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Mechanism of Facet Load Transmission as a Hypothesis for Low-Back Pain

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Low-back pain has a complex and multi-faceted etiology. The articular facets have been shown to be load-bearing structures and may be a site for low-back pain. The aim of this paper is to establish the mechanism for the transmission of axial load across a facet joint and to propose a facet-related hypothesis for low-back pain. The mechanism of load transmission was studied by two methods. Lumbar segments were instrumented with an intervertebral load cell (IVLC) to measure disc load so that facet load could be deduced. The applied load was moved 10 mm anteriorly and 12.5 mm posteriorly from the center of the vertebral body. The facets then were separated from the body and loaded axially to determine their stiffness in tension and compression and to observe the failure mode of the joint. It was shown optically that compressive loading of the isolated facet joints was equivalent to spinal extension and tensile loading to spinal flexion. Lastly, a finite element model of a lumbar motion segment was developed to simulate the transmission of facet load and to study the effects of disc degeneration on facet loads. Results of the study on six lumbar segments revealed that the normal facets carried 3-25%. If the facet joint was arthritic, the load could be as high as 47%. Experiments on isolated facet joints revealed that they behaved as a stiffening spring in compression and were weak in tension. The resistance supplied by the capsular ligaments was an order of magnitude less than that in compression. When loaded to failure in compression, the inferior lumbar facets rotated posteriorly relative to the superior facets of the vertebra below and caused the capsule to rupture at about 6 kN without bony fracture. The transmission of compressive facet load occurs through contact of the tip of the inferior facet with the pars of the vertebra below. The data also show that an overloaded facet joint will cause rearward rotation of the inferior facet, resulting in the stretching of the joint capsule. The finite element model predicted an increase in facet load due to a decrease in disc height. The following hypothesis is proposed: Excessive facet loads stretch the joint capsule and can be a cause for low-back pain. Further study is needed to prove this hypothesis. [Key words: facet load, low-back pain]

THE ETIOLOGY of low-back pain is complex and multi-faceted. Half a century ago, Ghormley[6] coined the term "facet syndrome," implying that the articular facets might be one of the sites for low-back pain. Since that time, there have been sporadic clinical and neurologic studies of facet joint involvement. These include the work of Lewin,[9] Shealy,[21] Mooney and Robertson,[13] and Wyke.[23] The biomechanical basis for implicating the facet joint has not been demonstrated fully. However, there is sufficient evidence to indicate that the facets are capable of transmitting axial (vertical) compressive loads along the spinal column. It is now necessary to provide a biomechanical basis for the mechanism of load transmission and to propose a hypothesis implicating this joint as a site for low-back pain. This paper establishes such a mechanism and proposes a hypothesis, using data obtained from tests on isolated spinal segments of the lumbar spine.

The spinal motion segment consists of two vertebrae and the intervening disc. Its anatomy is rather complex and has led to a certain degree of uncertainty regarding the anatomic function of the various portions of the motion segment; in particular, the ability of the articular facets to transmit a vertical load. The vertebral body and the intervertebral disc were considered to be the principal load-bearing elements of the spine and the facets were thought of as motion limiters, which restricted the motion of one vertebra relative to an adjacent vertebra. Such motions include sliding in the antero-posterior direction, axial torsion about a longitudinal axis, and spinal extension. There is a need to define the precise role of the facets in limiting spinal extension because they form a synovial joint, which is low in friction, and the articulating surfaces are almost vertical.

This paper reviews the literature related to axial facet loads, describes the experiments carried out to determine them quantitatively, and uses a finite element model to study the effects of disc degeneration on facet load. It then explains the mechanism of facet load transmission and proposes a hypothesis for low-back pain.

A REVIEW OF THE LITERATURE

Nachemson[14] measured intradiscal pressure with and without posterior elements and concluded that the facets were capable of carrying approximately 20% of the vertical load. This conclusion was retracted 3 years later,[15] but it only served to spur on further investigation of this phenomenon. However, early research was aimed at the determination of force-deflection properties of intervertebral joints, and the work of Brown et al,[4] Rolander,[20] and Markolf[12] needed to be analyzed to ascertain the role of the facets. Rolander's stiffness values were higher than those of Brown et al and of Markolf, as shown in Figure 1. This difference was attributable to the fact that Rolander used specimens with the posterior elements intact. Lin et al[10] indicated that the slope of the load-deflection curve is higher for posterior eccentric loading of the vertebral body than that for anterior eccentric loading. This, again,

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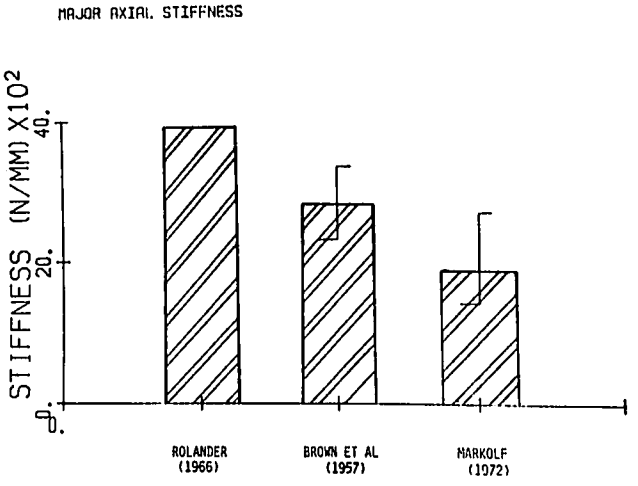

Fig 1. Stiffness data of lumbar segments obtained by Rolander,[20] Brown et al[4] and Markholf.[12]

is an indication of the role of the articular facets in supporting load.

