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1981 Volvo Award in Basic Science

Prolapsed Intervertebral Disc

A Hyperflexion Injury

M. A. ADAMS and W. C. HUTTON

Sixty-one lumbar intervertebral joints were compressed while wedged to simulate hyperflexion. Twenty-six of the joints failed by posterior disc prolapse. The results show that slightly degenerated discs at lower lumbar levels from subjects aged between 40 and 50 years are most susceptible to prolapse. [Key words: prolapsed intervertebral disc, hyperflexion, compression, disc degeneration]

VERY LITTLE is known about the mechanics of intervertebral disc prolapse. That extrusion of the nucleus pulposus can lead to compression of nerve roots is well established, but the precise mechanism producing the extrusion is as yet unknown. This is partly because it has never proved possible to produce a prolapse in a cadaveric specimen under controlled loading conditions. Pure compression of an intervertebral joint invariably leads to vertebral failure even with fatigue loading.^{4,7,10} Torsion has been shown to damage the articular facets, and if carried well beyond the physiologic limit to produce circumferential tears in the annulus fibrosus.^{1,5} Flexion just beyond the physiologic limit results in damage to the ligaments of the neural arch but not to the disc.¹ All these previous experiments seem to indicate that a prolapsed intervertebral disc (p.i.d.) is not a traumatic injury but the end result of a gradual or fatigue process.

However, there is considerable clinical evidence linking disc prolapse with sudden high loading of the spine, especially in flexed postures. It seems reason-

able, therefore, to assume that it may sometimes be a traumatic injury but one that has yet to be simulated in the laboratory.

Previous experiments by the authors have shown that, when an intervertebral joint is hyperflexed, the first structures to be damaged are the supra/interspinous ligaments.² These ligaments have been reported to be "almost invariably" damaged in cases of prolapsed intervertebral disc brought to surgery.¹¹ It was decided, therefore, to test the hypothesis that p.i.d. can be a compression injury to a joint hyperflexed a few degrees beyond its normal limit.

APPARATUS

The testing apparatus is shown in Figure 1. The combination of angle plate and rollers enabled the joints to be tested in compression with any predetermined angle of flexion ϕ . This configuration also produced a small amount of shear and did not restrict any "settling movements" of the specimen in the sagittal plane. A hydraulic servo-controlled testing machine applied a force increasing at 3000 N/second, while an X-Y recorder plotted applied force against vertical displacement.

Geometrical considerations show that the flexion angle, ϕ , is given by

$$\phi = \theta_2 + \theta_1$$

where θ_1 and θ_2 are defined as in Figure 1. Because

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Θ_1 and Θ_2 could be varied independently, an appropriate small shear force was selected for any given flexion angle.

CADAVERIC MATERIAL AND SETTING PROCEDURE

Thirty-three lumbar spines were removed at routine necropsies from subjects, aged between 14 and 78 years, who had been mobile prior to death. The spines were stored at -20°C in sealed plastic bags for up to three months until required; they were then thawed in their bags in a refrigerator for 12 hours.

Each spine was dissected into joints consisting of two vertebrae and the intervening soft tissues. In order to observe the posterior annulus clearly during testing, the laminae were sawn off using a hacksaw, and the contents of the neural canal was removed with forceps. The facet surfaces were left intact to resist any intervertebral shear forces as would occur in life, so this "laminectomy" did not place any increased stresses on the intervertebral disc. The upper and lower surfaces were scraped clean, and all the remaining posterior ligaments (except the posterior longitudinal ligament and inaccessible parts of the facet capsule) were cut so that they would not exert a hidden compressive force on the disc. The intervertebral disc and the sides of the vertebral bodies were left untouched. The specimen was then set in two cups containing mildly exothermic dental plaster, "Q.S. Stonehard," with the midplane of the intervertebral disc parallel to the ends of the cups. Much of the remaining neural arch was embedded, and fixation with the plaster was ensured by attaching metal hooks round the pedicles.

The vertebral bodies merely rested on the surface of the plaster so that no artificial strengthening of them might occur. The specimens were set with their anteroposterior axis at an angle (15° on average) to the sides of the cups, so that the apparatus produced a combination of lateral flexion and forward flexion. This arrangement ensured that maximum stretching of the annulus fibrosus occurred at a posterolateral margin.

METHOD

We wanted to flex our specimens just beyond the normal limit of flexion as indicated by damage to the supra/interspinous ligaments. However, the "laminectomy" meant that this limit had to be estimated from X-ray data. Previous X-ray studies were unsuitable because, with the exception of the work on college women by Clayson et al,¹² they included few results from young healthy people. Therefore, our own survey was necessary.

