Spine

SPINE Volume 39, Number 8, pp 664-672
©2014, Lippincott Williams & Wilkins

DIAGNOSTICS

Kinematic Magnetic Resonance Imaging to Define the Cervical Facet Joint Space for the Spine in Neutral and Torsion

Nicolas V. Jaumard, PhD,*† Jayaram K. Udupa, PhD,‡ William C. Welch, MD,* and Beth A. Winkelstein, PhD*†

Study Design. Prospectively acquire magnetic resonance images of the neck in normal subjects and patients with radiculopathy to measure and compare measures of the facet joint space thickness and volume.

Objective. The goal was to determine whether there is any difference in facet joint architecture between the 2 populations with the head in each of neutral and pain-eliciting rotation.

Summary of Background Data. Degeneration and altered mechanics of the facet joint can result in pathological nerve root compression and pain. Although lumbar facet joint space thinning has been reported in the context of low back pain, few studies have quantified the cervical facet joint space, especially in the context of pain.

Methods. The cervical spine of 8 symptomatic and 10 asymptomatic subjects was imaged in the sagittal plane in a 3T magnetic resonance scanner, using a T2-pulse sequence optimized for bone imaging. The facet joint space was identified and segmented in the acquired images. The thickness and volume of the facet joint space, and their changes between positions, were computed from the 3-dimensional representation for all cervical levels on both sides.

Results. Generally, the facet joint space thickness and volume were smaller in the symptomatic subjects than in the asymptomatic subjects. The differences were more robust on the left, especially in neutral and left torsion. The changes in both volume and thickness from neutral to torsion were also different in sign and magnitude at isolated joint levels between the 2 populations.

Conclusion. Quantification of the facet joint space architecture in the cervical spine of patients with radiculopathy is feasible using standard magnetic resonance imaging sequences. Measurements of the facet space thickness and volume, and their changes, from both pain-free and painful positions, can provide context for localizing potential sources of painful tissue loading.

Key words: MRI, cervical spine, facet joint, joint space, volume, thickness, radiculopathy, pain, axial torsion.

Level of Evidence: 3

Spine 2014;39:664–672

Neck pain is a common disability that affects upward of 50% of the general population, with an estimated annual incidence ranging between 10% and 21% depending on age, sex, and activities.[1–3] The facet joints are a potential source of axial and peripheral pain.[4,5] Specifically, facet joint pain has a prevalence of 54% to 67% in the patients treated for cervical pain.[6,7] Furthermore, facet spondylosis has been associated with cervical radiculopathy, and facet joint osteoarthritis accounts for up to 45% of the chronic low back pain cases.[6,8]

Cervical radiculopathy is produced by transient and/or sustained compression of the cervical nerve root that can result from disc herniation, bony trauma, and/or changes in the intervertebral joints.[8–11] It has been hypothesized that facet degeneration and reduction of the interfacet distance can also cause spinal spondylosis and stenosis.[12] The lumbar facet joint gap is smaller in patients with low back pain than in healthy asymptomatic volunteers.[13] In addition, facet cartilage degradation has been implicated as contributing to pain; a greater incidence of facet joint arthrosis is reported for lumbar stenosis than in healthy controls.[14]

The facet cartilage layers that permit the smooth motion of the adjacent vertebrae can be eroded because of aging, mechanical injury and wear, and osteoarthritis.[15–18] With the progression of cartilage thinning, the facet joint space also narrows, which can serve as a radiological marker of facet joint osteoarthritis.[13,19] The kinematics of the cervical facet joints and their articular surfaces are complicated by their complex anatomy that couples head and neck motions.[4,20] As such, head motions influence the relative positions of the

From the *Departments of Neurosurgery, †Bioengineering, and ‡Medical Image Processing Group, Department of Radiology, University of Pennsylvania, Philadelphia.

Acknowledgment date: July 24, 2013. First revision date: October 8, 2013. Second revision date: December 19, 2013. Acceptance date: January 2, 2014.

The manuscript submitted does not contain information about medical device(s)/drug(s).

Cervical Spine Research Society and the Catherine Sharpe Foundation funds were received to support this work.

Relevant financial activities outside the submitted work: grant, grants/grants pending, and expert testimony.

Address correspondence and reprint requests to Beth A. Winkelstein, PhD, Department of Bioengineering, School of Engineering and Applied Science, University of Pennsylvania, 210 S. 33rd St, Room 240 Skirkanich Hall, Philadelphia, PA 19104; E-mail: winkelst@seas.upenn.edu or http://spinepain.seas.upenn.edu/

DOI: 10.1097/BRS.0000000000000206

opposing articular surfaces in the cervical facet joints, which could manifest as changes in the joint space. In some patients with cervical radiculopathy, nerve root compression occurs only when there is head motion. It is hypothesized that the change in the joint architecture may be key to understanding relationships between neck biomechanics, the facet joint, nerve root, and pain.[21,22]

Imaging techniques can characterize osteoarthritis in the lumbar facets in patients with low back pain.[23–25] Radiography has proved useful to develop a qualitative assessment to screen facet joint osteoarthritis, but it can underestimate the degree of degeneration and can be insensitive to mildly diseased joints.[23] Both magnetic resonance imaging (MRI) and computed tomography (CT) provide finer resolution and have enabled a refined semiquantitative scoring of facet osteoarthritis.[24,25] Evaluating the presence of cartilage and bone erosions and osteophytes remains qualitative, whereas the narrowing of the facet joint space has been measured using CT and MRI.[24,25] Unlike radiography, CT and MRI do not depend on the relative orientation of the imaging system and the joint,[23,24] providing potential approaches for 3-dimensional (3D) measurements.[23,26] Although CT has better bone definition than MRI, concerns regarding radiation exposure and the ability to optimize imaging sequences for bone detection make MRI a potential tool for studying facet joint architecture.[19] Despite the potential pathological consequences of facet space narrowing and the availability of high-sensitivity MRI, no study has examined the feasibility of measuring the changes in facet joint architecture, including the facet joint space, because of changes in the kinematics of the opposing facets between pain-free and painful head positions.

