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# 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]

ERY 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. 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 reasonable, 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.<sup>2</sup> These ligaments have been reported to be "almost invariably" damaged in cases of prolapsed intervertebral disc brought to surgery.<sup>11</sup> 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  $\phi$ . 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,  $\phi$ , is given by

$$\phi = \Theta_2 + \Theta_1$$

where  $\Theta_1$  and  $\Theta_2$  are defined as in Figure 1. Because

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 $\Theta_1$  and  $\Theta_2$  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.

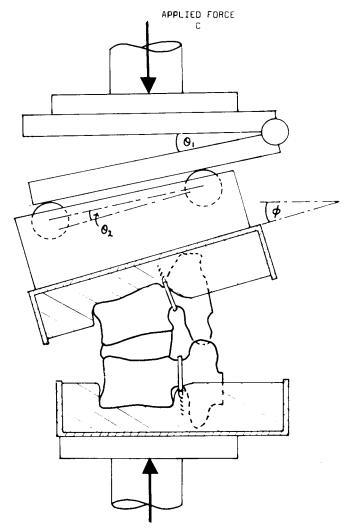
Each spine was dissected into joints consisting of two vertebrae and the intervening soft tissues. In order to observe the posterior annulus clearly during testing, the laminae were sawn off using a hacksaw, and the contents of the neural canal was removed with forceps. The facet surfaces were left intact 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,<sup>12</sup> 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.<sup>2</sup> 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:

- 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

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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 (		, ,	(1, 1,	(=, -, -,	(-1/			
Mean	<sup>25</sup>	36	35	32	28			
Standard deviation	±15	±11	±9	±9	±11			
Posterior disc elongation (%	.)							
Mean Č	<sup>′</sup> 44	54	52	58	33			
Standard deviation	±32	±24	±20	±24	±22			

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

- 3. 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.
- 4. 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.<sup>6</sup> 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 endplates 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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<sup>†</sup> Extrus \* Small

Table 2. The Results of 61 Specimens Tested

			Specimen details		1 61 Specimens		Results	
No.	sex–age	Body mass (kg)	Spinal level	Disc area (cm²)	Disc degen- eration	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 16	5,616 5,207	3 1*
5.	F-21	51 51	L3-L4 L5-S1	11.8 12.2	1	18	7,324	1
6. 7.	F21 <b>M</b> 22	51 71	L1-L2	14.1	1	14	8,790	
8.	F–22	57	L3-L4	16.2	1	14	7,790	3 3 3 3 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 11,124	3 3
12.	M-23	73	L5S1 L1L2	16.8 13.2	3 1	18 10	4,019	3
13. 14.	M-25 M-25	60 60	L3-L4	14.9	1	16	4,199	3 3
14. 15.	M-25	60	L5-S1	15.0	i	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 3
18.	F-31	61	L5-S1	13.4	1	12 8	4,430 8,088	2
19.	M-32	73	L4-L5 L3-L4	21.2 16.7	2	o 16	4,295	2 3
20. 21.	F-33 F-33	45 45	L5-L4 L5-S1	18.9	2 2 2	18	5,510	1 <sup>†</sup>
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 4
24.	F-38	51	L2-L3	13.3	3	12	_	
25.	F-38	51	L4-L5	14.7	3	16 12		4 4
26.	F-38	52 52	L3-L4 L5-S1	12.8 17.0	2 3	16	3,800	3
27. 28.	F-38 M-39	5∠ 88	L1–L2	15.8	2	10	4,340	ĺ
20. 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 2	16 10	4,930 3,820	2 3 3
33.	M-42	79 79	L2–L3 L4–L5	19.1 19.1	2	18	6,030	3
34. 35.	M-42 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	L1L2	10.8	2	8	3,420	1
38.	F-48	51	L3-L4	12.2	2 2 2 3	12	3,893 3,921	1
39.	F-48	51	L5-S1 L4-L5	12.1 18.5	2	8 7	4,160	2
40. 41.	F-50 M-50	90 80	L2-L3	17.3		14	6,693	2 3
42.	M-50	80	L4-L5	18.8	3 2 3	16	5,824	1
43.	F-51	70	L1-L2	13.7		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 9	3,408 3,293	3
46.	M-55 M-55	56 56	L2–L3 L5–S1	24.4 22.8	3 3	11	3,610	3
47. 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 3 3 3 3	12 14	2,920 4,561	2 3 3 2 3 2 3 3 3 3 3 3 3 3
53. 54.	M-59 F-69	64 62	L5-S1 L2-L3	15.8 18.1	4	6	2,170	3
54. 55.	F-69	62	L4-L5	18.9	4	8	3,397	3
56.	M69	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 12	3,700 4,255	3 3
59.	M-70	58 49	L4-L5 L1-L2	21.1 12.6	3 3	10	2,060	3
60. 61.	F–78 F–78	49 49	L1-L2 L4-L5	16.6	4	10	3,643	3
J1.	1-10							

<sup>\*</sup> The extruded nucleus flowed as a high-viscosity liquid.

