Torso Flexion Loads and the Fatigue Failure of Human Lumbosacral Motion Segments

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Study Design. Spine loads associated with lifting a 9-kg weight were estimated at three torso flexion angles (0°, 22.5°, and 45°), and lumbosacral motion segments were cyclically loaded using these loads until failure or to a maximum of 10,020 cycles.

Objectives. To simulate the postures and loads experienced by the lumbar spine during repetitive lifting of moderate weights in different torso flexion postures, and to analyze the fatigue failure response of lumbosacral motion segments.

Summary of Background Data. Previous fatigue failure studies of lumbar motion segments have not reproduced the combination of spinal postures, loads, and load rates anticipated in different torso flexion postures during lifting tasks characteristic of those in occupational settings.

Methods. Twelve fresh human lumbosacral spines were dissected into three motion segments each (L1–L2, L3–L4, and L5–S1). Motion segments within each spine were randomly assigned to a simulated torso flexion angle (0°, 22.5°, or 45°) using a partially balanced incomplete block experimental design. Spinal load and load rate were determined for each torso flexion angle using previously collected data from an EMG-assisted biomechanical model. Motion segments were creep loaded for 15 minutes, then cyclically loaded at 0.33 Hz. Fatigue life was taken as the number of cycles to failure (10 mm displacement after creep loading). Specimens were inspected to determine failure mechanisms.

Results. The degree of torso flexion had a dramatic impact on cycles to failure. Motion segments experiencing the 0° torso flexion condition averaged 8,253 cycles to failure (±2,895), while the 22.5° torso flexion angle averaged 3,257 (±4,443) cycles to failure, and motion segments at the 45° torso flexion angle lasted only 263 cycles (±646), on average. The difference was significant at $P <$ 0.0001, and torso flexion accounted for 50% of the total variance in cycles to failure.

Conclusions. Fatigue failure of spinal tissues can occur rapidly when the torso is fully flexed during occupational lifting tasks; however, many thousands of cycles canbe tolerated in a neutral posture. Future lifting recommendations should be sensitive to rapid development of fatigue failure in torso flexion.

Recent reviews of the epidemiology literature have concluded that several work-related physical factors show consistent and positive associations with the occurrence of low back disorders.[1–3] Among the occupational factors exhibiting positive associations are jobs involving manual materials handling activities,[1,4–6] jobs involving frequent bending,[7–9] and jobs possessing a demanding workload.[10–12] A common theme among these occupational risk factors is that all place the lumbosacral spine under high levels of stress. Moreover, these stresses may be experienced several hundred times during a typical workday,[8,13] especially in physically demanding occupations such as construction or mining.

Spinal tissues, like all materials, are subject to fatigue failure.[14] It is apparent that occupational lifting tasks are capable of providing both loading magnitudes and frequencies sufficient to initiate and propagate fractures of the vertebral endplates and the underlying trabecular arcades in fatigue testing.[13,15,16] However, previous studies have not clearly elucidated the role of torso flexion during lifting tasks on the fatigue life of lumbosacral motion segments. Torso flexion influences a number of factors that impact fatigue life of spinal tissues. One obvious impact is that the magnitude of loading may be doubled or tripled when lifting in flexion compared with the neutral posture.[17,18] However, several other factors that may influence fatigue life are also affected by torso flexion. For instance, flexion of the spine may also alter the pathway of loading in the spine, with zygapophysial joints bearing up to 20% of an axial load in the neutral posture,[19] whereas the interbody joints bear a larger brunt of the load in flexion.[20] Biomechanical models also indicate that shear forces acting on the spine are considerably greater in torso flexion than in the neutral posture.[21] The rate of spinal loading (load rate) has been demonstrated to increase under conditions of increasing lumbosacral moments and trunk motion, both of which

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would be greater in lifts from flexed torso postures compared with upright postures.[22] Since all spinal tissues exhibit changes in material characteristics because of their viscoelastic nature, proper modeling of load rate is an important consideration in simulating spinal loading experienced in tasks such as occupational lifting.[23,24] To date, no studies have examined the fatigue life of motion segments when exposed to the combinations of motion segment orientation, spinal loads, and load rates associated with varying torso flexion angles experienced in common occupational lifting tasks. Accordingly, the current study simulates the spinal postures and loading conditions present when lifting a 9-kg load initiated at different angles of torso flexion, and examines the impact in terms of the fatigue life and failure modes of human lumbosacral motion segments.

Materials and Methods

Realistic simulation of spinal loading associated with lifting of a given load at different angles of torso flexion necessarily involves simultaneous adjustment of several factors. In this study, we simulate lifting a 9-kg load initiated at three torso flexion angles (0°, 22.5°, and 45°). Important factors that change with lifting in these flexion angles include the load magnitude, the load rate, and the degree of flexion of the individual motion segments of the spine. Additionally, different motion segments of the spine will experience a different combination of compressive and shear forces within each flexion angle. In this study, we have attempted to simulate the entire amalgamation of changes in spinal loading that occur as a given load is lifted in different torso flexion angles. Thus, lifting this given load in a neutral torso posture would consist of a lower magnitude of loading, a lower load rate (important because of the viscoelastic nature of spinal tissues), and motion segment angles characteristic of an erect torso posture. Lifting the load in a flexed posture would be associated with a higher load magnitude, load rate, and flexion of motion segments. Thus, when we describe changes in the fatigue life of motion segments in different torso flexion angles, it must be recognized that these changes are the result of the changes in the entire ensemble of factors that would be expected to change with torso flexion in occupational (or nonoccupational) lifting tasks.

