Bone 106 (2018) 204–210

Contents lists available at ScienceDirect

Bone

journal homepage: www.elsevier.com/locate/bone

Full Length Article

Effects of bone damage on creep behaviours of human vertebral trabeculae

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ARTICLE INFO

Article history:
Received 22 May 2017
Revised 18 October 2017
Accepted 24 October 2017
Available online 6 November 2017

Keywords:
Vertebral fracture
Creep
Trabeculae
Mechanical test
Biomechanics

ABSTRACT

A subgroup of patients suffering with vertebral fractures can develop progressive spinal deformities over time. The mechanism underlying such clinical observation, however, remains unknown. Previous studies suggested that creep deformation of the vertebral trabeculae may play a role. Using the acoustic emission (AE) technique, this study investigated effects of bone damage (modulus reduction) on creep behaviours of vertebral trabecular bone. Thirty-seven human vertebral trabeculae samples were randomly assigned into five groups (A to E). Bones underwent mechanical tests using similar experimental protocols but varied degree of bone damage was induced. Samples first underwent creep test (static compressive stress of 0.4 MPa) for 30 min, and then were loaded in compression to a specified strain level (0.4%, 1.0%, 1.5%, 2.5%, and 4% for group A to E, respectively) to induce different degrees of bone damage (0.4%, no damage control; 1.0%, yield strain; 1.5%, beyond yield strain, 2.5% and 4%, post-ultimate strains). Samples were creep loaded (0.4 MPa) again for 30 min. AE techniques were used to monitor bone damage. Bone damage increased significantly from group A to E ($P < 0.05$), with >30% of modulus reduction in group D and E. Before compressive loading, creep deformation was not different among the five groups and AE hits in creep test were rare. After compressive loading, creep deformation was significantly greater in group D and E than those in other groups ($P < 0.05$). The number of AE hits and other AE measurements during creep test were significantly greater in group D and E than in group A, B, and C ($P < 0.05$ for all). Data suggested that with the increase of vertebral trabecular bone damage, substantial creep deformation may occur even when the vertebra was under physiological loads. The boosted creep deformation observed may be attributed to newly created trabecular microfractures. Findings provide a possible explanation as to why some vertebral fracture patients develop progressive spinal deformity over time.

1. Introduction

Vertebral compression fracture is one of the most common fractures in the elderly [1] and often causes back pain and other symptoms that need clinical treatment. As the world's older population grows, healthcare costs for vertebral compression fracture have increased continuously [2]. Although most patients with vertebral compression fracture have favourable clinical outcomes after appropriate treatments, there is a subgroup of patients who developed progressive vertebral collapse over time, resulting in disabling back pain, spinal deformity, or even neurological complications [3,4]. It is hence important to identify these patients for preventive clinical interventions. To date, however, a screening tool to identify vertebral fracture patients who are at risk of progressive vertebral collapse and deformity is absent [5]. This is partly due to the limited understanding on the determinants of progressive vertebral collapse that followed vertebral fracture.

Previous studies have revealed that under physiological load a vertebra may continue to deform in a "creep" process [6]. Creep deformation is partially irreversible and may contribute to progressive spinal deformity. Further experiments observed that the speed of creep deformation may associate with the degree of vertebra damage [7]. In theory, creep in some fractured vertebrae may be accelerated to such an extent that vertebral collapse, a severe consequence of creep, occurs. Yet, a clear quantitative relationship between the degree of bone damage and vertebral creep deformation remained undetermined. Although vertebral components, including trabecular bone, cortical shell and endplate, all contribute to vertebral creep deformation, trabecular bone plays a dominant role [8,9]. Studies on creep behaviour of vertebral trabecular bone, therefore, can provide important information on vertebral creep.

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https://doi.org/10.1016/j.bone.2017.10.022
8756-3282/

The mechanism underlying bone creep is not fully understood, though some studies suggested that it may relate to bone viscoelasticity and bone damage accumulation [10–12]. The acoustic emission (AE) technique is a non-invasive and non-destructive approach used to monitor the integrity of engineering materials. This technique is based on the phenomenon that a material under an external load will produce sound (AE signal) when it starts to fail, such as the cracking noise from a broken tree when it falls. As a well-developed damage-monitoring technique, AE has been used in studies of cortical [13–16] and cancellous bones [17,18]. Yet, the AE technique has not been used to study vertebral creep.

Using the AE technique to monitor the creep behaviours of vertebral trabeculae, the current study aims to determine the relationship between bone damage and creep deformation in human vertebral trabeculae.

2. Materials and methods

2.1. Experiment design

Thirty-seven cylindrical trabecular bone samples from human thoracic or lumbar vertebrae were randomly assigned to 5 groups (group A–E). All bone samples used the same experimental protocols but different levels of damage loading. First, trabecular samples underwent creep loading (static compressive stress of 0.4 MPa) for 30 min. Then, the load was removed for 30 min to allow for recovery. Following recovery, samples in each group were loaded in compression to a specified strain (0.4% in group A; 1.0% in group B; 1.5% in group C; 2.5% in group D and 4% in group E) to induce bone damage. Finally, the samples were creep loaded (0.4 MPa) again for an additional 30 min.