Lumbar radiographs were taken of 27 healthy subjects, aged between 20 and 58 years, in the erect standing and fully flexed postures. Measurements on these X-ray films then produced the flexion angle, the anterior disc compression, and the posterior disc elongation for each intervertebral joint in the fully flexed

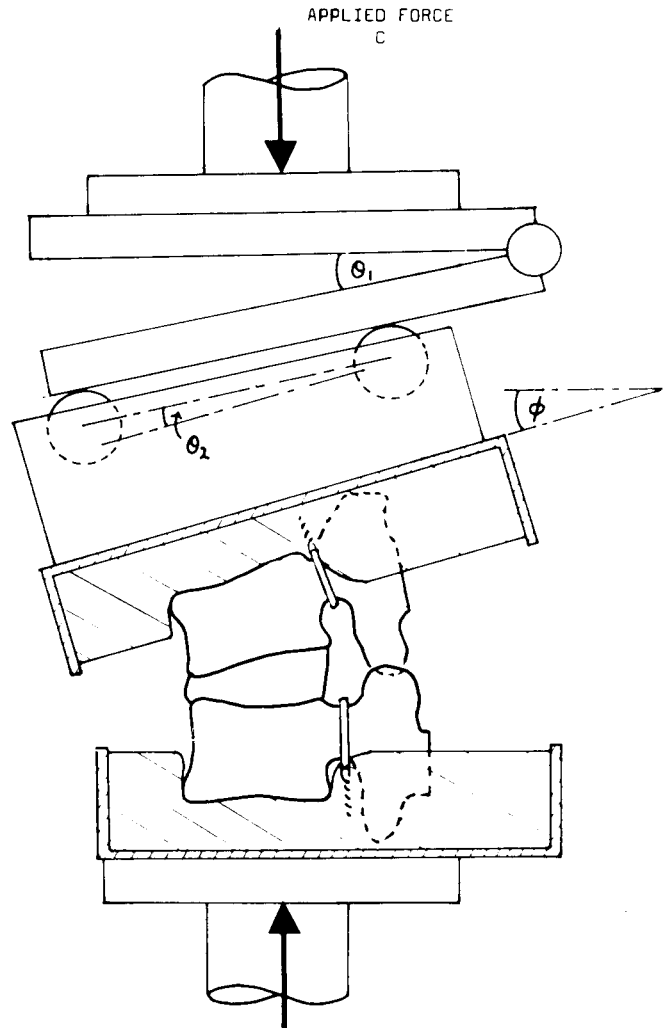


Fig 1. The apparatus used to hyperflex and compress the lumbar intervertebral joints.

posture. The average values are presented in Table 1; they are in good agreement with the results of Clayson.

It has been shown that in the erect standing posture each lumbar intervertebral joint is extended by about 2° , compared with an excised cadaver spine.² Therefore, in our experiments on cadaveric joints, the "limit of flexion" was taken to be 2° less than the average angle shown in Table 1. For example, the "limit of normal flexion" for an L3-L4 intervertebral joint was assumed to be about 10° .

The following test procedure was then adopted:

1. Each joint was flexed to the "limit of normal flexion" (unless it was extremely immobile, in which case testing started and finished at lower flexion angles).
2. While wedged in this way, the joint was compressed to a maximum that represented the likely compressive force generated by heavy lifting. This was estimated on the basis of about 8000 N calculated previously for a young man of average

Table 1. The Average Results from 27 Sets of X-rays of Healthy Subjects (Aged Between 20 and 58) in the Erect Standing and Fully Flexed Postures

	Spinal Level				
	L5-S1	L4-L5	L3-L4	L2-L3	L1-L2
Flexion angle (degrees)					
Mean	10.1	14.5	11.7	10.9	8.3
Standard deviation	±4.9	±2.9	±2.2	±3.1	±2.6
Range	(0, 17)	(10, 20)	(6, 16)	(5, 18)	(3, 12)
Anterior disc compression (%)					
Mean	25	36	35	32	28
Standard deviation	±15	±11	±9	±9	±11
Posterior disc elongation (%)					
Mean	44	54	52	58	33
Standard deviation	±32	±24	±20	±24	±22

weight.⁸ A force-deformation curve was then plotted.