Twelve fresh, frozen human lumbosacral spines (sacrum through L1) were procured from the anatomic gift program at Wright State University. Details of the specimens are contained in Table 1. As can be seen, the sample consisted of older spines, a product of the inability of the vendor to procure young specimens during the time-frame of the study. Spines were excised within 24 hours after death from subjects having no known history of spinal disease or prolonged bed rest before death. Intact lumbar spines were thawed at room temperature for several hours until tissues were sufficiently thawed for dissection into three separate motion segments: L1–L2, L3–L4, and L5–S1. Excess musculature, adipose tissue, and fascia were removed from the motion segments, and ligaments spanning multiple levels were sectioned. Ligaments confined to a single motion segment were preserved.

Anteroposterior and left lateral radiographs of each motion segment were taken at 50 kV, 5 mA, and a film focus distance of 38″. These initial radiographs served to detect the presence of existing defects in individual motion segments and were used to determine the relative angles of all four endplates of the motion segment.

Specimens were mounted in two trays using polymethylmethacrylate (PMMA). Specimens were aligned so that the superior endplate of the inferior vertebral body was placed parallel to the edge of the lower tray (*i.e.*, in a horizontal orientation), using the angles obtained in the radiographs. Once inferior vertebrae had been potted, specimens were x-rayed again and these radiographs were used to ascertain whether the superior endplates of the inferior specimens were indeed in a horizontal orientation, and to determine the Cobb angle of the motion segment (Figure 1).

Based on the Cobb angles measured in the second radiograph, the superior vertebra of each motion segment was potted so that an angle representative of a neutral posture for each motion segment was achieved when the upper and lower specimen trays were parallel to one another. Neutral posture Cobb angles for the three motion segments studied were operationally defined as: 20° for L5–S1, 8° for L3–L4, and 0° for L1–L2, based on data provided by Chen.[25] Flexion of the motion segments, necessary to simulate the 22.5° and 45° torso flexion angles, were calculated relative to the neutral motion segment posture.

It should be noted that achieving an overall torso flexion angle is the result of a summation of relatively small amounts of flexion at the motion segment level.[25–27] Indeed, the maximum amount of flexion possible in a lumbar motion segment in extreme flexion appears to be approximately 10°, with slightly

Table 1. Specimen Details

Specimen No.	Gender	Age (yr)	Cause of Death Listed on Death Certificate
1	M	77	Respiratory failure
6	F	82	Provisional
7	M	65	Blunt head trauma
8	M	74	Respiratory arrest
9	F	85	Pneumonia
10	M	80	Gastric carcinoma
11	M	85	Provisional
12	M	84	Congestive heart failure
13	F	91	Congestive heart failure
14	M	73	Cardiopulmonary arrest
16	M	79	Pancreatic carcinoma
17	F	93	Provisional

The sample consisted of 8 male and 4 female lumbosacral spines. The age of the specimens was 80.7 years (±7.8 years).

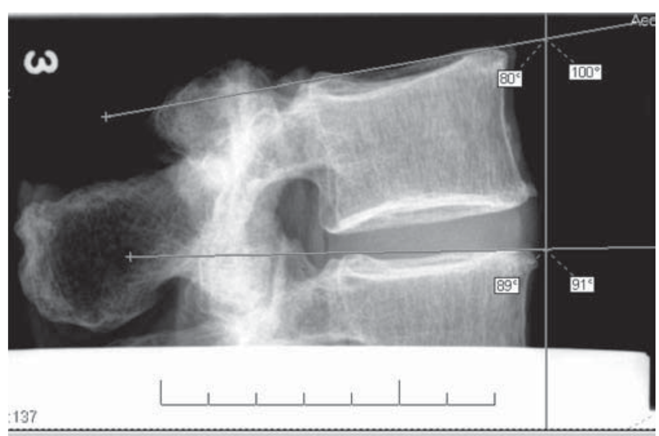

Figure 1. Radiograph illustrating measurement of Cobb angle for potted specimen.

Table 2. Loading Details for Motion Segments in the Present Experiment

Level	Trunk Posture (°)	Motion Segment Flexion From Neutral (°)	Resultant Force Angle (°)	Load Rate (N/s)	Minimum Load (N)	Maximum Load (N)	Total Cycle Time (s)	Loading Phase (s)	Relaxation Phase (s)
L1–L2	0	0	25	700	500	1300	3	1.14	1.86
	22.5	1	26	2100	750	2400	3	0.79	2.21
	45	3	24	4800	1050	3150	3	0.44	2.56
L3–L4	0	0	12	700	500	1300	3	1.14	1.86
	22.5	3	20	2100	750	2400	3	0.79	2.21
	45	6	31	4800	1050	3150	3	0.44	2.56
L5–S1	0	0	−23	700	500	1300	3	1.14	1.86
	22.5	3	−6	2100	750	2400	3	0.79	2.21
	45	6	7	4800	1050	3150	3	0.44	2.56

Angles of the resultant force are calculated with respect to the perpendicular of the superior endplate of inferior vertebral body for each motion segment. A positive angle is indicative of a posterior shear.

more flexibility than this in the central lumbar region and slightly less at either end.[26] Data from studies of radiographic and MRI studies used in this study suggest that lumbar motion segments are flexed approximately 50% to 75% of their maximum at 45° of torso,[25–27] as reflected by the values shown in Table 2.

