the time domain, with the relaxation ratio, and the frequency domain, with a parametric autoregressive model. **Methods:** In this study, 7 human cadaver intervertebral discs were included (4 from L3/4, 3 from L2/3) with a mean age of 52 years (range, 35-61). The relaxation indentation test was applied with an MTS 858 materials testing system using 2.5 mm hemispherical indenters on the posterior disc. Computational analyses, separated into time and frequency domains, were undertaken with an IBM-compatible computer using custom-designed software based on the Matlab (Mathworks, Natick, MA) programming environment. **Results:** In the time domain, the relaxation ratio averaged 60.7% in the control and 74.4% after cycling. There was a 13.7% increase after cycling. In the frequency domain, the best order of the relaxation function data was one in all specimens. The equation for the specimens was: $y(n) - (0.99835 \pm 0.0035) \times y(n-1) = e(n)$, where $e(n)$ is white noise. **Conclusion:** In our hypothesis, the degeneration of the disc arises mainly, in the first step, from mechanical changes in the AF after mechanical loading, followed by an induced sequence of mechanical and biological changes. Microtrauma of the AF because of repetitive or sustained loading will cause plastic deformation of the AF, leading to a vicious cycle of disc degeneration.

Key words: analysis of intervertebral disc, nondestructive indentation test, time and frequency domains

INTRODUCTION

The intervertebral disc (IVD) is composed of the annulus fibrosus (AF), nucleus pulposus (NP), and two cartilaginous endplates. The AF consists of an inner AF (capsular AF) and an outer AF (ligamentous AF). The dense outer AF is composed of numerous (15-20) concentric rings of fibrocartilagenous tissue, the fiber arrangement of which is at an angle of 25-30° to the adjacent sheet. The outer AF travels into the bony vertebra, whereas the inner AF travels into the cartilaginous endplate. The inner AF is a fibrocartilagenous component of the annulus, with collagen fibers running concentrically and vertically, without a high degree of lamellar orientation of the outer AF¹. There is

also a thin transitional zone of fibrous tissue between the inner AF and the NP. The NP is a hydrated gelatinous material that sits in the posterior center of the disc. It is rich in hydrophilic aminoglycans. There are two endplates at the cranial and caudal junctions between the IVD and the vertebra. They consist of hyaline cartilage, which binds the discs to their corresponding vertebral bodies. Unlike the AF and NP, the structure and function of the endplate have not been characterized. The adult endplate is around 1 mm thick². The portion of the vertebra under the endplate is usually called the "subchondral bone". It belongs to the structures of the vertebra, but is also important in IVD physiology.

Degenerative changes affect all components of a mobile segment. In biomechanics, it has been pointed out that degeneration will involve a change in the tensile and compressive behaviors of the AF, which in turn will change the fatigue resistance and fatigue properties³⁻⁶, decrease the intradiscal (osmotic) pressure⁷⁻⁹, increase instability¹⁰⁻¹², change the viscoelasticity properties¹³, etc. Degeneration can occur when the accumulating microdamage from fatigue outpaces the remodeling response of the discs⁶. There is a correlation between degeneration, fatigue,

Received: December 12, 2006; Revised: March 23, 2007;
Accepted: April 23, 2007

*Corresponding author: Leou-Chyr Lin, Department of Orthopaedic Surgery, Tri-Service General Hospital, Taipei, Taiwan, Republic of China. No. 325, Sec. 2, Cheng-Gong Road, Neihu 114, Taipei, Taiwan, Republic of China. Tel: +886-2-8792-7185; Fax: +886-2-8792-7186; E-mail: lchlin66@hotmail.com

and instability⁵. Normal spine movement requires cyclic loading and the deformation of the discs. The repeated deformation of the discs may lead to a fatigue failure of the matrix. This failure may appear as fissures, cracks, or myxoid degeneration, or as microdamage in the macromolecular matrix. This change in the matrix interferes with the cell loading environment and leads to a functional change in the cell. The tensile mechanical behavior is altered in degenerative intervertebral discs. In a previous study, it was demonstrated that a degenerated AF will have a lower Poisson's ratio, lower failure stress, and lower strain energy density³. Decreases in both the failure stress and strain energy density to failure indicate that the degenerated AF will fail at lower stresses and require less energy to fail than a nondegenerate AF. Best and colleagues⁴ demonstrated that the compressive modulus of the AF is 0.34 MPa for nondegenerate AF and 0.15 MPa for degenerate AF. It is uncertain whether these material property changes are a result or a cause of IVD degeneration and failure. It is grossly evident that the NP will undergo progressive fibrosis, with a change in its material mechanical properties, during the process of degeneration. Measurements of intradiscal pressure in *in vitro* and *in vivo* studies have indicated significant decreases in the hydrostatic pressure and osmotic pressure of the degenerative IVD^{7,9,14}. The intrinsic and maximum pressures in the nucleus are inversely related to the anatomical grade of disc degeneration¹⁴. In an *in vivo* human study, it was noted that the intradiscal pressure was significantly reduced and corresponded to the degree of disc degeneration as estimated by magnetic resonance imaging⁹. In the process of degeneration, inhomogeneous and structural changes in the AF and NP caused a reduction in the diameter of the central hydrostatic region of each disc of approximately 50%, and the pressure within this region fell 30%, when a technique was used that measures the distribution of compressive stress within cadaver IVDs¹⁵.