† Extrusion from out of a preexisting protrusion.

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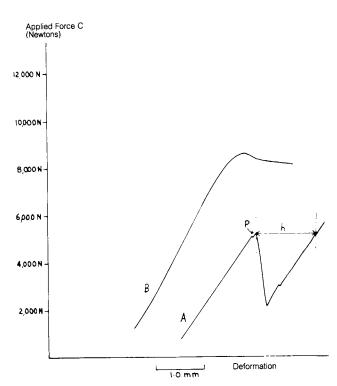
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<sup>\*</sup> Small lateral extrusion.



**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

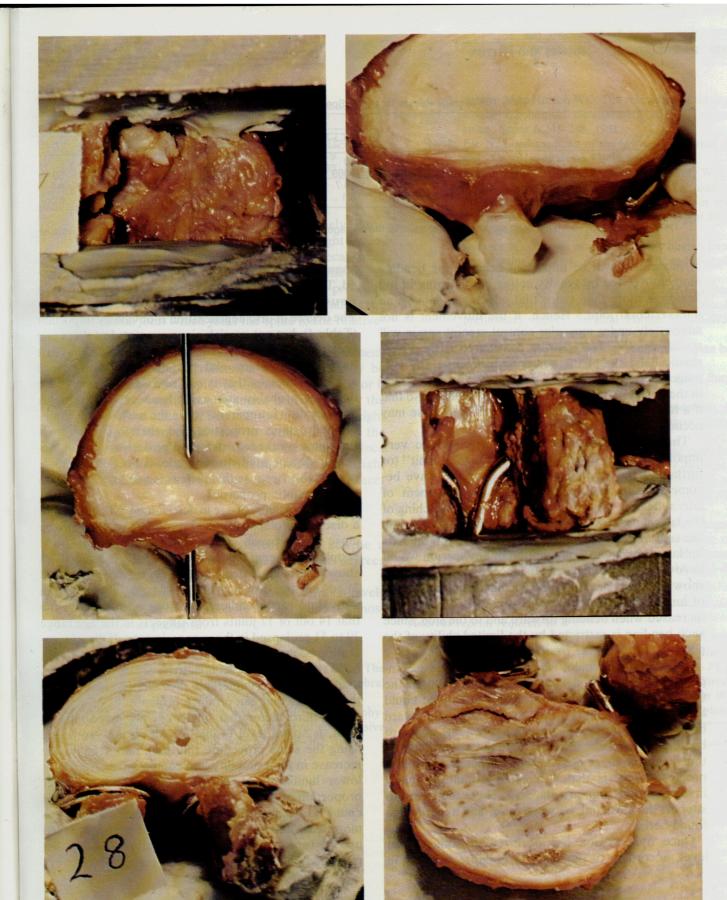
As described, of the 61 intervertebral joints tested, 26 produced a prolapse. However, testing conditions were chosen somewhat arbitrarily. It may be that, for a given specimen, a somewhat higher compressive load would have produced p.i.d. at smaller flexion angles or, conversely, that a higher flexion 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.<sup>9,11</sup> 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

<sup>\*</sup> 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.





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Fig 3 (top left). A posterior view of a specimen with the extruded nucleus lying slightly above center. Fig 4 (top right). A specimen with the intervertebral disc bisected, showing a particularly large nuclear extrusion. Fig 5 (middle left). A specimen with the intervertebral disc bisected. The rod shows the channel from the nucleus to the neural canal. Fig 6 (middle right). A posterior view of a specimen. The protruded annulus can be seen pressing on the metal hooks. Fig 7 (bottom left). The same specimen as shown in Figure 6 with the intervertebral disc bisected. Fig 8 (bottom right). A hyperflexion fracture. The intervertebral disc has been removed to reveal a fracture at the posterolateral edge of the vertebral body.

Table 3. Average Results for the 26 Specimens that Failed by p.i.d.

				Spinal level	-	
	Total	LS_S1	L4-L5	L3-L4	L2-L3	L1-L2
Number of specimens failing by p.i.d. Average compressive force (N) Average flexion angle (degrees) Limit of flexion (from X-rays)*	26 5448 (±2366) 12.8 (±3.8) 9.1 (±2.3)	10 5869 14.7 8.1	6 6106 13.3 12.5	4 6183 11.0 9.7	3 3289 11.7 8.9	3 3917 9.3 6.3

Figures in brackets indicate the standard deviations.

of movement will be important because the bending moment exerted by gravity acting on the trunk in full flexion is more than twice that which can damage an intervertebral joint. Hence, if the movement is not adequately under muscular control, a joint sprain will result. Another factor to be considered is the viscoelastic behavior of the posterior ligaments: any prolonged period in the flexed posture will produce creep in the ligaments and an increased flexion angle, so that if a heavy lift is then attempted, a disc prolapse may occur without ligamentous pain.