The objective of this study was to measure the volume and thickness of the cervical facet joint space using MRI in subjects with cervical radiculopathy and in asymptomatic volunteers to investigate the potential utility of such an approach. Measurements were performed using sagittal magnetic resonance images acquired with the head in both a pain-free neutral position and in a pain-provoking position, for both subject groups. Outcomes were compared between the 2 populations and also between the 2 head positions to evaluate and normalize the changes in facet joint space dimensions in both populations.

MATERIALS AND METHODS

Age-matched asymptomatic and symptomatic subjects were recruited (Table 1). The symptomatic subjects were determined to have cervical radiculopathy by clinical examination and based on positive findings with electromyograph and pain radiating down 1 or both arms during left ($n = 4$) and/or right ($n = 3$) head rotation according to Neck Disability Index and Verbal Rating Score.[22] Neck range of motion was measured for each subject in bilateral axial torsion using a goniometer (CROM; Performance Attainment Associates) before imaging. The direction and angle(s) of the rotated position that elicited pain in the symptomatic subjects were recorded. Procedures were approved by the institutional review board; each subject provided consent prior to the start of the study. Procedures adhered to the guidelines of the Committee for Research and Ethical Issues of the International Association for the Study of Pain.

All subjects underwent MRI of the cervical spine (C2–C7) using a 3T TimTrio scanner (Siemens Medical Solutions; Malvern, PA) and a standard collar-shaped antenna. The symptomatic subjects were imaged first with the head/neck in the neutral position and then again with the head in the rotation position producing pain. The asymptomatic subjects underwent 3 imaging scannings with the head: (1) in neutral position, (2) rotated to the left, and (3) rotated to the right. The head rotation used for the asymptomatic subjects approximated the average rotation inducing pain in the symptomatic subjects. A FLASH 3D pulse sequence with a matrix size of 512×512, voxel size of $0.3 \times 0.3 \times 1$ mm,[3] and TE/TR = 4 ms/9 ms, optimized for bone visualization, was used to acquire 120 slices in the sagittal plane over a 7-minute scan period.

Images were analyzed using customized 3DVIEWNIX software to visualize and measure the facet space volume and thickness.[27] The digital slices including the left and right facet joint spaces at all cervical levels were identified (Figure 1A). For each slice, the facet joint space was identified as the space between the subchondral zones of the superior and inferior articular pillars, consisting of the 2 opposing cartilage layers and the gap between them. The bony articular pillars were delineated using the semimanual segmentation "live-wire" technique that identifies the peripheral pixels of a region of interest, based on a threshold method, for the demarcation of the facet joint space.[28] The segmented slices were then assembled and filtered to create and render the facet joint space as a 3D object[29] (Figure 1B). The volume and thickness of each 3D-reconstructed facet joint space were calculated by 3DVIEWNIX. The thickness was measured along the third principal axis of inertia of the joint space volume (Figure 1C). For 3 of the asymptomatic subjects (AS2, AS5, and AS8), the images during head torsion were inadequate for segmentation; those subjects were excluded from image analysis for comparison with the symptomatic subjects for head rotation. One symptomatic subject (S4) experienced pain only in extension; so, facet space dimensions were absent from comparisons.

Because preliminary comparisons showed that the facet space measurements were similar in male and female subjects for both groups, they were grouped for all other comparisons. Because of the small sample size of this study, it would otherwise not allow meaningful sex comparisons. Average volumes and thicknesses were compared between the symptomatic and asymptomatic groups for each of the neutral and rotated head positions. Also, within each group, those measures were compared between the left and right sides and between the 2 head positions. The average change in measures from neutral to torsion was also normalized to values in the neutral position and compared between the 2 populations.

Although the pain-provoking position corresponded to axial torsion for all but 1 symptomatic subject (S4), the direction of head rotation was not necessarily toward the painful

TABLE 1. Demographics and ROM for Symptomatic and Asymptomatic Subjects

Group	ID	Age (yr)	Sex	ROM (Degree) Left	ROM (Degree) Right	Radiating Pain	Pain Provoking Position (Direction/Degree)
Symptomatic	S1	56	M	62	65	Bilateral	L-AT/45
	S2	56	F	51	38	R	R-AT/40
	S4	55	M	76	58	R	Extension/30
	S5	39	F	42	41	Bilateral	R-AT/40
	S8	39	F	55	70	L (R goes right)	R-AT/40
	S6	38	M	85	100	L	L-AT/30
	S7	34	M	40	42	L	L-AT/10
	S9	61	F	40	60	L	L-AT/25
Average		47.3		56.4	59.3		
SD		10.7		16.9	20.3		
Asymptomatic	AS1	27	M	58	70	n/a	
	AS2	62	F	54	60		
	AS3	37	F	75	80		
	AS4	26	M	65	60		
	AS5	27	F	70	80		
	AS6	42	M	70	70		
	AS7	28	M	60	80		
	AS8	28	M	82	68		
	AS9	28	M	64	82		
	AS10	64	M	48	60		
Average		36.9		64.6	71.0		
SD		14.7		10.1	9.1		

ROM indicates range of motion; M, male; L, left; AT, axial torsion; F, female; R, right; n/a, not applicable.

side for each of the subjects. Therefore, statistical tests compared the groups in left torsion and in right torsion using *t* tests; comparisons between the left and right facet joints at each level were performed using paired *t* tests. Within each population, an analysis of variance tested comparisons between the cervical spinal levels.