Once both vertebrae of the specimen were potted, the specimen was placed in a test jig designed to mimic the loading experienced during lifting tasks, as determined by a dynamic biomechanical model.[21] A loading jig consisting of angled metal plates (as seen in Figure 2) was used in concert with a servohydraulic test frame (MTS Bionix 858, Eden Prairie, MN) to impose a combination of compression and shear forces on the motion segment for specified levels of flexion. Compression and shear loads at the lumbosacral junction were developed using analyses of lifting tasks starting in each torso flexion angle using a dynamic EMG-assisted biomechanical model. Estimates of the load rate experienced with lifts initiated in each posture were also provided by the model. Estimates of compression and shear loads at the upper spinal levels within each spinal position (θ°, 22.5°, and 45° torso flexion) were obtained by treating the lumbar spine as a rigid object and resolving the forces experienced at the S1 endplate into normal (compressive) and tangential (shear) forces at the upper levels based on the angle of the resultant force with respect to the L4 and L2 superior endplates. Thus, while the spinal orientation was different in each torso flexion angle, it was assumed to be rigid within each torso flexion angle so that upper level loads could be estimated. Table 2 provides load estimates derived in this fashion. Figure 3 displays graphically the cyclic loading regimen employed for each torso flexion angle.

A randomized block partially confounded factorial design was used in this experiment to evaluate the effects of posture and lumbar level on fatigue strength (number of cycles to failure) of lumbar motion segments.[28] The three motion segments dissected from each spine (*i.e.*, L1–L2, L3–L4, and L5–S1) were tested at three levels of simulated torso flexion (0°, 22.5°, and 45°), using load rates and forces characteristic of those experienced during *in vivo* lifting tasks in these postures, based on results of the biomechanical model. Blocking on spines helped control for the large amount of biologic variability typically observed when testing cadaver materials. In particular, the influences of age and gender are known to have a large influence on the strength of the lumbar spine motion segments.[29] The current design allowed control of this variability and more precise estimates of the effects of lumbar level and torso flexion. The design confounded a portion of the level × posture interaction; however, intrablock information was available to recover the within blocks portion of this interaction. As a result, the interaction between lumbar level and posture remained es-

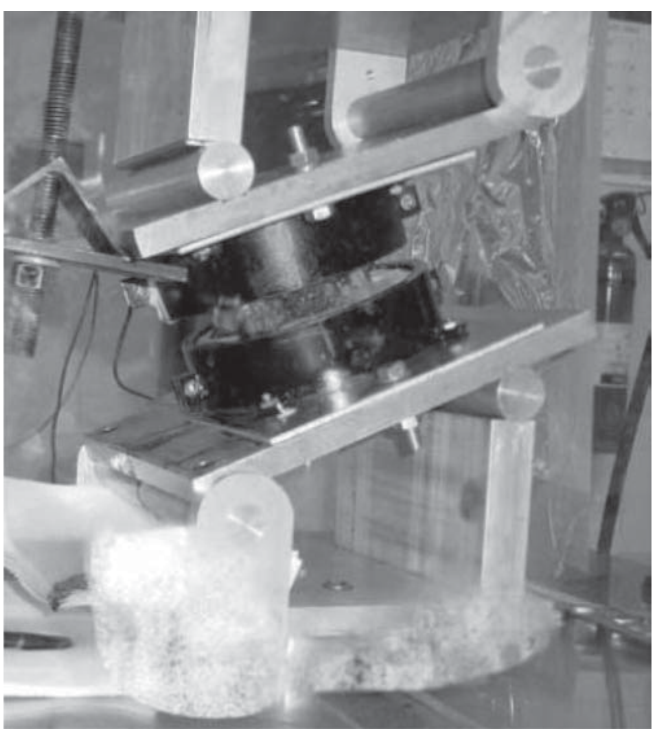

Figure 2. Illustration of loading jig with specimen attached, inside environmental chamber.

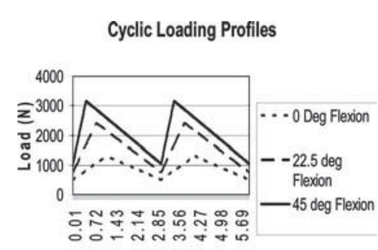

Figure 3. Loading profiles for simulated lifting of 9-kg load in 0°, 22.5°, and 45° of torso flexion.

timable. Two replications of the design were conducted, using 12 complete spines (36 motion segments).

All tests were performed in a humidified environmental chamber where the temperature was maintained at 37 C. A preload of 500, 750, or 1,050 N (dependent on the torso flexion angle) was placed on the specimen for a minimum of 15 minutes to precondition the specimen. The loss of height due to creep experienced by the specimen during this period was measured and recorded. Cyclic loads were placed on the specimens at a rate of 0.33 Hz (*i.e.*, one cycle every 3 seconds) to simulate a repetitive lifting task. Figure 3 depicts the loading cycles for simulated lifting of a 9-kg load in the three torso flexion conditions. Load deformation curves were collected at 100 Hz using an A/D data acquisition board and were monitored at regular intervals throughout the test. Fatigue-induced failure of the motion segments was operationally defined as occurring when the specimen deformed greater than 10 mm from its height at the conclusion of the 15-minute pretest creep loading period. Motion segments lasting the entire 10,020 cycles in the fatigue failure test (*i.e.*, censored observations) were subjected to a test of ultimate strength at the same load rate used in the cyclic loading regimen.

On completion of the mechanical testing, specimens were removed from the loading jigs and taken to the radiology clinic to evaluate damage using a lateral radiograph. These radiographs were read to evaluate the post-test status of the endplates, vertebral bodies, trabeculae, intervertebral discs, and posterior elements. Visual inspection of failed specimens was performed to evaluate external evidence of failure, particularly facet disruption and/or evidence of fracture to the vertebral bodies. The PMMA × specimen interface was scrutinized to ensure that failure did not occur at this interface. Zygapophysial joint damage was classified into three categories, based on visual inspection and functional tests involving movement of the motion segment. The first category was no damage to the joint capsule. Visual inspection confirmed that the joint was intact, and functional tests indicated normal joint motion. The second category of damage was increased joint laxity. Capsular ligaments were observed to be intact, although increased stretching of the ligaments was usually apparent, as was abnormal joint motion. The third category was frank disruption of the zygapophysial joint, where capsular ligaments were completely avulsed and the inferior and superior facets were completely disengaged. Failed specimens were dissected through the midplane of the intervertebral disc and digitally photographed, and a Galante disc degeneration score was assigned to each of the discs.[30] The endplates were then exposed and examined for damage.