- If failure of the joint was not indicated, the flexion angle was increased by 1° or 2°, and the same load was applied again. This process was repeated until failure did occur.
- If flexion produced any tearing noises from the capsular ligaments, then the joint was tested to failure at that angle. No joint was flexed beyond 18°.

After testing, the joint was photographed and the disc was bisected. Each intervertebral disc was examined macroscopically for the degree of degeneration and was scored on a scale from 1 to 4 according to the categories proposed by Galante.⁶ The cross-sectional area of the disc was calculated from the width *a* and breadth *b* by using the equation for the area of an ellipse ($\pi/4 ab$). This is included in Table 2 in order to give an impression of specimen size.

RESULTS

Twenty-six of the 61 joints tested failed by a prolapse of the intervertebral disc. A typical load-deformation curve for an intervertebral joint which failed in this way is shown as curve A in Figure 2. The disc prolapse (point P) causes a sudden reduction in disc volume, but because there is no bone damage and the annulus fibrosus is largely intact, the stiffness of the joint is little impaired and the gradient of the graph immediately after prolapse is similar to that before. This is in marked contrast to curve B which depicts a typical compression failure by endplate fracture. Here there is a slight reduction in volume as the nucleus is expressed into the vertebral body, but because the cortical bone shell of the vertebral body has been seriously disrupted, the joint just crumbles, with very little further resistance. Note that curve A is smooth and approximately straight right up until the point of failure: this indicates that the disc has not been damaged by the wedging angle and is resisting normally until the compressive force reaches some critical level.

Graph A also shows that prolapse produces a reduction in disc height (*h*). This reduction was measured to be 0.7 mm on average, with a maximum of 1.3 mm.

Table 2 shows specimen details, the flexion angle, and applied compressive force at failure (*C*) for each joint tested. It was found that the "mode of failure" could be classified in four distinct groups.

1. Nuclear Extrusion

Nuclear pulp appeared either on a posterolateral edge of the vertebral body or in the neural canal. The end-plates were intact, there was no evidence of vertebral body crushing, and no blood expressed from the anterior vertebral body or in the neural canal. Figures 3, 4, and 5 show typical cases of nuclear extrusion produced by the tests.

2. Annular Protrusion

The posterior annulus was very soft and bulging slightly into the neural canal, sometimes in a distinct tongue. The annular rings showed disruption at the point of bulging. The nucleus showed signs of degeneration and seemed to be displaced slightly posteriorly, behind the bulging annulus; there was sometimes a channel between the nucleus and the neural canal, but no nuclear pulp was expressed through it. All end-plates were intact, but there was some blood expressed from the vertebral bodies anteriorly or in the neural canal. Figures 6 and 7 show a typical case of annular protrusion.

3. Compressive Fracture

There was anterior crushing of a vertebral body occasionally with a fractured end-plate, and blood was expressed from the vertebral bodies, usually anteriorly. If the end-plate fracture were large, there was sometimes nuclear material in the vertebral bodies. End-plate fracture was found almost invariably in our previous compressive strength tests, when the flexion angles were smaller—around 3° to 8°.⁸