RESULTS

Torsion range of motion was greater in the asymptomatic group (67.8 ± 9.9°) than in the symptomatic group (57.8 ± 18.1°) but not significantly (Table 1). Within each of the groups in neutral, there was no difference between the left and right facet volumes at any cervical level. This same

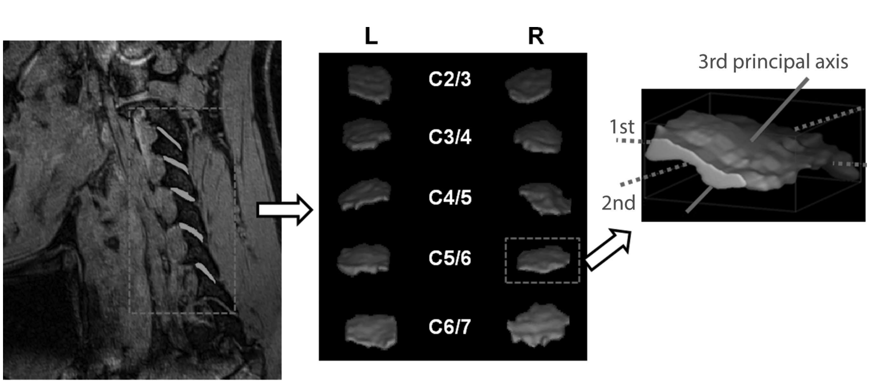

Figure 1. Segmentation procedure used to define facet joint space from sagittal magnetic resonance images. **(A)** Sagittal image showing facet joints. **(B)** Three-dimensional rendering of the left (L) and right (R) facet joint spaces shown from the posterior view. **(C)** Isolated C5–C6 joint space showing how the volume and thickness along the third principal axis of inertia were computed.

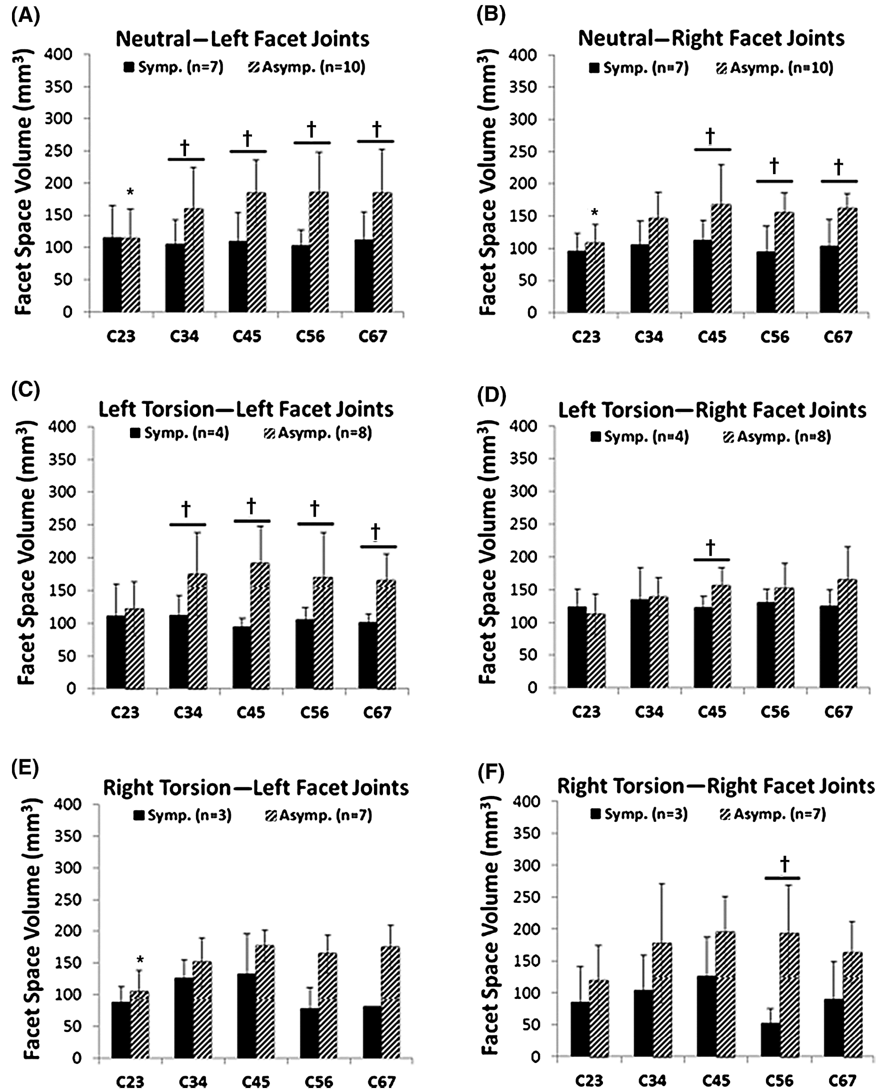

Figure 2. Average facet space volume for the symptomatic (Symp.) and asymptomatic (Asymp.) subjects with the head in neutral **(A and B)**, left torsion **(C and D),** and right torsion **(E and F)**. Symbols denote significant differences between C2–C3 (C23) and all other levels (*) ($P < 0.05$) and between the symptomatic and asymptomatic populations (†).

trend was also observed in both left torsion and right torsion (Figure 2 and Table 2). Similarly, there was no difference between the left and right joint space thickness at any level in either position, except in neutral for the asymptomatic subjects at C5–C6 and C6–C7, where thickness was significantly greater in the left joints (Figure 3B). In the asymptomatic group in neutral position, both the joint volume and the thickness of the left C2–C3 were significantly smaller ($P \leq 0.049$) than any other level (Figures 2A and 3A).