Results

Changes in spinal loading associated with lifting a simulated 9-kg load in different angles of torso flexion had a highly significant influence on the fatigue life of lumbosacral motion segments ($P < 0.0001$). Motion segments loaded in the 0° condition averaged 8,253 cycles to failure (±2,895), specimens at 22.5° lasted 3,257 (±4,443), while those at 45° lasted an average of only 263 (±646) cycles (Figure 4). The ratio of maximum to minimum variance between torso flexion conditions for the untransformed data exceeded 3.0; therefore, a log transformation of cycles to failure was performed and the subsequent analysis variance was found to satisfy the

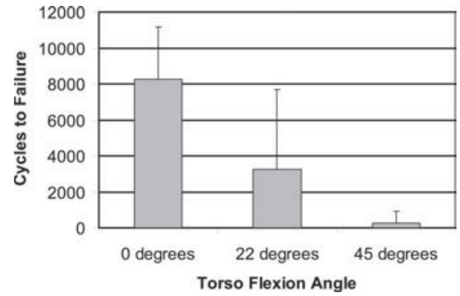

Figure 4. Cycles to failure by torso flexion angle. Spine loads associated with lifting 9 kg in 0° flexion resulted in long fatigue life; however, fatigue failure was rapid when exposed to loads associated with lifting the same weight in 45° torso flexion.

homogeneity of variance assumption.[31] The F statistic for torso flexion angle in the transformed analysis was $F_{2,16} = 36.10$ ($P < 0.0001$), and the estimates of the effect size for this factor indicate that it accounted for just over 50% of the variance in cycles to failure ($\theta^2 = 0.503$). Neither level of the motion segment nor the interaction of flexion × level significantly influenced cycles to failure ($P > 0.05$). Figure 5 provides data on the ultimate strength of specimens surviving the maximum number of loading cycles. Although neither motion segment level nor load rate was a significant factor in terms of ultimate strength ($P > 0.05$), this is likely because of a lack of power from the relatively small number of motion segments tested (11 specimens).

Table 3 contains a summary of post test analyses of the mechanisms and sites of failure for the lumbosacral motion segments used in this study. This table includes specimens failing *via* fatigue as well as those failing in tests of ultimate strength. Table 4 provides a breakdown of failure modes by simulated torso flexion angle for specimens experiencing fatigue failure. As indicated in this table, only four of 12 specimens failed by fatigue in the neutral posture, while mild and full flexion had 9 and 12 specimens fail *via* fatigue, respectively.

As can be seen, failure of the motion segments generally involved endplate fractures, compression/shear damage to the vertebral bodies, and/or disruption of the facet joints. Endplate fractures were frequently observed as a means of failure of the motion segment, often in combination with (and sometimes as part of) other vertebral body fractures. The classification system devel-

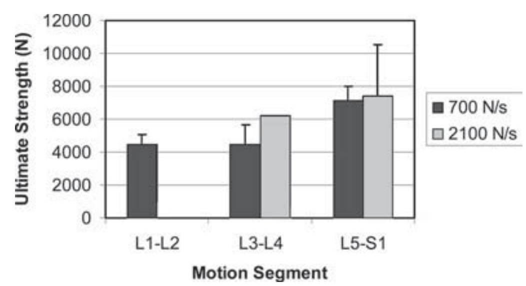

Figure 5. Ultimate strength for specimens lasting maximum number of loading cycles. Lack of a standard deviation bar indicates only one specimen was tested in this condition.

Table 3. Summary of Failure Mechanisms for All Specimens

Spine ID	Motion Segment	Torso Angle	Failure Mode	Disc Galante Grade	Inferior Endplate (superior VB)	Superior Endplate (inferior VB)	Superior Vertebral Body	Inferior Vertebral Body	Facet Disruption	Comments
11	L1–L2	22.5	FF	3	1,6	4	Comp/shear	Comp/shear	L	Left pedicle of L2 buckled
	L3–L4	45	FF	3		9	Comp/shear	Comp/shear		
	L5–S1	0	US	*	6	1,7				Neural arch fracture on L5
8	L1–L2	45	FF	2	6	6	Comp/shear		RL	L4 facet fractured
	L3–L4	0	US	2	6	6	Comp/shear			
	L5–S1	22.5	US	2	5	9		Comp/shear		
1	L1–L2	0	FF	3	2		Comp/shear		L	
	L3–L4	22.5	FF	2	2,1				L (lax)	
	L5–S1	45	FF	2	2,6		Comp/shear			
9	L1–L2	45	FF	3	4,5	5	Transverse			
	L3–L4	22.5	US	*	7	2	Comp/shear	Comp/shear	RL	
	L5–S1	0	FF	3	6	6		Comp/shear	RL (lax)	
7	L1–L2	22.5	FF	3	6.5	6	Comp/shear	LTD		Blood in posterior central disc
	L3–L4	0	US	2	6,3	2,4	Transverse			Annular protrusion, left pedicle of L4 fractured
	L5–S1	45	FF	4	6			Comp/shear	RL	Disc space narrowed
6	L1–L2	0	US	2	3,4	1	Comp/shear	Comp/shear		
	L3–L4	45	FF	2	1				RL (lax)	
	L5–S1	22.5	US	3				Comp/shear		Disc space narrowed
17	L1–L2	22.5	FF	3	1	6				
	L3–L4	45	FF	2	6	1,6	Comp/shear		L	
	L5–S1	0	FF	2		6			RL	
10	L1–L2	45	FF	2	1,4	8	Comp/shear			Shear fracture of L1 separated EP from VB
	L3–L4	0	US	2	7	8,4	Transverse		R,L (lax)	
	L5–S1	22.5	FF	3		6,4,5		Comp/shear	RL (lax)	
13	L1–L2	0	FF	3	6		Comp/shear		RL	Disc lax in torsional movement
	L3–L4	22.5	FF	2	1,6	6	Comp/shear			
	L5–S1	45	FF	2	1	1	Comp/shear			
16	L1–L2	45	FF	2	1		Comp/shear			
	L3–L4	22.5	FF	1	1,5		Comp/shear			
	L5–S1	0	US	2	1,6	6,4		Comp/shear	RL (lax)	Anular protrusion right posterolateral aspect
12	L1–L2	22.5	FF	3	2,7		Comp/shear		RL	
	L3–L4	0	US	3	2,7	1,6	Comp/shear		R,L (lax)	L3 spinous process pushed posterolaterally
	L5–S1	45	FF	3	2,5			LTD		
14	L1–L2	0	US	3	1,6	2	Comp/shear	Comp/shear		
	L3–L4	45	FF	2	1	6,5				
	L5–S1	22.5	FF	3	6,5	6,5				