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‡ Small

Table 2. The Results of 61 Specimens Tested

No.	Specimen details				Results			
	sex-age	Body mass (kg)	Spinal level	Disc area (cm ²)	Disc degeneration	Flexion angle (degrees)	Applied force C (N)	Mode of failure
1.	M-14	46	L2-L3	15.5	1	14	2,760	1*
2.	M-14	46	L4-L5	16.8	1	18	7,695	3
3.	M-19	76	L4-L5	16.6	1	17	4,830	1
4.	F-21	51	L1-L2	9.4	1	14	5,616	3
5.	F-21	51	L3-L4	11.8	1	16	5,207	1*
6.	F-21	51	L5-S1	12.2	1	18	7,324	1
7.	M-22	71	L1-L2	14.1	1	14	8,790	3
8.	F-22	57	L3-L4	16.2	1	14	7,790	3
9.	F-22	57	L5-S1	17.0	1	18	9,580	3
10.	M-23	73	L1-L2	15.4	2	12	9,175	3
11.	M-23	73	L3-L4	16.4	3	12	9,636	3
12.	M-23	73	L5-S1	16.8	3	18	11,124	3
13.	M-25	60	L1-L2	13.2	1	10	4,019	3
14.	M-25	60	L3-L4	14.9	1	16	4,199	3
15.	M-25	60	L5-S1	15.0	1	18	2,850	1
16.	M-29	52	L5-S1	14.6	1	16	5,530	3
17.	F-31	61	L3-L4	14.1	1	12	5,118	3
18.	F-31	61	L5-S1	13.4	1	12	4,430	3
19.	M-32	73	L4-L5	21.2	2	8	8,088	2
20.	F-33	45	L3-L4	16.7	2	16	4,295	3
21.	F-33	45	L5-S1	18.9	2	18	5,510	1†
22.	M-37	57	L2-L3	19.7	2	12	3,814	1
23.	M-37	57	L4-L5	20.4	3	16	8,002	2
24.	F-38	51	L2-L3	13.3	3	12	—	4
25.	F-38	51	L4-L5	14.7	3	16	—	4
26.	F-38	52	L3-L4	12.8	2	12	—	4
27.	F-38	52	L5-S1	17.0	3	16	3,800	3
28.	M-39	88	L1-L2	15.8	2	10	4,340	1
29.	M-39	88	L3-L4	16.2	2	10	6,330	1
30.	M-39	88	L5-S1	17.5	2	12	4,570	2
31.	F-40	47	L5-S1	12.6	2	16	5,601	1
32.	F-41	52	L5-S1	16.2	3	16	4,930	2
33.	M-42	79	L2-L3	19.1	2	10	3,820	3
34.	M-42	79	L4-L5	19.1	2	18	6,030	3
35.	M-46	80	L3-L4	18.8	2	6	9,306	1
36.	M-46	80	L5-S1	19.8	3	14	12,968	1
37.	F-48	51	L1-L2	10.8	2	8	3,420	1
38.	F-48	51	L3-L4	12.2	2	12	3,893	1
39.	F-48	51	L5-S1	12.1	2	8	3,921	1
40.	F-50	90	L4-L5	18.5	3	7	4,160	2
41.	M-50	80	L2-L3	17.3	3	14	6,693	3
42.	M-50	80	L4-L5	18.8	2	16	5,824	1
43.	F-51	70	L1-L2	13.7	3	10	3,990	1†
44.	F-51	71	L5-S1	13.1	3	14	8,130	1
45.	M-54	58	L5-S1	13.9	3	8	3,408	3
46.	M-55	56	L2-L3	24.4	3	9	3,293	2
47.	M-55	56	L5-S1	22.8	3	11	3,610	3
48.	M-58	72	L3-L4	18.1	3	13	2,771	3
49.	M-58	72	L5-S1	17.3	3	13	2,880	2
50.	M-58	72	L2-L3	21.8	3	13	5,247	3
51.	M-58	72	L4-L5	21.3	3	16	5,730	2
52.	M-59	64	L3-L4	16.6	3	12	2,920	3
53.	M-59	64	L5-S1	15.8	3	14	4,561	3
54.	F-69	62	L2-L3	18.1	4	6	2,170	3
55.	F-69	62	L4-L5	18.9	4	8	3,397	3
56.	M-69	74	L3-L4	17.2	3	12	4,800	3
57.	M-69	74	L5-S1	24.0	4	8	4,420	3
58.	M-70	58	L2-L3	19.8	4	10	3,700	3
59.	M-70	58	L4-L5	21.1	3	12	4,255	3
60.	F-78	49	L1-L2	12.6	3	10	2,060	3
61.	F-78	49	L4-L5	16.6	4	10	3,643	3

* The extruded nucleus flowed as a high-viscosity liquid.

† Extrusion from out of a preexisting protrusion.

‡ Small lateral extrusion.

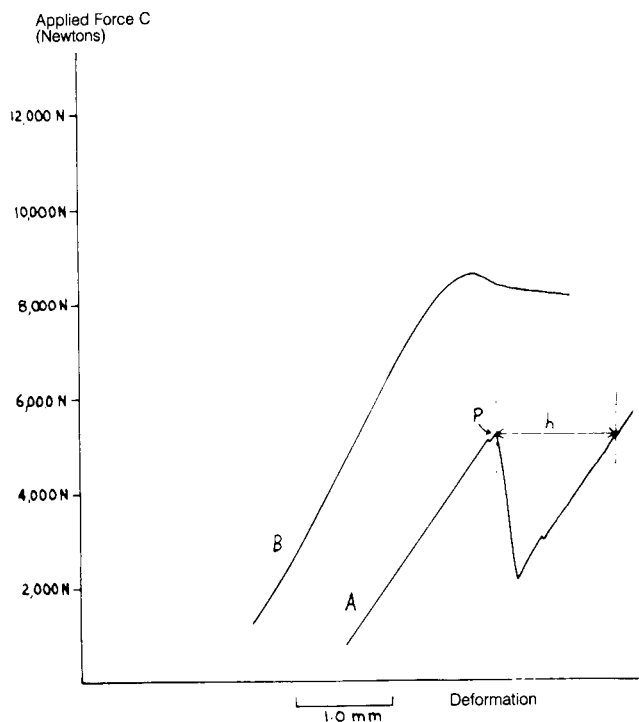


Fig 2. Two typical load-deformation curves. A is for a joint that failed by nuclear extrusion (at point P) and B is for a joint that sustained an end-plate fracture. *h* is the reduction in disc height caused by prolapse.