There was no difference in volume between neutral and torsion at any level, on either side, and in both populations (Figure 2 and Table 2). Thickness varied only at isolated levels in each group. For the symptomatic population, the average thickness in the right C3–C4 joint was greater ($P = 0.011$)

TABLE 2. Summary of Major Findings Comparing Facet Joint Space Dimensions Among Symptomatic and Asymptomatic Groups

No difference between the left and right facet joint space dimensions in either population.
No difference in facet space volume between head positions in either population.
The facet space volume is smaller in the symptomatic than in the asymptomatic population, on both sides below C4, in the neutral position.
The facet space volume is smaller in the symptomatic than in the asymptomatic population, on the left side below C3, in left torsion.
The facet space thickness is smaller in the symptomatic than in the asymptomatic population, only on the left side below C4, in both neutral position and left torsion.

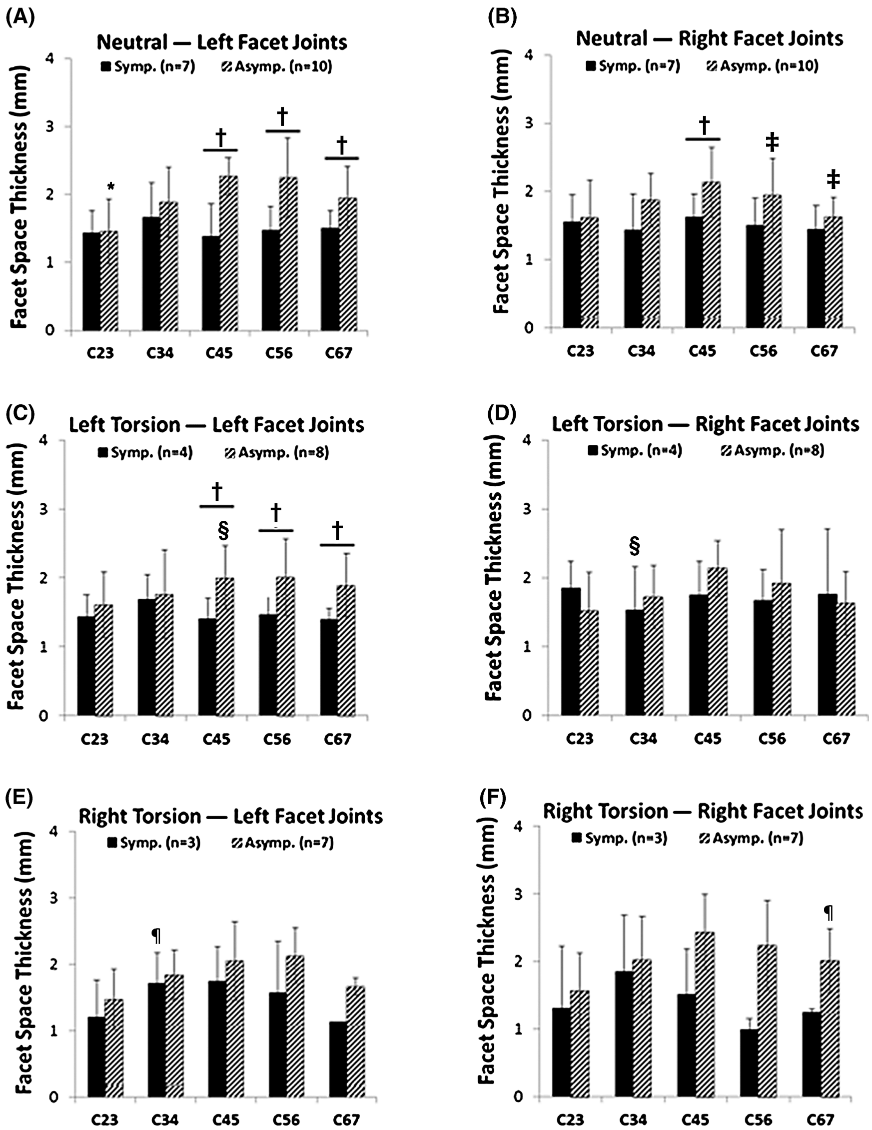

Figure 3. Average facet space thickness for the symptomatic (Symp.) and asymptomatic (Asymp.) subjects in neutral **(A and B)**, left torsion **(C and D)** and right torsion **(E and F)**. Symbols denote significant differences between C2–C3 (C23) and all other levels (*), between the symptomatic and asymptomatic populations (†), the left and right facet joints (‡), between neutral and left torsion (§), and between neutral and right torsion (¶) ($P \leq 0.05$).

during left torsion and smaller ($P = 0.016$) during right torsion than in neutral (Figure 3). For the asymptomatic subjects, the average thickness in the left C4–C5 joint was decreased from neutral ($P = 0.034$) during left torsion but was increased ($P = 0.034$) during right torsion in the right C6–C7 joint (Figure 3).

In both head positions, facet space volumes of the symptomatic subjects were nearly half those of the asymptomatic subjects at all levels except C2–C3 (Figure 2). Differences were significant on the left at all levels except C2–C3 in neutral and left torsion ($P \leq 0.042$) (Figure 2). On the right, the differences in volume were significant at C4–C5, C5–C6, and C6–C7 in neutral ($P \leq 0.031$), at C4–C5 in left torsion ($P = 0.034$), and at C5–C6 in right torsion ($P = 0.002$) (Figure 2). Similarly, the left and right facet space thickness in the symptomatic subjects was nearly 2.3 times smaller than in the asymptomatic subjects, at all levels except C2–C3 and C3–C4, in both neutral and torsion (Figure 3). These differences were significant at C4–C5, C5–C6, and C6–C7 in the left facets for both neutral ($P \leq 0.022$) and left torsion ($P \leq 0.043$) (Figure 3). There was no difference between the groups ($P \geq 0.455$) in thickness on the left during right torsion (Figure 3E). On the right side, thickness was up to 30% greater in the asymptomatic population at all levels in neutral, but this was significant only at C4–C5 ($P = 0.026$) (Figure 3B). There was no difference in right joint space thickness between the 2 populations in torsion to either side (Figure 3).