The quantification of facet load first was performed by Prasad et al[18] who were able to increase the level of acceleration for vertebral fracture by 80% by simply hyperextending the spine of embalmed cadavers during caudocephalad acceleration. The computation of facet load required the use of an intervertebral load cell (IVLC), which measured the load borne by the disc and an assumption that the total load borne by the spine was equal to the product of the vertical acceleration and the mass of the body above the spinal level of interest. The difference between the total load and that measured by the IVLC was termed facet load. Figure 2 shows the computed facet load and confirming evidence from a strain gage mounted on the posterior surface of the lamina. Hakim and King[7] showed that there was a significant facet load when a spinal segment was loaded statically. They also duplicated the dynamic loading sequence in a materials testing machine and measured the total load. The facets again were shown to be load-bearing structures.

More indirect evidence of facet load was obtained by Adams and Hutton,[1] who loaded isolated lumbar segments in compression using a specially designed angle plate, which could control the direction of load application. They concluded that a lordotic spine (in an erect standing posture) transmitted 16% of its load via the facets. The authors also stated that with increasing hyperextension of the spine, more of the load would be borne by the facets, and that this load was a mechanism for the limitation of spinal extension. The load was assumed to be transmitted via bony contact, but the exact mechanism of load transmission was not delineated.

Lorenz et al[11] attempted to measure vertical facet load by means of a pressure sensitive film (Prescale by Fuji). The film was inserted into the joint space and the vertebral segment was subjected to a series of compressive loads. Their data were compared with those of Hakim and King.[7] Figure 3 shows that the facet load measured by Lorenz et al decreased with increasing total load, a trend opposite to that of previous data. Because of the fact that the facet joint is synovial and that the joint surfaces are almost vertical, it is likely that the measured contact pressure between the joint surfaces is not representative of the vertical facet load alluded to by Hakim and King[7] and Adams and Hutton.[1]

There is also clinical evidence of the load-bearing role of the facets. Osteoarthritis of the facets with deformation of the joint capsule is seen in autopsy. Adams and Hutton[2] were of the opinion that this was a symptom for low-back pain. In a recent comment by Andersson,[3] he stated that osteoarthritis of the facet joint was commonly found in the presence of a variety of spinal deformities.

MATERIALS AND METHODS

This study is divided into three parts: two experimental sections and a modeling effort. Lumbar segments first were tested to quantify the fraction of load borne by the facets as a function of the location of the applied load. Isolated facets then were loaded axially to determine their stiffness characteristics and their failure mode in both compression and tension. For the purposes of this paper, the term "facet load" is defined as an axial load transmitted by the facets. Lastly, a finite element model was used to verify the measured facet load and to study the effects of disc degeneration.

Facet Load Measurement. Direct measurement of facet load cannot be made until the mechanism of load transmission is understood fully. Since this has not been accomplished, an indirect method needs to be used to perform the measurement from which the mechanism of load transmission then can be deduced. The use of an IVLC still was considered to be the most reliable means of measuring the facet load indirectly. For a lumbar segment tested *in*

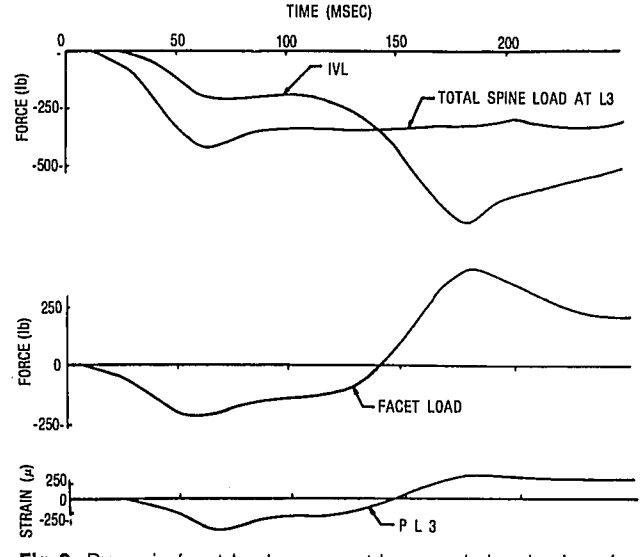

Fig 2. Dynamic facet load measured in an embalmed spine of an intact cadaver during caudocephalad acceleration.

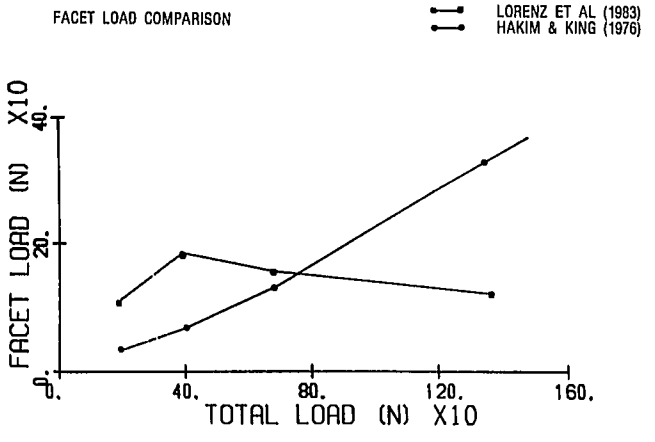

Fig 3. Comparison of static facet load measured by Lorenz et al[11] and Hakim and King.[7]

vitro in a materials testing machine, the facet load is the difference between the total load and that measured by the IVLC, the disc load. This statement underestimates the facet load, if the tension developed in the posterior ligaments are not negligible, particularly when the segment is in flexion. A study of the flexion loading condition was made to show that ligamentous loads were small in comparison with the facet load. The analysis is given in the modeling section of this paper. The use of the IVLC suffers from the disadvantage of having to cut the anterior longitudinal ligament.