4. Hyperflexion Fracture

Cracking noises were heard while the joint was still being flexed and before a high compressive load was applied. There was a chip fracture of the posterior rim of the vertebral body and crushing of the anterior vertebral body wall. The end-plates were otherwise intact, the disc was undamaged, and there was little blood expressed. Figure 8 shows a typical fracture.

In the foregoing, groups 1 and 2 are referred to as disc prolapses. Several detailed observations on these may be significant. Nuclear extrusion was always either central or on the side away from the component of lateral bending where the annulus was stretched the most. The fissure through which the nuclear pulp was extruded usually occurred at the boundary between the annulus and the cartilage end-plate. Large central nuclear extrusions ruptured the posterior longitudinal ligament, whereas smaller extrusions either formed a bulge behind it (giving the impression of a bulging annulus) or were deflected sideways and appeared on one or both posterior lateral margins of the disc. The nuclear material was always soft and deformable, even from "grade 3" discs, and could easily be pushed away from the site of the extrusion. In two specimens from subjects under age 30, the pulp "flowed" under its own weight. Attempts to push extruded material back into the disc were always unsuccessful—rather like trying to push toothpaste back into the tube.

Table 3 shows that the average flexion angle and applied compressive force required to produce p.i.d. in the 26 specimens were 12.8° and 5448 N. Of these, 12 prolapsed at 12° or below and 14 below 5000 N (see Table 2). Comparison with the radiographic results indicates that about 1° to 6° of flexion beyond the normal limit was required to produce p.i.d.

Tables 4, 5, and 6 show how the susceptibility of a disc to prolapse depends on age, degree of disc degeneration, and spinal level. Slightly degenerated lower lumbar discs of people aged between 40 and 50 seem particularly vulnerable.

DISCUSSION

As described, of the 61 intervertebral joints tested, 26 produced a prolapse. However, testing conditions were chosen somewhat arbitrarily. It may be that, for a given specimen, a somewhat higher compressive load would have produced p.i.d. at smaller flexion angles or, conversely, that a higher flexion angle might have been more appropriate for a very mobile joint.

The experimental method ensured that the applied compressive force was usually within the capacity of the extensor muscles, according to our previous calculations.⁸ However, the flexion angles clearly exceed (as intended) the normal range as shown radiographically and must be termed hyperflexion.* Therefore, to obtain sufficient flexion to render the posterior annulus vulnerable, the posterior ligaments must first be overstretched. Rissanen and Newman have stated that the supra/interspinous ligament is usually found to be ruptured or slack in patients presenting for surgery for prolapsed disc.^{9,11} It should be appreciated that "hyperflexion," as used here, does not necessarily mean high angles of flexion. A particularly stiff intervertebral joint may have a normal flexion range of 6°, in which case 7° would be hyperflexion. Similarly, a very mobile lumbosacral joint might be wedged to 18° and still not be hyperflexed. It follows that a gymnast might easily adopt some extremely flexed posture in complete safety, while an inactive person could hyperflex at some level when only slightly stooped. Our radiographs showed this great variation between individuals and also between different levels of the same spine; for example, for one subject (male aged 28 years), we found the following flexion angles at each level: 2° at L5-S1, 18° at L4-L5, 11° at L3-L4, 18° at L2-L3, and 11° at L1-L2.

The likelihood of a joint exceeding its normal range of flexion will depend upon several factors. The speed

* We must emphasize that the prolapses produced here are not mere artifact resulting from too high flexion angles. We deliberately just exceeded the limit of normal flexion because there is clinical and pathologic evidence to suggest that hyperflexion is a prerequisite for prolapsed intervertebral disc. Had we obtained prolapses at low angles, we should then have been faced with the task of explaining why we all do not suffer from this injury.



Fig 3 (top le) intervertebra bisected. The protruded a intervertebra posterolater