During left torsion, the normalized change in volume of symptomatic subjects on the left increased and was greater than that of asymptomatic subjects at C3–C4 and C4–C5 (Figure 4A). Similar differences were evident in the right joints but were significant only ($P \leq 0.038$) at C3–C4 and C4–C5 (Figure 4B). The normalized change in thickness tended to increase in the symptomatic population and decrease in the asymptomatic population (Figure 4). The only significant differences between the groups were at C3–C4 and C4–C5 on the left ($P \leq 0.043$) and at C2–C3 and C3–C4 on the right ($P \leq 0.049$) (Figure 4). During right torsion, the normalized

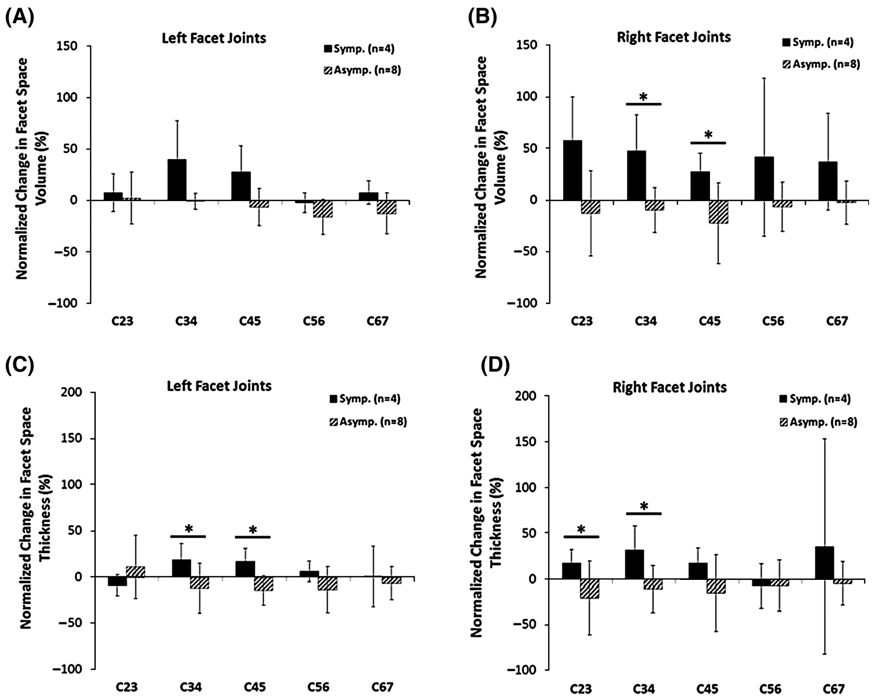

Figure 4. Normalized changes in volume **(A and B)** and thickness **(C and D)** in the left and right facet joints in the symptomatic (Symp.) and asymptomatic (Asymp.) subjects from neutral to left torsion. Significant differences (*$P \leq 0.05$) between symptomatic and asymptomatic groups were identified at isolated levels.

change in volume decreased in both groups only on the left (Figure 5). No significant differences were detected between the 2 populations. The normalized change in thickness was similar in both populations on both sides at all levels except on the right at C6–C7 where it was significantly greater ($P = 0.029$) in the asymptomatic group (Figure 5).

DISCUSSION

This is the first *in vivo* quantification of cervical facet joint space volume and thickness in pain-free and pain-provoking positions. Although only limited significant differences were detected (Table 2), facet joint space volume and thickness were smaller in neutral at all levels in subjects with cervical radiculopathy than in asymptomatic volunteers (Figures 2 and 3). The bilateral joint space thicknesses of asymptomatic subjects in neutral at all levels (Figure 3) agree with a prior report of the distance between the subchondral margins at C3–C4 (2.38 mm) for asymptomatic volunteers.[30] The difference in thickness between the studies may be due to differences in ages and measurement methods. Cascioli *et al*[30] measured the