Description of the intervertebral load cell (IVLC). A new IVLC was designed and fabricated to measure axial load and bending moment about two orthogonal axes in the transverse plane. It is approximately 12 mm thick and 38 mm in diameter and is composed of three, individual, uniaxial, load cells set between two parallel plates. A double-bladed saw was used to insert the device. A slot was cut in the vertebral body of a lumbar vertebra, using an anterior approach. The width of the slot was made equal to the height of the IVLC, which then was inserted and held in place by means of a self-locking plastic strap around the processes. This method was essentially the same as that described by Prasad et al.[18] The effect of replacing a portion of the body by a load cell was studied by comparing the stiffness of the segment with and without the IVLC.

Method of mounting spinal segment. Lumbar segments were used in this study. A two- or three-vertebrae segment was used to measure facet load. The superior portion of the top vertebra was embedded in a cup containing a low melting point alloy (Ostalloy, melting point, 47 C). The inferior portion of the bottom vertebra also was embedded in a cup of Ostalloy by inverting the assembly and dipping the bottom vertebra into the alloy without changing the natural curvature of the segment. A jig was used to ensure that the top and bottom surfaces of the cups were parallel.

Testing procedure. After the alloy solidified, the entire assembly was placed in an Instron testing machine. The lower support consisted of an adjustable rotary table, which was used to move the segment in the antero-posterior direction for the application of eccentric loads. The load was applied through a 750-mm long plate with a semi-circular loading head, as shown in Figure 4. The load was symmetric with respect to the mid-sagittal plane. Figure 5 shows a segment under test in the Instron machine with the IVLC in place.

The first step in the testing procedure was to subject an intact, lumbar segment to a series of eccentric loads. The eccentricity was measured from the center of the body and varied from 10-mm anterior to 12.5-mm posterior to the center at 2.5-mm intervals. The compressive load was applied as a ramp function at a rate of 93 N/s up to a maximum of 1,112 N. This load was chosen to permit repeated applications of load to the specimen without fracture. The unloading rate was also at 93 N/s. There was a 5-minute rest period between each loading cycle to permit the specimen to recover partially. After completion of this series of tests, the IVLC was inserted and the experiment repeated to measure facet loads. The IVLC was located in the superior half of the lower vertebra for the shorter two-vertebrae segments and in either the superior or inferior half of the center vertebra in the longer three-vertebrae segments. The segment length was increased after six tests because the loading head acted as a constraint on the motion of the upper segment, restricting the free transfer of load on to the facets. By using three vertebrae and two discs, the facet joints between the discs were free to assume their share of the load.

While using the IVLC, the posterior eccentricity was limited to 12.5 mm to prevent excessive extension, since the anterior ligament was cut. At the peak load of 1,112 N and an eccentricity of 12.5 mm posteriorly, there was no indication of a gap along the anterior border of the IVLC/bone interface. Separation occurred at larger eccentricities.

Fig 4. An intact lumbar segment being loaded in an Instron testing machine.

Axial Loading of Isolated Facet Joints. Following tests on the lumbar segments, the facet joints were dissected free of the vertebral bodies at the pedicle/centrum junction. They were loaded then in axial compression and tension to determine their stiffness and mode of failure. The ends of the specimen again were

Fig 5. A lumbar segment instrumented with an intervertebral load cell (IVLC).

Fig 6. A pair of isolated facet joints with both ends potted in preparation for testing in an Instron testing machine.

potted in Ostalloy, reinforced by a pin through the specimen and the sides of the cup to increase its ability to withstand tensile loads. The natural orientation of the facet joint was maintained during the potting procedure. Figure 6 shows the anterior aspect of the isolated facet joint, including the lamina. Optical measurements of facet joint rotation during earlier tests on an intact lumbar segment showed that it was minimal and that compression of the facets was representative of spinal extension and tensile loading of the facets was equivalent to spinal flexion. The procedure was to perform a series of nondestructive tests on the facet joints in compression and tension to gather stiffness and hysteresis data. The final test was to check failure in either compression or tension. Some specimens initially were loaded in compression and tested in both modes during a single loading cycle. This type of testing was necessary to identify the neutral position of the joint and to make a direct comparison of the relative stiffness of the joint in tension and compression.

Formulation of a Finite Element Model. A three-dimensional finite element model of a lumbar motion segment was developed for two purposes. First, it was employed to demonstrate the negligible effect of ligament tension on the computation of facet load. More importantly, it was used to study the effects of disc degeneration on changes in facet load. The model was based on the finite element model of a single vertebra developed by Hakim and King,[8] but the number of nodes and elements for each vertebra was reduced to conserve computation time. The motion segment was simulated by two vertebrae and a disc. Isoparametric elements were used to represent the centrum, pedicles, facets, laminae, and disc. The cortical bone of the body and the processes were represented by thin shell or plate elements. The nucleus pulposus was simulated by liquid elements. There were a total of 228 elements and 261 nodes. Bilateral symmetry was assumed so that only one-half of the motion segment needed to be simulated. The mid-sagittal plane was the plane of symmetry. Figure 7 and 8 show the top and lateral view of the model. The FEAP code developed by Taylor and Sackman[22] was used to determine the response of the motion segment to loads applied to the upper end-plate and facet joint. The bottom of the segment was supported on boundary spring elements to simulate the vertebrae and discs below it. The model was exercised to compute the effect of posterior ligaments on facet load. Linearly elastic elements were used to represent the supraspinous and interspinous ligament. The stiffness was assumed to vary from 10 N/mm to 200 N/mm/. It also was used to study the effect of disc degeneration on facet load. Degeneration was simulated by a 67% decrease in the elastic modulus of the annulus fibrosus resulting in a decrease in disc height.