The changes in viscoelastic properties in degenerative disc disease are still a controversial topic. One study concluded that the NP undergoes a transition from "fluid-like" behavior to a more "solid-like" behavior with aging and degeneration, primarily due to loss of hydration. This means that there is a decrease in the viscous component of the viscoelastic property¹³. A previous work discovered, using a nondestructive indentation test, that degenerative tissue has more viscoelastic mechanical properties for a given hydration level than does normal intact tissue¹⁶. This included property changes in stress relaxation, creep, and hardness.

Nondestructive indentation testing techniques provide

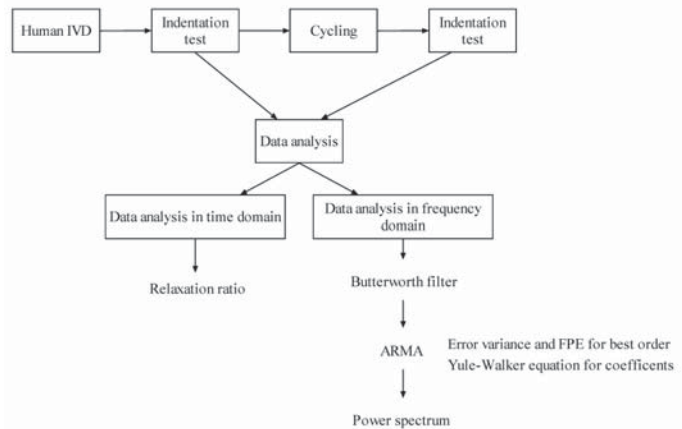


Fig. 1 The figure depicts the experimental procedures of this study. IVD, intervertebral disc; ARMA, autoregressive moving average model; FPE, final prediction error.

a means of determining elastic — plastic and viscoelastic material properties, with the key advantage of recording data at multiple points along a prescribed time or loading history. Previously, Evans and Lebow¹⁷ demonstrated a relationship between indentation testing data and the elastic modulus of bone. Wang et al.¹⁸ found that the fracture toughness of bone strongly correlates with indentation hardness, but not with mineral density, porosity, elastic modulus, yield strength, or ultimate strength. Indentation testing has also been used to determine the mechanical properties of soft tissues^{19–22}, whereas creep and stress relaxation methods have been used to characterize the viscoelastic nature of biomaterials^{23–30}.

The objective of this study was to use a nondestructive indentation technique to quantify the degradation of the material properties of the AF resulting from nontraumatic cyclic fatigue, and to analyze the data in both the time domain with the relaxation ratio and the frequency domain with a parametric autoregressive model^{31,32}.

METHODS

The method of this study is summarized in Fig. 1.

Seven human cadaver IVDs were included (4 from L3/4, 3 from L2/3) with a mean age of 52 years (range, 35–61). Specimen preparation involved cutting the pedicles and removing the posterior processes. The test unit consisted of hemivertebrae, potted in polyurethane, and the intervening disc. During the dissection and removal of the soft tissue around the disc, great care was taken to maintain disc hydration by misting it with saline at regular intervals.

A custom-made apparatus was constructed to apply a

2000 N cycling load at a rate of 0.5 Hz with a flexion angle of 5°. Each specimen was tested before and after 6000 flexion—compression cycles. The relaxation indentation test was applied with an MTS 858 materials testing system using a 2.5 mm hemispherical indenter. The indentation test was programmed to impact the whole IVD at the posterior center, with 10 preconditioning cycles, held for 60 s. The peak indentation pressure was 0.7 MPa for all specimens.

The data sampling rate was 10 Hz. The computational analysis was separated into the time domain and frequency domain.