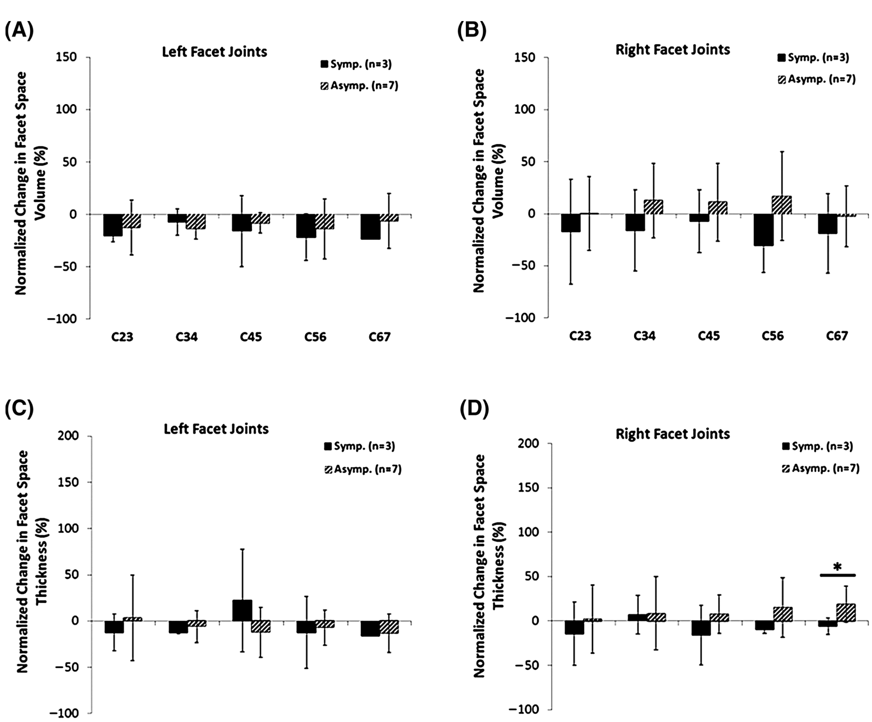

Figure 5. Normalized changes in volume **(A and B)** and thickness **(C and D)** in the left and right facet joints in the symptomatic (Symp.) and asymptomatic (Asymp.) subjects from neutral to right torsion. A significant difference (*$P = 0.029$) between symptomatic and asymptomatic groups was identified only at C6–C7 (C67) on the right.

linear joint space on planar radiographs, in contrast to the 3D rendering of the joint space used here (Figure 1). Also, the asymptomatic subjects in our study are older (Table 1) than the mean of 23 years old reported in that study.[30] The facet joint space thickness decreases with age in both healthy and low back pain subjects,[13] which would explain our lower average thickness (1.90 mm). A lack of differences in volume and thickness between the left and right joints in neutral in both populations (Figures 2 and 3) also agrees with literature.[13] The thicknesses for asymptomatic subjects in neutral are of the same order of magnitude as for those with normal facet joints.[24,31] Weishaupt *et al*[24] reported the healthy lumbar facet joint width, defined as the thickness of the 2 cartilaginous layers, to be 2 to 4 mm. Because cervical facets are smaller than lumbar joints, the normal cervical facet width is expected to be similarly less. Indeed, using sagittal sections of cadaveric human cervical spines, Yoganandan *et al*[31] approximated the cartilage layer to be 0.7 mm at C2 and 0.4 to 0.6 mm at C3–C7. Including the interfacet gap ranging from 0.3 to 0.7 mm[32] would place the estimated cervical joint space between 1.1 and 2.1 mm, which is consistent with our measurements.

There was no significant difference in volume and thickness of the facet joint space between neutral and torsion in either population. However, both of these measurements for the facet joint space were smaller in the symptomatic than in the asymptomatic group. Similarly, the normalized changes in volume and thickness from neutral to torsion were different in the 2 populations and were greater in the symptomatic group. The fact that both the volume and thickness of the joint were smaller in the symptomatic population supports the notion that changes in facet joint architecture may be related to pathology. In addition, the fact that the normalized changes in both volume and thickness were greater in the left joints of the symptomatic population when undergoing left torsion suggests that facet joint kinematics may be altered in the symptomatic group. However, large variations in the results prevent concluding whether such architectural changes cause or result from the pathology.

The differences in architectural relationships detected between the 2 populations were more significant on the left side than on the right side (Figures 2 and 3 and Table 2). This is likely due to the fact that the pain was diagnosed at the same cervical level on the left side for the symptomatic subjects who experienced pain when turning to their left. In contrast, for those symptomatic individuals with pain when turning to the right, the painful level varied across individuals. The volume was smaller in the symptomatic group below C3 on the left side in both the neutral position and the left torsion and below C4 on the right side only in neutral position. Similarly, the thickness was significantly smaller in the symptomatic group only on the left side below C4 in both positions. The volume and thickness of the left joint spaces being smaller in the symptomatic population in both the neutral and the left torsion (Figures 2 and 3) suggests that the morphology of the joint space and the architecture of the joint may indirectly contribute to modified kinematics of the spine. This is also supported by the finding that the normalized changes in volume and thickness during left torsion tended to increase in the facet joints of the symptomatic volunteers whereas they decreased in the asymptomatic group (Figure 4). Indeed, although the range of motion was not different between the 2 groups, it was smaller in the symptomatic group (Table 1). This lack of difference may be attributed to the fact that the symptomatic subjects experienced pain when rotating only to 1 side and that for half of them, pain was elicited only at the extreme rotation (Table 1). Although pathological tissue loading could be elicited at a single or multiple levels, the overall head rotation measured in symptomatic subjects could be reached through compensatory motions at nonpathological levels, which would suggest that the source of pathology may be localized at a single level, not necessarily corresponding to the radicular pain level. In addition, the increase in pain associated with head torsion suggests that nerve root impingement may occur from a transient change in the bone/nerve architectural relationship, such as foraminal narrowing, because of a reduction in facet joint space. Although the differences in facet joint space between the 2 groups were not overwhelmingly significant, the present data suggest that changes in facet joint architecture may provide anatomical evidence of pathophysiology that relates to symptoms of radicular pain. Indeed, these outcomes support the notion that pain and functional dysfunction are related, which can also alter joint architecture and mechanics, leading to further pathology.[33]

It could be hypothesized that the differences in facet joint architecture between symptomatic and asymptomatic subjects are a consequence, and not the cause, of pathology. For instance, altered joint architecture could result from disc degeneration at the index or adjacent level,[34,35] which was not evaluated here but is possible also using MRI. For 3 of the 4 symptomatic subjects (S1, S6, and S9) experiencing pain during left torsion, electromyograph identified the left C7 level as the pain source (data not shown). Although both the C6–C7 volume and the thickness were significantly smaller in symptomatic subjects during left torsion, that was also the case at other levels and was evident in neutral (Figures 2 and 3). If the altered architecture of the left C6–C7 facet is the source of pathology for those subjects, the C6–C7 normalized changes from neutral to left torsion would likely also be different from the asymptomatic subjects, which was not the case (Figure 4). Therefore, the altered facet joint architecture, facet mechanics, and changes in the architectural relationship between the nerve root and surrounding tissues are likely mutually influential and all contribute to radicular pain. For instance, cervical musculature plays an important role in modifying spinal mechanics and in altering facet mechanics and possibly leading to facet pain, in particular. Nearly 23% of the cervical facet capsular ligament is covered by muscle,[36] which can provide an additional load path for that joint when such muscles are activated. In addition, the multifidus can generate an axial torque of 0.3 N·m, which combined with the moment-generating potential of other paraspinal muscles could sufficiently modify the relative motions of adjacent vertebrae and alter the architecture of the facet joint.[37] Muscular weakness and atrophy have been reported in cases of neck and back pain,[38–41] which could also change facet joint space architecture. Certainly, additional studies are needed to assess the contribution of muscles on facet joint architecture and biomechanics.