2.2. Specimens

Five human spines (3 men and 2 women) donated for medical research were obtained from Science Care (USA). The donors were 36 to 73 years old (mean 57 years), with no known history of disease involving bone metabolism. Materials were stored at −20 °C till test. Each spine was thawed at 3 °C and T8 to L5 vertebrae were dissected for study. Each vertebra underwent fluoroscopy and only those integral vertebrae without suspicious pathology were included. As a result, 43 vertebrae were obtained, from which 21 were randomly selected for the current study (Table 1).

Cylinder cores of trabecular bone were obtained from each vertebra using an 8 mm external diameter diamond coated hole saw (THK Diamond Tools, China). During coring, the vertebra was clamped firmly to ensure that the longitudinal axis of the sample was perpendicular to the vertebral endplate. Samples were cooled with phosphate-buffered saline (PBS) during drilling. After coring, bone samples were visually assessed for any presence of mechanical damage. Samples showing any sign of damage were discarded. For each vertebra, typically 2 cylindrical bone samples (axial diameter 6.3 mm, height 19.3–28.4 mm) were obtained from left and right regions of the vertebral body. A third sample can be obtained from the middle region for some vertebrae of large size. Bone samples were sealed in plastic bags and stored at −20 °C until required for testing. As a result, 37 cylindrical bone samples were obtained, which were randomly assigned to group A to E. There are 9 samples in group A (1 sample from spine #1, #4 and #5, 2 samples from spine #3, and 4 samples from spine #2), 8 in group B (1 sample from spine #1, 2 samples from spine #3 and #4, and 3 samples from spine #2), 7 in group C (1 sample from spine #3, #4 and #5, and 4 samples from spine #2), 6 in group D (1 sample from spine #1 and #3, and 4 samples from spine #2), and 7 in group E (1 sample from spine #4 and #5, 2 samples from spine #3, and 3 samples from spine #2).

Table 1
Details of cadaveric spines in the study.

Cadaveric spine	Donor information		Vertebrae dissected	Vertebrae used in this study
	Age	Sex		
1	73	M	L1–L5	L2, L4
2	55	M	T9–L5	T10–L5
3	36	F	T8–L5	T8, T11, L1–L4
4	64	F	T8–L5	T10, L2, L4
5	56	M	T8–L4	T12, L2

2.3. Mechanical tests and AE measurement

The height and diameter of each sample was measured using a Vernier calliper. If necessary, a sample was shortened to keep the aspect ratio (height/diameter) <4, as recommended, to minimize end artefacts in mechanical testing [19]. The sample was then press-fit into two custom-made stainless steel endcaps, and held in place with cyanoacrylate adhesive. A custom-made jig was used to ensure that both endcaps were in alignment with the longitudinal axis of the cylinder sample [20] so that only uniaxial loading would occur during mechanical testing.

The mechanical test was performed using a Mach-1™ material testing device (Biomomentum, Canada) equipped with a 100 N load cell in a displacement resolution of 0.001 mm and a load resolution of 0.005 N. Load and displacement signals were sampled at 100 Hz. A custom-made testing chamber (70 mm × 70 mm × 45 mm) was fixed to the base plate of the testing device (Fig. 1). The sample was placed in the centre of the testing chamber and pressed by a flat-bottomed circular compression plate (20 mm in diameter). During testing, the chamber was filled with PBS solution at room temperature.

An AE sensor (R15UG, Mistras Group Ltd., UK; operating frequency 50–200 kHz) was attached to the testing chamber (Fig. 1) using cyanoacrylate adhesive [21]. Prior to experimental setup, the operation and performance of the AE transducer was confirmed with a pencil lead break test using an acrylic rod as outlined in ASTM. E976-10 [22]. Before each testing period, pencil lead break test was performed to verify the integrity of AE measurement setup. AE signals from AE sensor were transferred to the AE channel of the USB AE node (Model 1283, Mistras Group Ltd., UK), and load signals from the testing machine were input to

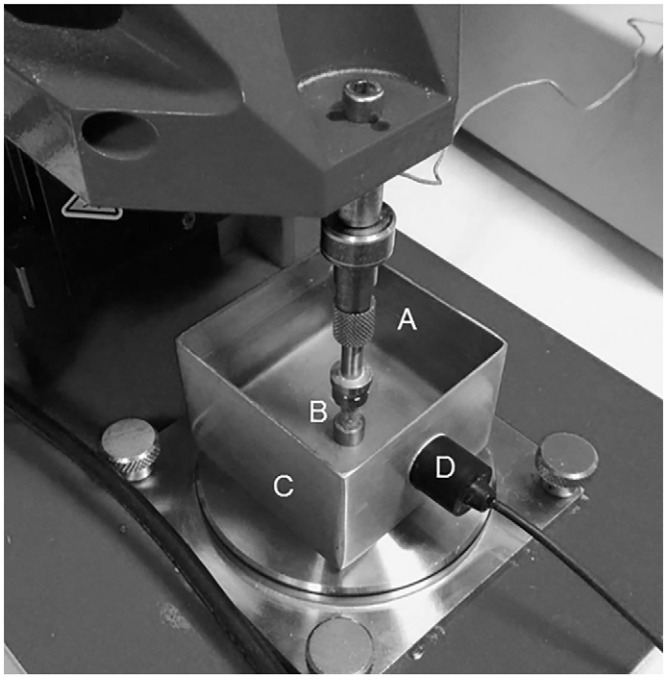

Fig. 1. Setup of mechanical testing apparatus (A, compression plate; B, bone sample; C, testing chamber; D, acoustic emission sensor).