In the Time Domain

The relaxation ratio (R) was defined by the following equation:

$$Rx = \frac{F_0 - F(t)}{F_0} \times 100\%$$

where F_0 is the initial indentation force and $F(t)$ is the force data gathered by the load cell over time. x is 0 for the specimen data before cycling and 1 for the specimen data after cycling. The relaxation change was calculated by:

$$R_{change} = \frac{R_t - R_0}{R_0} \times 100\%$$

A typical figure is shown in Fig. 2.

In the Frequency Domain

In statistical analysis, autoregressive moving average (ARMA) models, sometimes called “Box—Jenkins models” after G. Box and G. M. Jenkins³³, are typically applied to time series data. They involve a linear prediction formula that attempts to predict the output of a system based on previous outputs and inputs. For example, the output ($y[n]$) of a system is based on the previous outputs ($y[n-1]$, $y[n-2]$...) and inputs ($x[n]$, $x[n-1]$, $x[n-2]$...). We can derive a linear prediction model that involves determining the coefficients a_1 , a_2 , ... and b_0 , b_1 , b_2 , ... in the equation:

$$y[n] = a_1 \times y[n-1] + a_2 \times y[n-2] + \dots + b_0 \times x[n] + b_1 \times x[n-1] + \dots$$

The data analysis in the frequency domain was performed with an IBM-compatible computer using custom-designed software based on the Matlab (Mathworks, Natick, MA) programming environment (the Matlab code can be by request). The data were first processed with the Butterworth filter (order = 5, cutoff frequency at 1 Hz, gain at pass band (G_p) = -2 dB, gain at stop band (G_s) = -20 dB) because noise had a high power at 2.8 Hz. The sampling interval was 0.1 s. The autoregressive model defined here was p order:

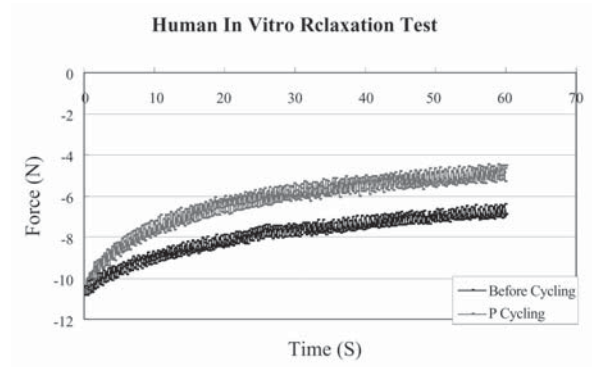


Fig. 2a Relaxation test of a specimen (cycling, as mentioned in the text, is 6000 cycles of a 2000 N load at a rate of 0.5 Hz; N, Newton).

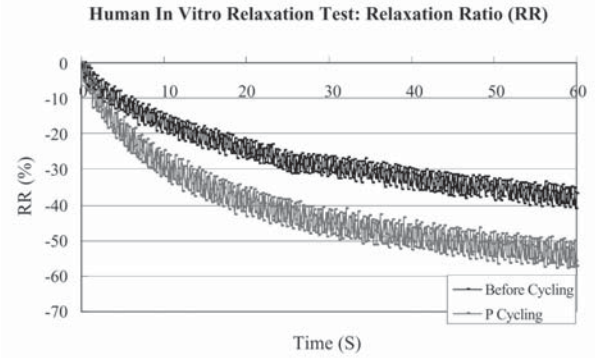


Fig. 2b Relaxation ratio of the same specimen (RR, relaxation ratio).

$$y(n) + a(1)y(n-1) + a(2)y(n-2) + \dots + a(p)y(n-p) = x(n)$$

where n is the time index, $y(n)$ is the output, $x(n)$ is the input, and p is the model order.

Both the error variance and the final prediction error (FPE) were used as predictors of the best order of the ARMA model^{31,32}. The coefficient was then determined by minimizing the total squared error, which was found by solving the set of equations called the Yule—Walker equations³². Because the relaxation function has a power distribution at most frequencies below 0.25 Hz, we calculated the power in regions with frequencies below 0.25 Hz and compared the power before and after cycling (Fig. 3).

RESULTS

In the time domain, the average relaxation ratio was 60.7% for the control and 74.4% after cycling. There was a 13.7%

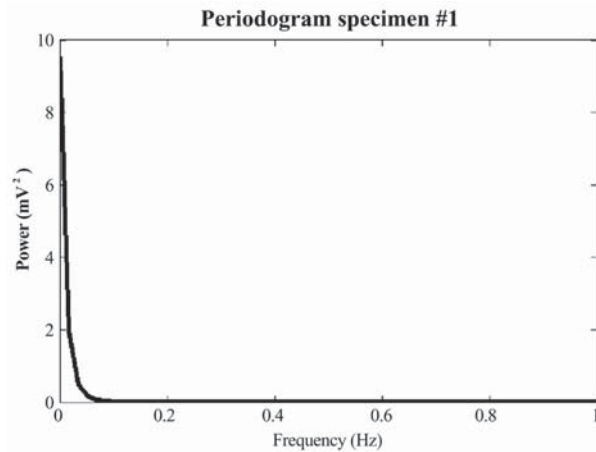


Fig. 3 The relaxation function was transformed into a periodogram in the frequency domain.

increase after cycling (Table 1).