Although preliminary, this is the first study to quantify cervical facet joint space architecture in pain-provoking conditions for both healthy subjects and those with pain from cervical radiculopathy. Although CT is more clinically relevant, use of MRI permits the analysis of other soft tissues,[22] and its relatively low radiation dosing enables longer imaging times. In addition, the results show that a standard T2 MRI sequence can permit measurements of the facet space with resolution sufficient to detect differences (Table 2). Despite a relatively small group size, the statistical power ranged between 49% and 100% for the differences detected between the symptomatic and asymptomatic groups, supporting the assertions made here. Although there can be a mismatch between clinical symptoms and MRI findings, the work in this study and a companion investigation that identified differences in the architectural relationships between the bony and neural tissues between these 2 groups[22] both support that changes in the architectural relationships between the many spinal tissues are associated with radiculopathy. This work detected a significant difference in facet joint space architecture between cervical radiculopathy and asymptomatic populations in both neutral and torsion positions. However, the data do not support a direct correlation between the changes in facet joint architecture during head rotation and radiculopathy symptoms. Nonetheless, this study does demonstrate that *in vivo* measurements of facet joint architecture can be performed using standard MRI techniques, which could be implemented for the diagnosis and follow-up of spinal arthrosis. Such measurements can potentially help better contextualize and identify the source of pain. Although these analyses are currently time-consuming, advances in medical image processing, particularly in anatomy recognition and delineation,[42] will improve automation for faster diagnosis and monitoring of clinically relevant measures.

➢ Key Points

- ❑ Facet joint space volume and thickness at the lower cervical levels are smaller in subjects with radicular pain than in asymptomatic subjects. These differences are significant in both the neutral and the left torsion position, especially in the left facet joints.
- ❑ There is no difference in the volume or thickness of the C2–C3 facet joint space between symptomatic and asymptomatic subjects in any head position.
- ❑ The normalized changes in volume and thickness from neutral to a pain-provoking head rotation to the left decreased in the C3–C4 joints on both sides in the asymptomatic subjects, whereas they increased in these same joints in the symptomatic subjects at the C7 level only on the left side.
- ❑ A standard MRI T2 sequence optimized for bone is feasible for measuring facet joint space dimensions and their changes and can potentially provide context for understanding mechanisms by which tissue loading relates to pain symptoms.

Acknowledgments

The authors acknowledge the Cervical Spine Research Society and the Catherine Sharpe Foundation for their financial support.

References

1. Côté P, Cassidy JD, Carroll LJ, et al. The annual incidence and course of neck pain in the general population: a population-based cohort study. *Pain* 2004;112:267–73.
2. Hogg-Johnson S, van der Velde G, Carroll LJ, et al. The burden and determinants of neck pain in the general population: results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *J Manipulative Physiol Ther* 2009;32 (2 suppl):S46–60.
3. Hoy DG, Protani M, De R, et al. The epidemiology of neck pain. *Best Pract Res Clin Rheumatol* 2010;24:783–92.
4. Vora AJ, Doerr KD, Wolfer LR. Functional anatomy and pathophysiology of axial low back pain: disc, posterior elements, sacroiliac joint, and associated pain generators. *Phys Med Rehabil Clin N Am* 2010;21:679–709.
5. Wang YX, Griffith JF, Ahuja AT. Non-invasive MRI assessment of the articular cartilage in clinical studies and experimental settings. *World J Radiol* 2010;2:44–54.
6. Boswell MV, Singh V, Staats PS, et al. Accuracy of precision diagnostic blocks in the diagnosis of chronic spinal pain of facet or zygapophysial joint origin. *Pain Phys* 2003;6:449–56.
7. Yin W, Bogduk N. The nature of neck pain in a private pain clinic in the United States. *Pain Med* 2008;9:196–203.
8. Caridi JM, Pumberger M, Hughes AP. Cervical radiculopathy: a review. *HSS J* 2011;7:265–72.
9. Borenstein D. Does osteoarthritis of the lumbar spine cause chronic low back pain? *Curr Pain Headache Rep* 2004;8:512–7.
10. Kalichman L, Hunter DJ. Lumbar facet joint osteoarthritis: a review. *Semin Arthritis Rheum* 2007;37:69–80.
11. Laplante BL, DePalma MJ. Spine osteoarthritis. *PM R* 2012;4 (5 suppl):S28–36.
12. Goel A, Shah A, Jadhav M, et al. Distraction of facets with intraarticular spacers as treatment for lumbar canal stenosis: report on a preliminary experience with 21 cases [published online ahead of print September 16, 2011]. *J Neurosurg Spine* 2013;19:672–7. doi: 10.3171/2011.8.SPINE11249.
13. Simon P, Espinoza Orías AA, Andersson GB, et al. In vivo topographic analysis of lumbar facet joint space width distribution in healthy and symptomatic subjects. *Spine* 2012;37:1058–64.
14. Abbas J, Hamoud K, Peleg S, et al. Facet joints arthrosis in normal and stenotic lumbar spines. *Spine* 2011;36:E1541–6.
15. Buckwalter JA, Mankin HJ. Articular cartilage: degeneration and osteoarthritis, repair, regeneration, and transplantation. *Instr Course Lect* 1998;47:487–504.
16. Temple-Wong MM, Bae WC, Chen MQ, et al. Biomechanical, structural, and biochemical indices of degenerative and osteoarthritic deterioration of adult human articular cartilage of the femoral condyle. *Osteoarthritis Cartilage* 2009;17:1469–76.
17. Bekkers JEJ, Creemers LB, Dhert WJA, et al. Diagnostic modalities for diseased articular cartilage—from defect to degeneration: a review. *Cartilage* 2010;1:157–64.
18. Saarakkala S, Julkunen P, Kiviranta P, et al. Depth-wise progression of osteoarthritis in human articular cartilage: investigation of