RESULTS

Spinal segments from twelve cadavers were used in this study. Relevant data regarding the cadavers used, and the state of degeneration of the lumbar discs are given in Table 1. The condition of the discs was graded by Galante's[5] method. The criteria for the choice of specimens was based on age and cause of death. The maximum age was 70, and the terminal disease should not have resulted in a long period of bed rest prior to death.

The results of this study can be subdivided into four categories. Stiffness data of lumbar segments are presented first to ascertain that the change in stiffness due to the introduction of an IVLC was minimal. This is followed then by the results of the facet load experiments and stiffness data on isolated facet joints. Finally, results of the finite element model are given.

Lumbar Segment Stiffness

The insertion of an IVLC into a vertebral body is likely to affect the response of the spinal segment to applied load. The severing of the anterior longitudinal ligament precludes the application of loads, which cause excessive spinal extension. However, even if the loads did not result in large extension moments, it was necessary to

Table 1. Relevant Information on Cadaveric Specimens and Disc Grade*

Specimen no.	Cadaver no.	Sex	Age	Cause of death	Disc grade T12-L1	L1-L2	L2-L3	L3-L4	L4-L5
1	5144	F	51	Ventricular fibrillation	†				
2	75	M	66	Intractable myocardial failure	†				
3	99	M	47	Grade III astrocytoma	†				
4	140	M	54	Right heart failure	†				
5	170	M	21	CO poisoning	†				
6	164	M	60	Cardiac arrest	NA‡	3	3	3	3
7	172	F	46	Myocardial infarction	2	2	3	3	2
8	194	M	65	CO poisoning	NA	NA	NA	3	3
9	259	M	29	Cardiopulmonary arrest	2	2	2	NA	1
10	274	F	57	Cardiac asystole arteriosclerosis	2	NA	2	2	2
11	277	M	66	Cardiovascular disease	2	3	2	3	3
12	286	F	56	Cardiovascular disease	2	3	2	3	4†

*Per Galante (1967).
†Facet joint test only.
‡NA = not available.

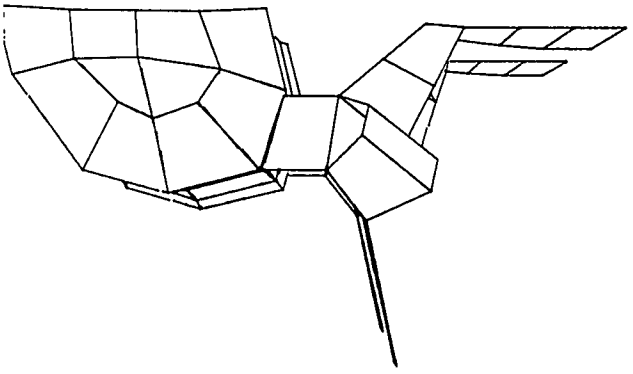

Fig 7. Top view of a bilaterally symmetric finite element model of a lumbar motion segment.

verify that the stiffness of the spinal segment did not change appreciably after the insertion of an IVLC. Force-deformation curves of the form shown in Figure 9 were obtained with and without the IVLC for all segments used in this study. There was a shift in the curves, usually to the right after the insertion of the

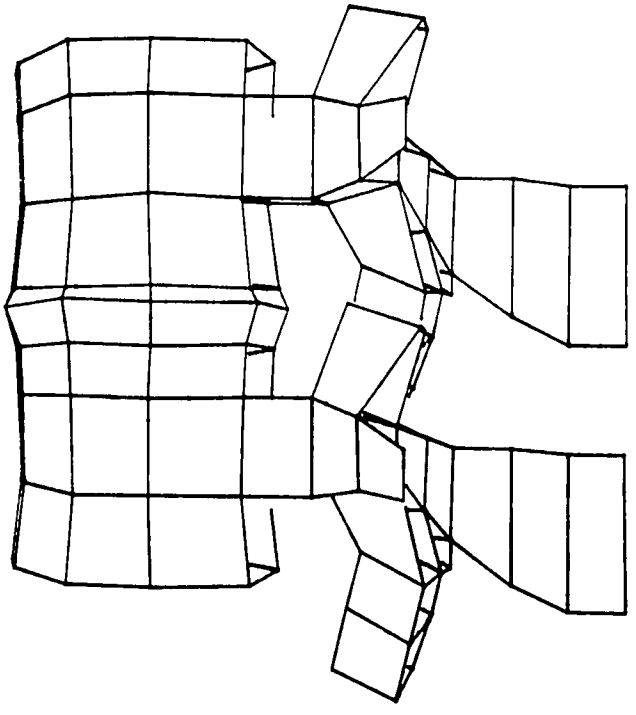

Fig 8. Side view of the finite element model.