In the frequency domain, the best order of the relaxation function data was 1 for all the specimens. The equation derived by the calculation described in the Methods was:

$$y(n) - (0.99835 \pm 0.0035) \times y(n-1) = e(n)$$

where $e(n)$ is white noise.

The periodogram revealed power distributed predominantly below 0.25 Hz. The power before and after cycling was 4.42×10^{-2} and 7.79×10^{-3} mV², respectively. The power decrease after cycling compared with the precycling power was 74.8% (Table 1).

DISCUSSION

In this study, a nondestructive indentation technique was used to quantify the degradation of the material properties of the AF resulting from nontraumatic cyclic fatigue. The relaxation ratio in the time domain was larger after cycling fatigue, indicating that this degradation can be quantified by the extension of relaxation in the indentation test. This is compatible with previous work that discovered that degenerative tissue has more viscoelastic mechanical properties than does normal intact tissue in the nondestructive indentation test¹⁶.

To the best of our knowledge, there is no report that analyzes viscoelastic relaxation time domain data in the frequency domain. Converting the data into a frequency domain revealed that the power of the low frequency in the periodogram remained only one fifth that of the control after 6000 cycles. The power change at low frequency is more sensitive than the quantification method in the time

Table 1

Specimen	R0	R1	(R1-R0)/R0	Power before cycling	Power after cycling
1	50.84%	70.98%	39.61%	4.26E-02	5.04E-03
2	69.59%	81.15%	16.61%	5.88E-03	2.15E-03
3	37.58%	46.25%	23.008%	2.11E-01	4.35E-02
4	65.95%	85.95%	30.32%	2.11E-03	7.03E-04
5	78.52%	93.45%	19.01%	8.25E-04	3.88E-04
6	47.07%	61.76%	31.20%	4.44E-02	3.56E-03
7	75.04%	81.34%	8.39%	2.58E-03	4.83E-04
Average	60.66%	74.41%	24.03%	4.42E-02	7.97E-03
SD	15.53%	16.09%	10.48%	7.60E-02	1.57E-02

R0: relaxation ratio before cycling; R1: relaxation ratio after cycling; Power units, minivolts squared.

domain in this study. The stress relaxation property is closely dependent on the past history of the output. In other words, the correlation between contingent outputs is high. The phenomenon was observed in this study using an autoregressive model by linear prediction of the output based on the previous output. The sign was easily confirmed with a simple Poincare plot, which showed a closed linear cluster with $y(n)$ vs $y(n+1)$. The cycling procedure, which simulates the degeneration of discs, causes a significant power decrease in the low frequency zone. Therefore, we can predict disc degeneration in the future using the power of a specimen in the frequency domain.

REFERENCES

1. Turek S, Buckwatler J. Turek's Orthopaedics: Principles and Their Application. Philadelphia: Lippincott Williams & Wilkins, 1994.
2. Edwards WT, Zheng Y, Ferrara LA, Yuan HA. Structural features and thickness of the vertebral cortex in the thoracolumbar spine. Spine 2001;26:218-225.
3. Acaroglu ER, Iatridis JC, Setton LA, Foster RJ, Mow VC, Weidenbaum M. Degeneration and aging affect the tensile behavior of human lumbar anulus fibrosus. Spine 1995;20:2690-2701.
4. Best BA, Guilak F, Setton LA, Zhu W, Saed-Nejad F, Ratcliffe A, Weidenbaum M, Mow VC. Compressive mechanical properties of the human anulus fibrosus and their relationship to biochemical composition. Spine 1994;19:212-221.
5. Yu CY, Tsai KH, Hu WP, Lin RM, Song HW, Chang GL. Geometric and morphological changes of the intervertebral disc under fatigue testing. Clin Biomech 2003;18:S3-9.
6. Adams MA, Dolan P. Could sudden increases in physical activity cause degeneration of intervertebral discs? Lancet 1997;350:734-735.