composition, structure and biomechanics. *Osteoarthritis Cartilage* 2010;18:73–81.
19. Gellhorn AC, Katz JN, Suri P. Osteoarthritis of the spine: the facet joints. *Nat Rev Rheumatol* 2013;9:216–24.
20. Jaumard NV, Bauman JA, Guarino BB, et al. ProDisc cervical arthroplasty does not alter facet joint contact pressure during lateral bending or axial torsion. *Spine* 2013;38:E84–93.
21. Farmer JC, Wisneski RJ. Cervical spine nerve root compression. An analysis of neuroforaminal pressures with varying head and arm positions. *Spine* 1994;19:1850–5.
22. Jaumard NV, Udupa JK, Siegler S, et al. A three-dimensional stress kinematic magnetic resonance imaging approach to detect potentially altered cervical architecture in radiculopathy. *Med Hypotheses* 2013;81:738–44.
23. Pathria M, Sartoris DJ, Resnick D. Osteoarthritis of the facet joints: accuracy of oblique radiographic assessment. *Radiology* 1987;164:227–30.
24. Weishaupt D, Zanetti M, Boos N, et al. MR imaging and CT in osteoarthritis of the lumbar facet joints. *Skeletal Radiol* 1999;28:215–9.
25. Kalichman L, Suri P, Guermazi A, et al. Facet orientation and tropism: associations with facet joint osteoarthritis and degeneratives. *Spine* 2009;34:E579–85.
26. Duan CY, Espinoza Orías AA, Shott S, et al. In vivo measurement of the subchondral bone thickness of lumbar facet joint using magnetic resonance imaging. *Osteoarthritis Cartilage* 2011;19:96–102.
27. Udupa JK, Odhner D, Samarasekera S, et al. 3DVIEWNIX: An open, transportable, multidimensional, multimodality, multiparametric imaging software system. *SPIE Proc* 1994;2164:58–73.
28. Falcao A, Udupa JK, Samarasekera S, et al. User-steered image segmentation paradigms: live wire and live lane. *Graphical Models Image Process* 1998;60:233–60.
29. Udupa JK, Odhner D. Fast visualization, manipulation, and analysis of binary volumetric objects. *IEEE Comput Graph Appl* 1991;11:53–62.
30. Cascioli V, Corr P, Till Ag AG. An investigation into the production of intra-articular gas bubbles and increase in joint space in the zygapophyseal joints of the cervical spine in asymptomatic subjects after spinal manipulation. *J Manipulative Physiol Ther* 2003;26:356–64.
31. Yoganandan N, Knowles SA, Maiman DJ, et al. Anatomic study of the morphology of human cervical facet joint. *Spine* 2003;28: 2317–23.
32. Rohlmann A, Lauterborn S, Dreischarf M, et al. Parameters influencing the outcome after total disc replacement at the lumbosacral junction. Part 1: misalignment of the vertebrae adjacent to a total disc replacement affects the facet joint and facet capsule forces in a probabilistic finite element analysis [published online ahead of print July 20, 2013]. *Eur Spine J* 2013;22:2271–8. doi:10.1007/s00586-013-2909-z.
33. Barker KL, Shamley DR, Jackson D. Changes in the cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. *Spine* 2004;29:E515–9.
34. Butler D, Trafimow JH, Andersson GB, et al. Discs degenerate before facets. *Spine* 1990;15:111–3.
35. Fujiwara A, Tamai K, Yamato M, et al. The relationship between facet joint osteoarthritis and disc degeneration of the lumbar spine: an MRI study. *Eur Spine J* 1999;8:396–401.
36. Winkelstein BA, McLendon RE, Barbir A, et al. An anatomical investigation of the human cervical facet capsule, quantifying muscle insertion area. *J Anat* 2001;198:455–61.
37. Anderson JS, Hsu AW, Vasavada AN. Morphology, architecture, and biomechanics of human cervical multifidus. *Spine* 2005;30: E86–91.
38. Chae SH, Lee SJ, Kim MS, et al. Cervical multifidus muscle atrophy in patients with unilateral cervical radiculopathy. *J Korean Acad Rehabil Med* 2010;34:743–51.
39. Hides J, Gilmore C, Stanton W, et al. Multifidus size and symmetry among chronic LBP and healthy asymptomatic subjects. *Man Ther* 2008;13:43–9.
40. Jordan A, Mehlsen J, Ostergaard K. A comparison of physical characteristics between patients seeking treatment for neck pain and age-matched healthy people. *J Manipulative Physiol Ther* 1997;20:468–75.
41. Kang J-I, Kim S-Y, Kim J-H, et al. The location of multifidus atrophy in patients with a single level, unilateral lumbar radiculopathy. *Ann Rehabil Med* 2013;37:498–504.
42. Udupa JK, Odhner D, Tong Y, et al. Fuzzy model-based body-wide anatomy recognition in medical images. *SPIE Proc* 2013;8671:86712B-1–7.