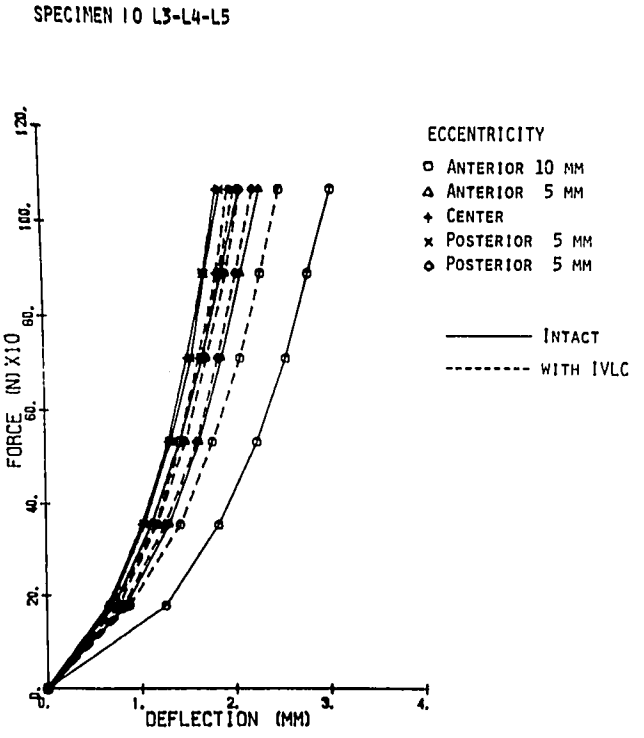

Fig 9. Comparison of segment stiffness with and without the IVLC.

IVLC, but the final slope of the force-deformation curves remained unchanged for four of the six segments tested. Figure 10 is an example of a large shift due in part to the slot being either wider or narrower than the height of the load cell. The experiment was performed for the entire range of eccentricities and the variation in stiffness at 1000 N is shown in Table 2 for the six three-vertebrae segments. In general, the stiffness increased as the load moved posteriorly. However, there is an apparent drop in stiffness at a posterior eccentricity of 10 mm. This occurs with and without the IVLC. A similar trend is seen in the data reported by Lin et al.[10] Specimens that showed a large shift in the load-deflection curve also had a significant change in stiffness.

Facet Load As a Function of Eccentricity of Loading

As mentioned above, facet load is the difference between the applied load and that measured by the IVLC (disc load). The data were comparable to those reported by Hakim and King,[7] but greater care was taken to measure facet load for different eccentricities. The position of the applied load used the geometric center of the vertebral body as a reference. Thus, the applied load could have an anterior eccentricity or a posterior eccentricity. The

Table 2. Stiffness Data of Lumbar Segments With and Without an IVLC (N/mm)

		Load location (Eccentricity)									
		10 mm Anterior		*5 mm Anterior*		*Center*		*5 mm Posterior*		*10 mm Posterior*	
Specimen no.	*Joint*	*Intact*	*IVLC*	*Intact*	*IVLC*	*Intact*	*IVLC*	*Intact*	*IVLC*	*Intact*	*IVLC*
9	L3-L4-L5	560	525	—	—	655	630	—	—	833	735
10	L3-L4-L5	761	854	875	972	1094	1094	1094	1000	875	946
11	T12-L1-L2	1129	875	1167	875	1250	1167	1296	1167	1029	972
12	T12-L1-L2	795	427	972	583	1029	673	1029	833	1094	921
11	L3-L4-L5	1296	625	1575	875	1750	921	1750	673	1750	1167
12	L3-L4-L5	1167	1207	1458	1346	1489	1458	1522	1556	1400	1458

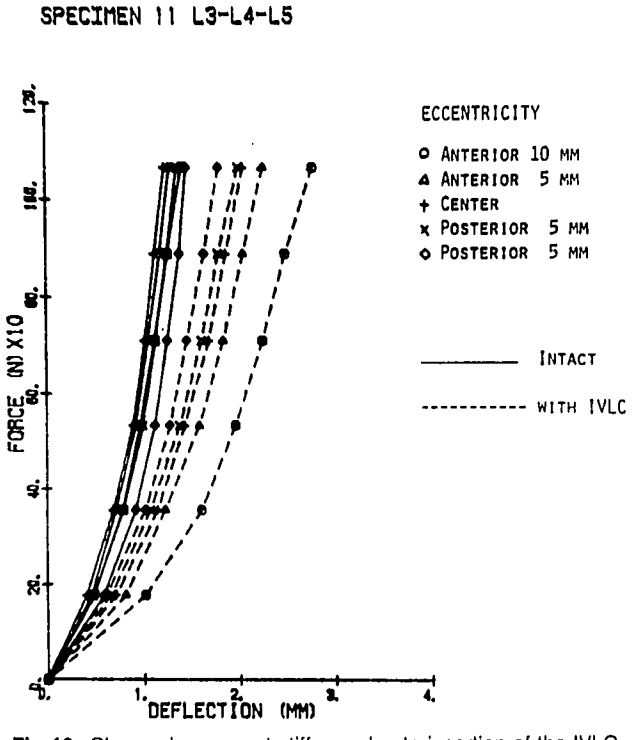

Fig 10. Change in segment stiffness due to insertion of the IVLC.

choice of this reference point is consistent with the work of Panjabi[16] and others. However, it does not imply that the vertebral body pivots about this point, nor does it imply that this point has any biomechanical significance. Table 3 and Figure 11 show the mean facet load as a percentage of total load for different eccentricities, for the six two-vertebrae segments. The increase in facet load as the load became more posterior was relatively small. The average facet load for four of the six three-vertebrae segments is shown in Figure 12 and the upper portion of Table 4. The increase in facet load is more uniform and its magnitude is smaller than that shown in Figure 11. The fact that the vertebrae and discs were allowed to adjust to the applied load is well-demonstrated here. One of the specimens had severe osteoarthritis of the right facet joint at the L4-L5 level, and the facet load reached 47% of the total load, indicating that the joint was abnormal. The lower position of Table 4 shows facet load for two segments with severe degeneration.