See Figure 5 for endplate fracture classifications. Failure mechanisms included endplate fracture, compression and shear fractures of the vertebral bodies, and zygapophysial joint damage.
Note: * = Galante grade not determined, FF = fatigue failure, US = ultimate strength, LTD = localized trabecular disruption, L = left facet, R = right facet, (lax) = increased zygapophysial joint laxity.

oped by Brinckmann *et al*[13] did not fully characterize the nature of the endplate fractures observed in this investigation; thus, a modified version of the classification system was developed. This new classification system, illustrated in Figure 6, includes several of the Brinckmann *et al*[13] classifications but adds some additional classifications of endplate damage based on fracture patterns observed in the present study. The endplate fracture patterns derived from the Brinckmann *et al*[13] classification scheme include stellate, endplate depression, intrusion, step, and edge fractures. New endplate damage classifications include the ring fracture, lateral fracture, anteroposterior fracture, and soft (spongy) endplate.

The most frequent endplate damage findings were endplate depressions (78% of specimens) and stellate endplate fractures (47% of specimens). Lateral fractures (9 instances), ring fractures (8 instances), and endplates with soft, spongy areas (8 instances) were each present in approximately one fourth of all of the specimens tested. Step fractures (5 instances), intrusions (2), anteroposterior fractures (2), and edge fractures (2) were seen more rarely. As can be seen in Table 3, combinations of endplate damage modes were often observed. The reader is cautioned when examining these results that it is difficult to know the degree to which preexisting endplate damage may have been present and how existing damage may have influenced the observed results. While a significant portion of the damage would be expected from the cyclic loading imposed on the motion segments, it cannot be stated with certainty that the endplate damage observed was solely the product of these loads.

Another recurrent finding in post test examinations was compression/shear fractures of one or both of the vertebral bodies of the motion segment. Such fractures were more common in superior compared with inferior vertebral bodies. Transverse vertebral body fractures

Table 4. Failure Modes by Torso Flexion Angle for Specimens Failing by Fatigue

		0° (4 fatigue failures)		22.5° (9 fatigue failures)		45° (12 fatigue failures)	
Damage	Type	Inferior EP (Superior VB)	Superior EP (Inferior VB)	Inferior EP (Superior VB)	Superior EP (Inferior VB)	Inferior EP (Superior VB)	Superior EP (Inferior VB)
Endplate damage	None	1	2	1	3	1	5
	Stellate			5		5	2
	Lateral	1		2		2	
	Ring				1	2	1
	Soft endplate			3	1	2	3
	EP depression	2	2	4	4	4	4
	Step			1			
	Intrusion						1
	Edge						1
Zygapophysial joint damage		Left	Right	Left	Right	Left	Right
	None		1	5	7	9	10
	↑ laxity	1	1	3	2	1	1
	Disruption	3	2	1		2	1
Vertebral body fractures		Superior	Inferior	Superior	Inferior	Superior	Inferior
	Comp/shear	2	1	5	1	6	2
	Transverse					1	
	Localized trabecular disruption				1		1

Increasing torso flexion was associated with a greater no. of fatigue failures and increasing severity of damage.

were observed in only 3 cases, always in the superior vertebral body of the motion segment. Localized trabecular derangement was observed in radiographs in two inferior vertebral bodies showing no overt external evidence of fracture.

Examination of the failed specimens also turned up a number of cases where zygapophysial joints exhibited significant laxity or were disrupted entirely. Complete disruption of the zygapophysial joints was more common in the neutral (0°) torso posture, zygapophysial joint laxity was the more common finding at 22.5° of torso flexion, and damage to the facets was rarely seen in full flexion. Intervertebral discs tended not to incur a great deal of overt damage. However, examination of the sectioned disc on several occasions led to the observation that the internal lamellas of the disc appear to have experienced some distortion, as previously observed by Adams and Hutton,[32] which may have been the result of exposure to the experimental conditions.