The component of lateral flexion could be very important in life. If we bend forward to the "limit" (or further, if the supra/interspinous ligaments have become flaccid) and then introduce a component of lateral flexion, this will produce an extra stretching of the lateral posterior annulus without further extending the supra/interspinous ligaments which lie on the axis for lateral bending. The addition of any torsion would have only a marginal effect since torsion has been shown to be irrelevant to the mechanical derangement of lumbar discs.3 Also, the possibility of an injury is increased when bending forward and to the side, since such bending is restricted by the limited ability of the pelvis to rotate in the frontal plane.

A bending movement which could easily be accomplished by forward flexion would be difficult in the forward and lateral direction, and if attempted could lead to injury. Many clinical surveys state that forward bending and lifting, especially sudden or awkward bending to one side, often precede the onset of symptoms, and the range of symptoms associated with disc prolapse suggests that it is often a multiple injury involving more than just the disc. These facts tend to support the close link between p.i.d. and hyperflexion found here.

Once hyperflexion has occurred at some spinal level, according to our tests, a prolapse is likely in some of the population if even a modest lift is attempted. The analysis of results (Tables 4, 5, and 6) can now be used to indicate which discs are particularly at risk. The degree of disc degeneration is obviously important (Table 4), but not crucial, since even grade 1 discs can prolapse. A slightly degenerated disc (grade 2) has lost its gelatinous shine and has distinct annular rings around a nucleus that is soft and deformable. These discs are probably vulnerable because some annulus degeneration is apparent, while the nucleus still exhibits hydrostatic properties and therefore can burst through a weakened, stretched posterior annulus. Severely degenerated discs (grade 4) do not prolapse, presumably because the nucleus is a fibrous mass with no hydrostatic properties.

The importance of age in determining whether or not a disc will prolapse (Table 5) probably stems from the close link between age and disc degeneration. Most people between the ages of 30 and 50 have discs showing moderate degenerative changes (grades 2 and 3) so that the disc is at its most vulnerable when an individual can be relatively strong and active. Note that 14 out of 17 joints from subjects in the age range 39 to 51 prolapsed in these experiments. If disc degeneration occurs later than usual, when muscle strength, bone strength, and level of activity are all reduced, it is unlikely that the vulnerable disc will be subjected to the traumas that can produce prolapse.

The third factor that influences the probability of p.i.d. is spinal level (Table 6). All discs were tested using the same procedure that took into account the increase in joint mobility and compressive strength at lower lumbar levels in life; nevertheless, a higher proportion of L4-L5 and L5-S1 discs prolapsed. This could be because these discs had been subjected to more wear and tear in life and so had been "softened up" by repeated minor trauma or fatique loading that produced failure of collagen. Disc collagen has a very

Table 4. Prolapsed Disc and Disc Degeneration

1000								
	Degree of disc degeneration							
	1	2	3 -	4				
Number of specimens tested Percent failing by p.i.d.	15 33	17 71	24 38	5 0				

Table 5. Prolapsed Disc and Age

	Age of cadaver (years)							
	< 30 30-39 40-49 50-59							
Number of specimens tested Percent failing by p.i.d.	16 31	14 50	9 78	14 50	8 0			

<sup>\*</sup> The angles from Table 1 have been reduced by 2° to allow for the average 2° of extension per lumbar joint in the erect standing posture.

Table 6. Prolapsed Disc and Spinal Level

	Spinal level					
	L5-S1 L4-L5 L3-L4 L2-L3					
Number of specimens tested Percent failing by p.i.d.	19 53	12 50	13 31	9 33	8 38	

low turnover, and so any repair is extremely slow. Alternatively, these lower lumbar discs might be intrinsically more susceptible to prolapse because of morphologic or biochemical factors. That the upper lumbar discs very rarely prolapse in life is probably because hyperflexion is much more likely to affect the lumbosacral joint which joins the spine to the relatively rigid pelvis.

Two effects of p.i.d. observed on our specimens may be of clinical significance. First, there may be pain if the supra/interspinous ligament is sprained or if the posterior longitudinal ligament is disrupted by the displaced disc material. Second, the loss of disc height of about 0.7 mm could lead to symptoms from the apophyseal joints, especially in lordotic posture, since it has been shown that such a reduction in disc height can greatly increase the compressive force on the facet tips.2

# CONCLUSION

A prolapsed lumbar intervertebral disc can be a compression injury to a joint flexed a few degrees beyond the normal limit.

Slightly degenerated discs at lower lumbar levels from subjects aged between 40 and 50 years are most susceptible to prolapse.

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Address reprint requests to

W. C. Hutton Principal Lecturer PCL115 New Cavendish Street London W1 England

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