Table 3. Two-Segment Facet Load (% Total Load)

		Eccentricity		
Specimen no.	*Joint*	*Anterior 10 mm*	*Center*	*Posterior 10 mm*
3	L3-L4	19.91%	16.08	23.75
6	L1-L2	NA*	26.98	21.24
6	L4-L5	20.14	23.38	32.37
7	L1-L2	24.95	29.67	34.73
7	L4-L5	21.67	29.45	40.47
8	L4-L5	24.67	25.19	29.84
Mean		22.27	25.13	30.40
SD ±		2.42	5.05	7.11

*NA =not available—instrumentation problem.

Stiffness of Isolated Facet Joints

Having reconfirmed the load-bearing role of the articular facets, it was now necessary to identify their response to load and mode of failure. They first were loaded cyclically in compression and tension before the failure test. The specimens were loaded under stroke control in the Instron testing machine. The time traces for applied compressive displacement and load are shown in Figure 13. The tensile load is barely noticeable because it is plotted on the same scale as the compressive load. By cross-plotting these data, a load-deformation curve was obtained, as shown in Figure 14. In compression, the facets behaved as a stiffening spring, and the

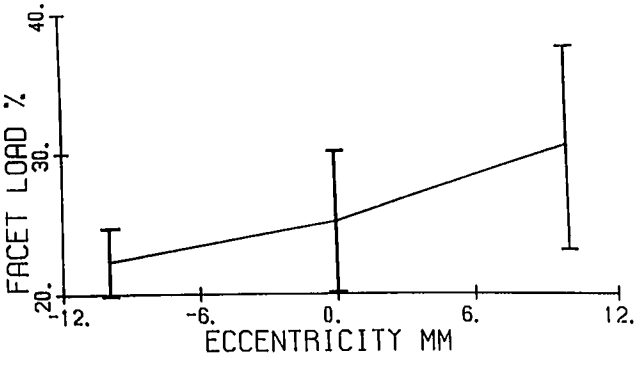

Fig 11. Facet load measured from two-vertebrae segments.

Table 4. Three-Segment Facet Load (% Total Load)

		Eccentricity (mm)								
		Anterior			Center		Posterior			
Specimen no.	*Joint*	*10.0*	*7.5*	*5.0*	*2.5*	*2.5*	*5.0*	*7.5*	*10.0*	*12.5*
9	T12-L1-L2	6.66%	*	*	*	12.86	*	*	*	19.37	*
10	L3-L4-L5	2.90	4.35	5.70	6.70	8.20	8.80	9.80	12.35	13.55	14.40
11	T12-L1-L2	0.48	3.15	5.98	7.44	7.85	8.97	9.86	11.71	14.20	18.36
12	T12-L1-L2	3.19	6.81	9.64	11.17	15.20	16.70	18.84	19.72	21.68	24.03
Mean		3.31	4.77	7.11	8.44	11.03	11.49	12.77	14.59	17.20	18.93
SD ±		2.54	1.87	2.20	2.40	3.60	4.51	5.09	4.45	3.96	4.84
11	L3-L4-L5	17.90	20.10	25.00	27.90	31.10	34.70	37.90	40.80	44.00	46.90
12	L3-L4-L5	13.22	16.69	20.03	24.33	28.68	33.29	38.25	43.67	47.34	52.25
Mean		15.56	18.40	22.52	26.12	29.89	34.00	38.08	42.24	45.67	49.48

*Not measured.

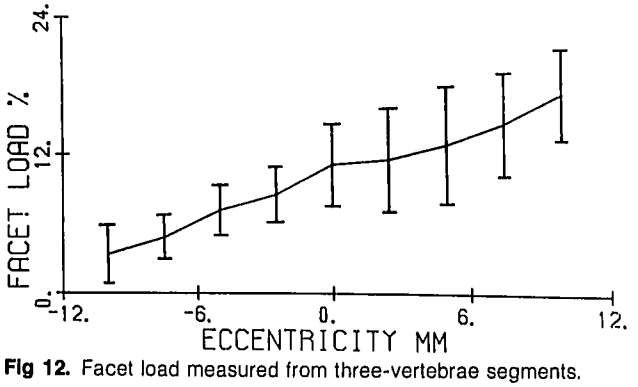

Fig 12. Facet load measured from three-vertebrae segments.

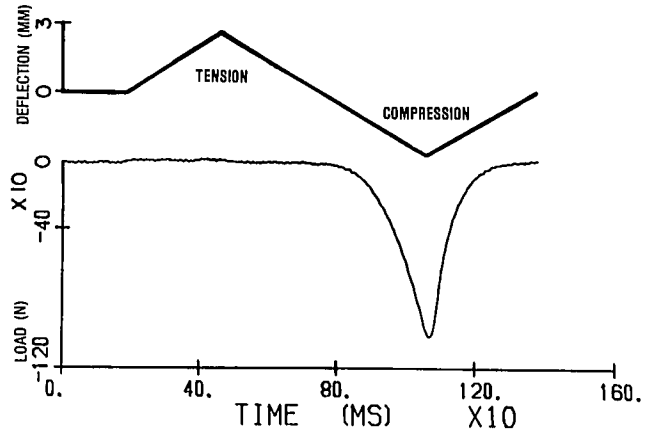

Fig 13. Time history of stroke and load for tests on isolated facet joint.