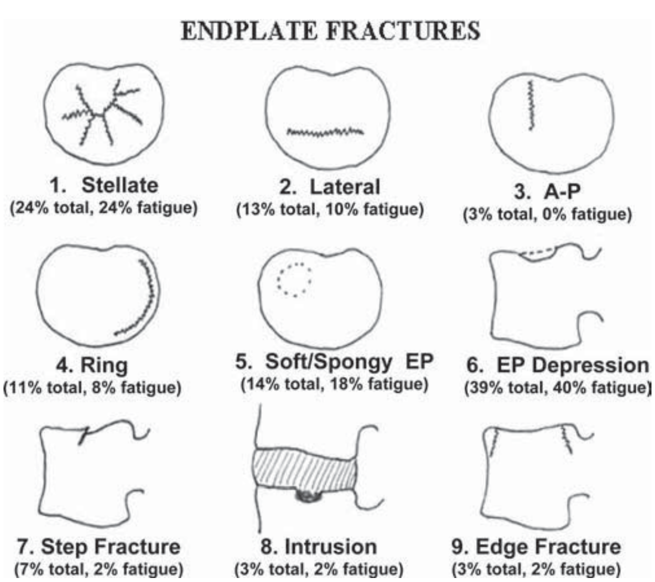

Figure 6. Vertebral endplate fracture classification system. Several classifications of endplate damage are derived from Brinckmann *et al.*[13] However, additional classifications were included based on fracture patterns observed in the present study. Numbers in parentheses represent the incidence of observed endplate fractures in all tests (combined fatigue and ultimate compressive strength tests), and in specimens failing *via* fatigue.

Discussion

Changes in spine loading attendant to simulated load lifting in a range of torso flexion postures had a dramatic impact on the fatigue life of lumbosacral motion segments. It is remarkable that, even with the elderly cohort of spines tested in this study, specimens could withstand several thousand loading cycles when subjected to a load simulating lifting a 9-kg weight in an upright torso position. Even with mild flexion, specimens lasted over 3,000 cycles, on average. However, the loading conditions associated with full flexion resulted in rapid fatigue failure of motion segments.

These findings are congruent with evidence from various sources associating torso flexion with low back disorders. For example, several recent epidemiologic studies have indicated that torso flexion is an important risk factor for low back pain.[7–9,33] The retrospective case-referent study by Punnett *et al*[9] disclosed a dose-response association between the degree of torso flexion (mild flexion, odds ratio [OR] = 4.9; severe flexion, OR = 5.7 *vs*. with neutral) and the risk of low back disorders, mirroring the fatigue life results in the current study. Holmstrom *et al*[7] found that time spent in torso flexion was strongly associated with severe low back disorders (OR = 2.6), while Marras *et al*[8] found that peak sagittal flexion angle was a significant predictor of membership in high-risk *versus* low-risk group membership for low back disorders (univariate OR = 1.6). In this study, the high-risk group had an average maximum sagittal flexion angle of 18°, while the low-risk group averaged 10° maximum sagittal flexion.[8] In addition, interventions designed to limit trunk flexion have been shown to be effective in reducing the incidence of low back pain.[34] Results of the current study may have etiologic significance regarding these relationships.

While the effect of torso flexion on fatigue failure of the lumbar spine is impressive, there are reasons to believe that the number of cycles to failure for workers in manual lifting would be greater than those reported here. In the first place, fatigue failure appears to occur more rapidly during *in vitro* testing compared with that experienced *in vivo*.[35] This may be the result of several factors, including the lack of physiologic remodeling capability in nonliving tissue and the possibility that different strain limits exist in physiologic as opposed to nonphysiologic conditions. Furthermore, the age of the current cohort is quite high, and specimens undoubtedly possess reduced bone mineral content compared with those of working age. Even relatively small reductions in bone mineral content can significantly affect vertebral ultimate strength, which will in turn impact fatigue life.[39] The authors are currently attempting to procure a younger cohort of spines for an additional replication of the current experimental design to evaluate differences in response between older and younger specimens. Nonetheless, despite the age and *in vitro* factors, spinal loading associated with lifting in different torso flexion angles clearly has a major impact on fatigue failure of lumbosacral motion segments.

Lumbar level did not have a significant influence on fatigue life, either alone or *via* interaction with torso flexion. It should be recognized that the rigid body assumption used to estimate loads at the upper levels imposed loads that were somewhat greater than those that would be experienced by this region *in vivo*. Although literature regarding loading at upper lumbar levels is sparse, estimates have suggested that the differences in loading between L5–S1 and L3–L4 during lifting may be relatively small (about 5%), while the difference between L5–S1 and L1–L2 loading may be between 10% and 20% or more.[36,37] Even so, differences between lumbar levels were not observed for fatigue life.

Evidence of motion segment failure was manifest most frequently in the vertebral endplates, vertebral bodies, and zygapophysial joints. For specimens failing by fatigue failure, endplate damage in the neutral posture tended to be characterized by endplate depression, with little evidence of fracture. Endplate fracturing was more evident with increased torso flexion loads. Zygapophysial joint damage was most evident in specimens failing by fatigue failure in the neutral posture, with progressively less damage as the motion segment flexed. This pattern of damage corresponds to previous observations that have shown damage to these joints resulting from impaction of the superior facets with the lamina below in neutral postures.[24] Compression/shear fracture patterns observed in the vertebral bodies were fairly frequent but may have been influenced to a degree by decreased bone mineral content of these older specimens and the pressures exerted by mounting the specimens in PMMA, which differs from the manner of loading that would be experienced *in vivo*.