7. Naylor A, Smare DL. Fluid content of the nucleus pulposus as a factor in the disk syndrome; preliminary report. *Br Med J* 1953;4843:975-976.
8. Panjabi M, Brown M, Lindahl S, Irtam L, Hermens M. Intrinsic disc pressure as a measure of integrity of the lumbar spine.[see comment]. *Spine* 1988;13:913-917.
9. Sato K, Kikuchi S, Yonezawa T. In vivo intradiscal pressure measurement in healthy individuals and in patients with ongoing back problems. *Spine* 1999;24:2468-2474.
10. Panjabi MM. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. *J Spinal Disord* 1992;5:390-396; discussion 397.
11. Oxland TR, Panjabi MM. The onset and progression of spinal injury: a demonstration of neutral zone sensitivity. *J Biomech* 1992;25:1165-1172.
12. Panjabi MM. Clinical spinal instability and low back pain. *J Electromyogr Kinesiol* 2003;13:371-379.
13. Iatridis JC, Setton LA, Weidenbaum M, Mow VC. Alterations in the mechanical behavior of the human lumbar nucleus pulposus with degeneration and aging. *J Orthop Res* 1997;15:318-322.
14. Panjabi MM, Thibodeau LL, Crisco JJ 3rd, White AA 3rd. What constitutes spinal instability? *Clin Neurosurg* 1988;34:313-339.
15. Adams MA, McNally DS, Dolan P. 'Stress' distributions inside intervertebral discs. The effects of age and degeneration. *J Bone Joint Surg Br* 1996;78:965-972.
16. Hedman TP, Chuang SY, Syed B, Gray D. Biomechanical Benefits of Crosslink Augmentation in Spinal Discs. *Proceedings of IMECE'03 Washington DC*, November 15-21.
17. Evans FG, Lebow M. Regional differences in some of the physical properties of the human femur. *J Appl Physiol* 1951;3:563-572.
18. Wang XD, Masilamani NS, Mabrey JD, Alder ME, Agrawal CM. Changes in the fracture toughness of bone may not be reflected in its mineral density, porosity, and tensile properties. *Bone* 1998;23:67-72.
19. Kawchuk GN, Elliott PD. Validation of displacement measurements obtained from ultrasonic images during indentation testing. *Ultrasound Med Biol* 1998; 24, 105-111.
20. Scapino RP, Canham PB, Finlay HM, Mills DK. The behaviour of collagen fibres in stress relaxation and stress distribution in the jaw-joint disc of rabbits. *Arch Oral Biol* 1996;41:1039-1052.
21. Vannah WM, Childress DS. Modelling the mechanics of narrowly contained soft tissues: the effects of specification of Poisson's ratio.[erratum appears in *J Rehabil Res Dev* 1994;31(2):178]. *J Rehabil Res Dev* 1993;30:205-209.
22. Mow VC, Gibbs MC, Lai WM, Zhu WB, Athanasiou KA. Biphasic indentation of articular cartilage--II. A numerical algorithm and an experimental study. *J Biomech* 1989;22:853-861.
23. Dunn MG, Silver FH. Viscoelastic behavior of human connective tissues: relative contribution of viscous and elastic components. *Connect Tissue Res* 1983;12:59-70.
24. Geiger D, Trevisan D, Bercovy M, Oddou C. Harmonic and impulse rheological tests of biomaterials. *Biorheology* 1984;Suppl 1:193-200.
25. Har-Shai Y, Bodner SR, Egozy-Golan DB, Lindenbaum ES, Ben-Izhak O, Mitz V, Hirshowitz B. Mechanical properties and microstructure of the superficial musculoaponeurotic system. *Plast Reconstr Surg* 1996; 98:59-70; discussion 71-73.
26. Kazarian LE. Creep characteristics of the human spinal column. *Orthop Clin North Am* 1975;6:3-18.
27. Keller TS, Spengler DM, Hansson TH. Mechanical behavior of the human lumbar spine. I. Creep analysis during static compressive loading. *J Orthop Res* 1987; 5:467-478.
28. Koeller W, Meier W, Hartmann F. Biomechanical properties of human intervertebral discs subjected to axial dynamic compression. A comparison of lumbar and thoracic discs. *Spine* 1984;9:725-733.
29. Purslow PP, Wess TJ, Hukins DW. Collagen orientation and molecular spacing during creep and stress-relaxation in soft connective tissues. *J Exp Biol* 1998; 201:135-142.
30. Silver-Thorn MB. In vivo indentation of lower extremity limb soft tissues. *IEEE Trans Rehabil Eng* 1999;7: 268-277.
31. Jackson L. *Digital Filters and Signal Processing* 1995 (Springer).
32. Shiavi R. *Introduction to Applied Statistical Signal Analysis* 1999 (Elsevier).
33. Morris H, DeGroot A. A conversation with George Box. *Statist Sci* 1987;2:239-258.