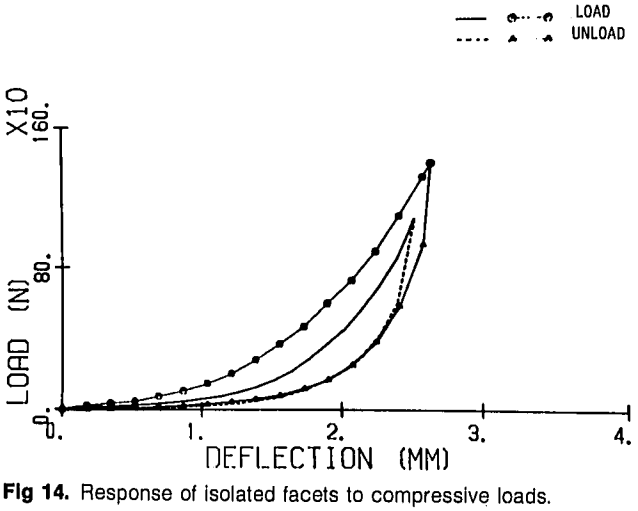

Fig 14. Response of isolated facets to compressive loads.

response was unchanged by the cutting of all posterior ligaments, ie, the ligamentum flavum, the interspinous ligament, and the supraspinous ligament. Because the tests were carried out under stroke control and the neutral position was difficult to attain in the potting procedure, the peak magnitude of load for a given deflection was highly variable (400–1,300 N). However, the stiffness near the end of the stroke is more meaningful since it does not depend on the neutral zone or shift along the abscissa. Two

Table 5. Compressive Stiffness of Facet Joints (at 2.2 mm deflection)

Specimen no.	*Joint*	*Stiffness*
4	L1-L2	736 N/mm
6	L1-L2	1079
5	L1-L2	1751
7	L1-L2	1331
1	L4-L5	1160
2	L4-L5	1043
3	L3-L4	779
4	L4-L5	515
6	L4-L5	2017
5	L4-L5	736
7	L4-L5	2522
8	L4-L5	1821
Mean		1291
SD ±		614

typical hysteresis loops are shown in Figure 14. Stiffness data at a deflection of 2.2 mm are listed in Table 5. In another series of tests, the specimens were cycled through their neutral position and a composite load-deformation curve in tension and compression was obtained (Figure 15). Due to the large disparity in stiffness, only a small portion of the compressive response is shown. The tensile resistance of the capsular ligaments is very low because these tests were carried out with all posterior ligaments severed and with the capsular ligaments intact. When the facet joints were loaded to failure in compression, the capsule was torn by the rearward rotation of the inferior facet of the upper vertebra. There was no bony fracture. The load to failure was approximately 6000 N and the load-deflection curve to failure is shown in Figure 16. Tensile failure was due to capsular rupture but the failure load was only 750 N.

Response of the Finite Element Model

The finite element model was exercised briefly to ascertain the validity of its predictions. The computed intradiscal pressure was compared with experimental data obtained by Ranu et al[19] and shown in Figure 17. The material constants used for this validation attempt are given in Table 6. The model then was used to stimulate the experimental loading condition for the measurement of facet load. The aim was to compute the magnitude of the forces generated in the supraspinous and interspinous ligaments due to a

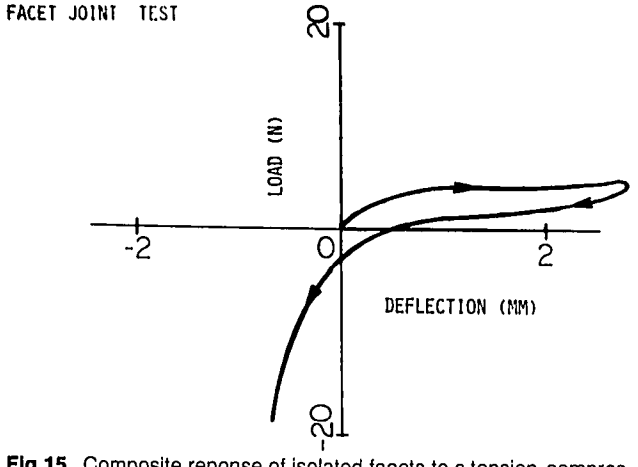

Fig 15. Composite reponse of isolated facets to a tension-compression cycle.

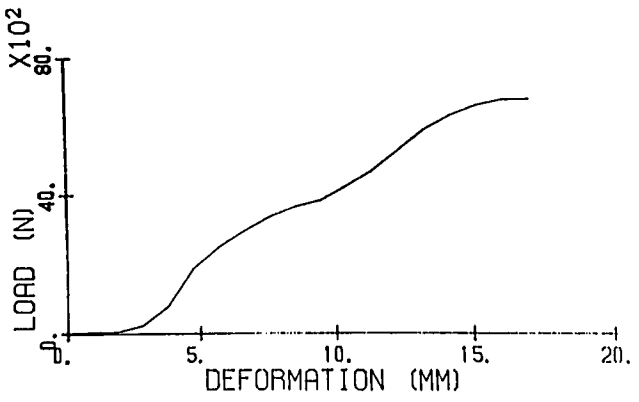

Fig 16. Load-deflection curve showing compressive failure of facet joints.