Limitations of this study include customary caveats regarding *in vitro* testing, which have been well addressed by previous authors.[13,35,38] In addition, the old age of specimens in the current study is a limitation. While the effects of torso flexion would be expected to significantly impact the fatigue life of younger specimens in a similar manner, the absolute fatigue life of younger specimens would be expected to be greater because of increased bone mineral content and increased strength of the vertebrae.[15,39] In addition, the likelihood that older spines may experience spondylosis and other disorders that may affect the mode of failure must be acknowledged. A final limitation is related to the simplifying assumption that treated the lumbosacral spine as a rigid body for determination of upper level loads. Although this represents a common simplifying assumption made in biomechanics, it is not entirely satisfying since we know of no object that truly reacts as a rigid body, the lumbosacral spine included. While some advances have been made regarding an understanding of lines of muscle and ligament forces in different postures,[24,40,41] our understanding has not yet achieved a level of sophistication to estimate loads on various motion segments in different torso flexion postures with any high degree of accuracy.[42] Other models of spinal loading (*i.e.*, the "follower" load hypothesis) have recently been proposed that would suggest an increased ratio of compression to shear force compared with current models.[43,44] Results obtained in tests of fatigue failure will clearly be sensitive to the loading assumptions made by different models of spine loading.

The findings of this study would appear to have important implications for the current and future manual lifting guidelines. Based on the data presented in this study, current lifting guidelines may not pay sufficient attention to the role that fatigue failure may play in the development of low back disorders, particularly the rapid development of fatigue failure under conditions of full flexion of the lumbar spine. For example, the National Institute for Occupational Safety and Health revised lifting equation[45] provides a mild discount in weight for lifts originating near the floor (*i.e.*, requiring torso flexion) and a variable discounting factor for frequent lifting when a load originates at or below waist height (75 cm). However, the present data suggest that fatigue failure may be strongly influenced by a combination of these factors. That is, fatigue failure is most likely to occur when frequent lifting is performed in combination with torso flexion. The current recommendations treat lifting frequency the same no matter what degree of torso flexion is required. As a result, prevention of fatigue failure of spinal tissues in occupational lifting tasks may require greater sensitivity to the combined interactive impact of torso flexion and lifting frequency.

Conclusion

Simulation of spine loads when repetitively lifting a 9-kg weight indicates that torso flexion has a dramatic impact on the fatigue life of lumbosacral motion segments. The average number of cycles to failure in 0° trunk flexion was 8,253, specimens at 22.5° flexion averaged 3,257 cycles to failure, and specimens at 45° torso flexion lasted an average of only 263 cycles. The level of the motion segments did not significantly influence the number of cycles to failure, nor did the interaction of torso flexion × lumbar level. Endplate fractures, vertebral body fractures, and damage to the zygapophysial joints were the most common failure modes observed in fatigue testing. Endplate damage was characterized by endplate depression in loading conditions corresponding to upright postures, with overt endplate fracturing seen with the higher loads associated with torso flexion. Endplate damage patterns differed somewhat from previous studies, perhaps the result of changed loading pathways in torso flexion, and a revised classification system was proposed. Zygapophysial joint disruption was more frequent in the neutral posture, apparently because of impaction of the descending facet with the lamina of the inferior vertebral body.

Key Points

- Lifting a load in torso flexion as opposed to an upright posture will affect the magnitude of loading, load rate, and loading path experienced by the lumbosacral spine. These may all impact the development of fatigue failure of motion segments.
- Twelve fresh, frozen human lumbosacral spines were dissected into three motions each (L5–S1, L3–L4, L1–L2) and subjected to loads estimated to occur when lifting 9 kg in three torso flexion postures (0°, 22.5°, and 45°).
- Loads associated with the torso flexion angles have a dramatic impact on fatigue failure. On average, motion segments lasted 8,253 cycles in an upright posture, 3,257 cycles in 22.5° of flexion, and only 263 cycles in 45° torso flexion.
- Future lifting recommendations should be sensitive to the potential for rapid development of fatigue failure when lifting with a flexed torso.

References

1. Hoogendoorn WE, Poppel MNM, Bongers PM, et al. Physical load during work and leisure time as risk factors for back pain. *Scand J Work Environ Health* 1999;25:387–403.
2. National Academy of Science. *Musculoskeletal Disorders and the Workplace: Low Back and Upper Extremities*. Washington, DC: National Academy Press, 2001.
3. National Institute for Occupational Safety and Health [NIOSH], *Musculoskeletal Disorders and Workplace: A Critical Review of Epidemiological Evidence for Work–related Musculoskeletal Disorders of the Neck, Upper Extremity, and Low Back*. Cincinnati: U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, 1997.
4. Frymoyer JW, Pope MH, Clements JH, et al. Risk factors in low-back pain. *J Bone Joint Surg Am*. 1983;65:213–8.
5. Kelsey JL, Githens PB, White AA, et al. An epidemiological study of lifting and twisting on the job and risk for acute prolapsed intervertebral disc. *J Orthop Res* 1984;2:61–6.
6. Riihimaki H. Low-back pain, its origin and risk indicators. *Scand J Work Environ Health* 1991;17:81–90.
7. Holmstrom EB, Lindell J, Moritz U. Low back and neck/shoulder pain in construction workers: occupational workload and psychosocial risk factors: Part 1. Relationship to low back pain. *Spine* 1992;17:672–7.
8. Marras WS, Lavender SA, Leurgans SE, et al. The role of dynamic three-dimensional motion in occupationally-related low back disorders: the effects of workplace factors, trunk position, and trunk motion characteristics on risk of injury. *Spine* 1993;18:617–28.
9. Punnett L, Fine LJ, Keyserling WM, et al. Back disorders and nonneutral trunk postures of automobile assembly workers. *Scand J Work Environ Health* 1991;17:337–46.
10. Burdorf A, Govaert G, Elders L. Postural load and low back pain of workers in the manufacturing of prefabricated concrete elements. *Ergonomics* 1991; 34:909–18.
11. Heliovaara M, Makela M, Knekt P, et al. Determinants of sciatica and low-back pain. *Spine* 1991;16:608–14.
12. Videman T, Nurminen M, Troup JGD. Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation, and physical loading. *Spine* 1990;15:728–40.
13. Brinckmann P, Biggemann M, Hilweg D. Fatigue fracture of human lumbar vertebrae. *Clin Biomech* 1988;3(suppl 1):1–23.
14. Litsky AS, Spector M. Biomaterials. In: Simon SR, ed. *Orthopaedic Basic Science*. Rosemont, IL: American Academy of Orthopaedic Surgeons Press, 1994: 447–86.
15. Hansson T, Keller T, Spengler D. Mechanical behavior of the human lumbar spine: II. Fatigue failure during dynamic compressive loading. *J Orthop Res* 1987;5:479–508.
16. Hansson T, Keller T, Jonson R. Fatigue fracture morphology in human lumbar motion segments. *J Spinal Disord* 1988;1:33–8.
17. Nachemson AL, Elfstrom G. Intravital dynamic pressure measurements in lumbar discs. *Scand J Rehabil Med* 1971;3(suppl 1):1–40
18. Schultz AB, Andersson GBJ, Haderspeck K, et al. Analysis and measurement of lumbar trunk loads in tasks involving bends and twists. *J Biomech* 1982; 15:669–75.
19. Dunlop RB, Adams MA, Hutton WC. Disc space narrowing and the lumbar facet joints. *J Bone Joint Surg Br* 1984;66:706–10.
20. White AA, Panjabi MM. *Clinical Biomechanics of the Spine*. Philadelphia: Lippincott, 1978.
21. Granata KP, Marras WS. An EMG-assisted model of trunk loading during free-dynamic lifting. *J Biomech* 1995;28:1309–17.
22. Fathallah FA, Marras WS, Parnianpour M. An assessment of complex spinal loads during dynamic lifting tasks. *Spine* 1998;23:706–16.
23. Hawkins D. *Biomechanics of Musculoskeletal Tissues*. Davis, CA: University of California–Davis, 2001.
24. Bogduk N. *Clinical Anatomy of the Lumbar Spine and Sacrum*, 3rd ed. New York: Churchill Livingstone, 1997.
25. Chen Y-L. Predicting the vertebral inclination of the lumbar spine. *Ergonomics* 2000;43:744–51.
26. Pearcy M, Portek I, Shepherd J. Three-dimensional x-ray-analysis of normal movement in the lumbar spine. *Spine* 1984;9:294–7.
27. Jorgensen MJ. *Quantification and modeling of the lumbar erector spinae as a function of sagittal plane torso flexion* [Dissertation]. Columbus, OH: Ohio State University, 2001.
28. Kirk RE. *Experimental Design: Procedures for the Behavioral Sciences*, 3rd ed. Pacific Grove, CA: Brooks/Cole, 1995.
29. Jager M, Luttmann A. Compressive strength of lumbar spine elements related to age, gender, and other influences. In: Anderson PA, Hobart DJ, Danoff JV, eds. *Electromyographical Kinesiology*. Amsterdam: Elsevier, 1991:291–4.
30. Galante JO. Tensile properties of the human lumbar annulus fibrosus. *Acta Orthop Scand* 1967;38(suppl):1–91.
31. Box GEP. Some theorems on quadratic forms applied in the study of analysis of variance problems: I. Effect of inequality of variance in the one-way classification. *Ann Math Statist* 1954;25:290–302.

32. Adams MA, Hutton WC. Gradual disc prolapse. *Spine* 1985;10:524–31.
33. Riihimaki H, Tola S, Videman T, et al. Low back pain and occupation: a cross-sectional questionnaire study of men in machine operating, dynamic physical work and sedentary work. *Spine* 1989;14:204–9.
34. Snook SH, Webster BS, McGorry RW. The reduction of chronic, nonspecific low back pain through the control of early morning lumbar flexion: 3-year follow-up. *J Occup Rehabil* 2002;12:13–9.
35. Adams MA, Dolan P. Recent advances in lumbar spinal mechanics and their clinical significance. *Clin Biomech* 1995;10:3–19.
36. Daggfeldt K. *Biomechanics of Back Extension Torque Production About the Lumbar Spine*. Stockholm: Karolinska University Press, 2002.
37. *3D Static Strength Prediction Model* [computer program], version 4.3. Ann Arbor, MI: University of Michigan, 2000.
38. Adams MA. Spine update. Mechanical testing of the spine: an appraisal of methodology, results, and conclusions. *Spine* 1995;20:2151–6.
39. Hansson T, Roos B. The relation between bone mineral content, experimental compression fractures and disc degeneration in lumbar vertebrae. *Spine* 1981;6:147–53.
40. McGill SM. Dynamic low back models: theory and relevance in assisting the ergonomist to reduce the risk of low back injury. In: Karwowski W, Marras WS, eds. *The Occupational Ergonomics Handbook*. Boca Raton, FL: CRC Press, 1999:945–65.
41. Macintosh JE, Bogduk N, Pearcy MJ. The effects of flexion on the geometry and action of the lumbar erector spinae. *Spine* 1993;18:884–93.
42. Chaffin DB, Andersson GBJ, Martin BJ. *Occupational Biomechanics*, 3rd ed. New York: John Wiley and Sons, 1999.
43. Patwardhan AG, Havey RM, Meade KP, et al. A follower load increases the load-carrying capacity of the lumbar spine in compression. *Spine* 1999;24:1003–9.
44. Patwardhan AG, Meade KP, Lee B. A frontal plane model of the lumbar spine subjected to a follower load: Implications for the role of muscles. *J Biomech Eng Trans ASME* 2001;123:212–7.
45. Waters TR, Putz-Andersson V, Garg A. *Applications Manual for the Revised NIOSH Lifting Equation* [Publication No. 94-1994]. Cincinnati: DHHS (NIOSH), 1994:110.