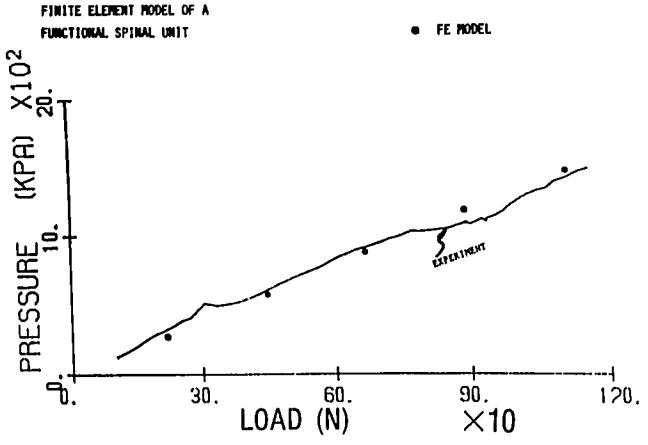

Fig 17. Validation of the finite element model, a comparison of measured and computed intradiscal pressure.

large flexion moment. By assuming a stiffness of 10 N/mm for both ligaments, the maximum total load in both ligaments was 9 N for an applied load of 1,112 N with a 8-mm anterior eccentricity. If the stiffness was increased to 200 N/mm, the corresponding total ligament force was 48 N. Thus, the effect of posterior ligament tension was negligible in the computation of facet load, based on measured disc load (IVLC data).

The simulation of disc degeneration is of significance since low-back pain is often associated with disc disease. The model was used to compare the facet load for normal and degenerated discs. The diseased condition of the disc was simulated by decreasing the elastic modulus of the annulus fibrosus from 92 MPa to 31 MPa. This resulted in a significant increase in facet load, as shown in Table 7. The model also predicts the facet load quite well for posterior loading. Effort is underway to improve its response for anterior loading.

DISCUSSION

Experimental data from this study show that facet load is a function of eccentricity of the applied load. Data from the loading of isolated facet joints further reveal the nonlinear behavior of the facet joint in spinal extension and flexion. In extension or compression of the facets, the joint stiffens rapidly with deformation, reaching a stiffness of 1.3 kN/mm at a deformation of 2.2 mm. This is an indication of bony interaction through a thin intervening layer of soft tissue. Thus, the mechanism of load transmission is due to the bottoming-out of the tip of the inferior facet on the pars interarticularis of the vertebra below. Direct verification of this mechanism is beyond the state of the art of existing transducer technology, but there is sufficient evidence to state this mechanism.

The mode of failure of the facet joint provides additional insight into this mechanism. The capsules were ruptured by the rearward rotation of the inferior facet. This observation tends to confirm the bottoming-out theory since rotation of the facet under compression requires a pivot point. Data provided by Panjabi et al[16] show that when a segment is loaded in extension, there is a rearward translation and rotation of the body with negligible translation along the other two axes. The observed decrease in segment stiffness for posterior loads with eccentricities equal to or greater than 10 mm also supports the rearward rotation of the facets. This motion causes a relatively large deformation with only a small increase in load.

The hypothesis that there is a causal relationship between facet load and low-back pain also is based on the observed rotation of the facet due to a high compressive load. Pedersen et al[17] found the capsule of the articular facets to be innervated richly by receptor endings. This was confirmed by Wyke,[23] who was able to cause these endings to fire by stretching the capsular ligaments. However, he was unable to relate stretch to applied loads. The results of this study give a mechanical basis for the stretching of the capsule and, thus, a cause for low-back pain. Additional research is necessary to quantify the relative motion of the facets under load and to demonstrate their neurologic effect on the receptors in the capsule.

It should be kept in mind that the proposed hypothesis for low-back pain constitutes one of many causes of low-back pain.

Table 6. Material Properties Used in Finite Element Model

Description	*Modulus (MPa)*	*Poisson ratio*	*Thickness (mm)*
Centrum—trabecular bone	345	0.2	—
Disc—nucleus (bulk modulus)	2,255	0.5	—
Disc—annulus	92	0.45	—
Facets, laminae, pedicles	11,032	0.25	—
End-plate—peripheral	12,480	0.28	0.81
End-plate—central	12,480	0.28	0.51
Vertebral body—cortex	11,032	0.25	0.64
Disc—cortex (anterior and posterior ligaments)	138	0.45	1.27
Processess (spinous and transverse)	12,480	0.28	1.27
Ligament (supraspinous and interspinous)	10 N/mm.		

Table 7. Model Predictions of Facet Load for Normal and Degenerated Discs

	Eccentricity		
	Center	*5 mm Posterior*	*10 mm Posterior*
Normal	0.9%	10.1%	17.5%
Degenerated	0.9%	13.2%	25.8%

However, the model was able to show that there was an increase in facet load due to disc degeneration. Increased facet loads also can be a cause for the initiation of osteoarthritis in the facet joint, which tends to deform the joint capsule. Present results do not permit further speculation regarding the relationship of facet load to low-back pain. The use of chymopapain would appear to aggravate the problem, but its intended purpose is to eliminate the extruded nucleus in patients who do not have a facet syndrome.

CONCLUSIONS

From this study, the authors have made the following conclusions: (1) An indirect method of measuring facet load was used to show that facet load increased with increasing extension moment. (2) The response of the isolated facet joint to compressive loading can be explained by the bony interaction of the tip of the inferior facet with the pars interarticularis of the vertebra below. (3) The bottoming-out of the facet on the pars is the mechanism of facet load transmission. (4) Excessive facet loads cause the inferior facet to pivot about the pars and to stretch the joint capsule. (5) A mechanically based hypothesis is proposed to link facet loading with low-back pain. This hypothesis is consistent with clinical and anatomic evidence, which indicate that the facets are possible sites for low-back pain. (6) A three-dimensional, finite element model of a motion segment was formulated to simulate the transmission of facet load and the computation of facet load from measured disc load. (7) The model predicted an increase in facet load due to disc degeneration. (8) Much work remains to be done before the hypothesis can be established as a scientific fact. Research to prove or disprove this hypothesis needs to be pursued to shed more light on the complex problem of low-back pain.

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