Gradual Disc Prolapse

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Fifty-two cadaveric lumbar motion segments were subjected to fatigue loading in compression and bending to determine if the intervertebral discs could prolapse in a gradual manner. Prior to testing, the nucleus pulposus of each disc was stained with a small quantity of blue dye and radiopaque solution. This enabled the progress of any gradual prolapse to be monitored by direct observation and by discogram. Six discs developed a gradual prolapse during the testing period. The injury starts with the lamellae of the annulus being distorted to form radial fissures and then nuclear pulp is extruded from the disc and leaks into the spinal canal. Discs most commonly affected were from the lower lumbar spine of young cadavers. Tests on ten older discs with pre-existing ruptures showed that such discs are stable and do not leak nuclear pulp. Key words: intervertebral disc, prolapse, mechanical fatigue]

DISC PROLAPSE CAN OCCUR as a sudden injury. This has been demonstrated on cadaveric discs[2] and explains why the symptoms of some back sufferers start after an incident involving bending or lifting. However, the majority of patients with low-back pain and sciatica experience no preceding trauma, and often they have no large fragment of disc in the spinal canal. This suggests that a disc can sometimes prolapse slowly, perhaps by the formation of a radial fissure and subsequent gradual "leaking" of nuclear pulp into the spinal canal.

We decided to test this possibility by trying to simulate such "gradual prolapses" on cadaveric discs. We have previously shown that fatigue loading of a disc wedged in flexion can distort the lamellae of the annulus in a manner suggestive of posterolateral radial fissures.[3] In the present experiment, this testing technique was made more physiologic and applied for longer periods in the hope that complete fissures would be formed and allow nuclear material to "leak" into the spinal canal. Prior to testing, a small quantity of radiopaque solution and colored dye was injected into the nucleus. Then, by direct observation and by using discograms, we could monitor the progress of any gradual prolapse caused by the fatigue loading.

DEFINITIONS

We wish to define certain terms we will use in the foregoing. A "radial fissure" is a tear in the annulus. If it extends right to the edge of the disc and allows the escape of fluid injected into the nucleus, then it is "complete radial fissure" and the disc is "ruptured" (but not necessarily prolapsed). A "prolapse" involves the displacement of nuclear material and can be either an "annular protrusion" (when the displaced nuclear material or "pulp" causes the outer annulus to bulge outwards) or a "nuclear extrusion" (when the pulp escapes from the disc). A "sudden prolapse" is a disc injury that can be caused by a single high compressive load applied to a hyperflexed disc (Adams and Hutton 1982[2]). The "gradual prolapse" described in this article is a progressive injury of several stages; an intermediate stage is an annular protrusion but the final stage is a nuclear extrusion.

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MATERIALS AND METHODS

Cadaveric Material. Forty-four lumbar spines were removed at routine necropsies from subjects who had been fully mobile before their death. Four spines were tested "fresh"; the others were stored at −20 C in sealed plastic bags and then thawed at room temperature for 12 hours prior to dissection.

Each spine was dissected into motion segments consisting of two adjacent vertebrae and the intervening soft tissue. Care was taken not to damage the disc or the intervertebral ligaments.

After dissection, the disc of each motion segment was injected with radiopaque fluid (Conray 480) containing a few drops of blue dye (aniline blue). A 21-gauge needle was inserted anteriorly until the tip reached the posterior half of the nucleus pulposus and then a 5 ml syringe was used to inject the fluid. The volume accepted by each intact disc at maximum thumb pressure varied from 0.2 ml to 1.5 ml, with an average of 0.6 ml. Any ruptured discs were revealed by the leaking blue dye, and these were assigned to Group C (see below). Discograms were then taken in the sagittal and coronal planes to determine the size and position of the nucleus prior to testing.

Method. The method of testing the specimens (motion segments) is shown in Figure 1. The specimen is wedged at a predetermined flexion angle and cyclically compressed. The specimen was set in two cups of dental plaster with its sagittal plane at about 15° to the plane of Figure 1, so that one of the posterolateral corners of the disc was more highly stressed than the

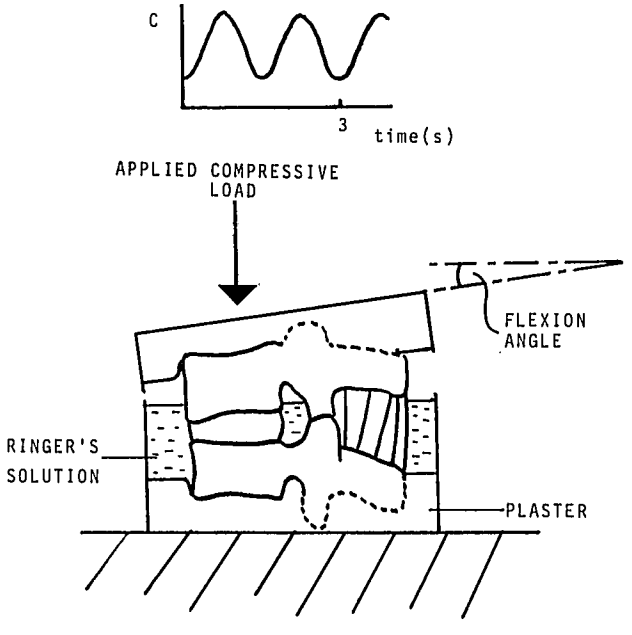

Fig 1. The apparatus used to flex and compress the specimens.

Table 1. Results for the 52 Specimens

	Specimen Details					Testing Conditions			
						Peak load (N)/Flexion Angle			
Group	Number	Sex-Age	Body mass(kg)	Level	Disc degeneration	Initial	Final	Duration(hours)	Mode of Failure
A	1	F-16	56	1-2	1	1,500/14°	—	4	End-plate fracture
	2	M-26	—	4-5	1	2,900/10°		4	End-plate fracture
	3	F-29	—	5-1	2	3,000/10°		2	End-plate fracture
	4	M-31	76	4-5	2	2,000/13°		5	No failure
	5	F-37	—	4-5	2	1,500/12°		1	End-plate fracture
	6	M-39	72	2-3	2	1,500/10°		5	No failure
	7	M-43	72	4-5	2	4,000/9°		5	No failure
	8	M-44	73	4-5	2	2,000/10°		5	No failure
	9	M-45	78	2-3	2	2,500/11°		5	No failure
	10	F-47	—	5-1	2	1,500/10°		5	No failure
	11	F-47	70	5-1	2	2,000/9°		5	No failure
	12	F-47	63	5-1	3	2,000/10°		5	No failure
	13	M-54	—	4-5	3	2,300/9°		4	End-plate fracture
B	1	F-8	32	3-4	1	800/11°	2,600/16°	6	Vertebra crumbled
	2	F-8	32	5-1	1	800/14°	3,000/19°	8	Vertebra crumbled
	3	M-14	47	2-3	1	1,100/11°	1,500/17°	4	End-plate fracture
	4	M-14	47	4-5	1	800/10°	2,000/18°	7	End-plate fracture
	5	F-15	73	2-3	1	1,500/9°	3,500/15°	6	End-plate fracture
	6	F-15	73	4-5	1	1,500/9°	3,750/16°	7	Gradual prolapse
	7	F-20	53	5-1	1	1,250/12°	4,000/18°	6	Sacrum crumbled
	8	F-23	43	5-1	2	1,500/10°	3,500/14°	2	Gradual prolapse
	9	F-23	57	3-4	1	2,000/10°	3,500/13°	6	End-plate fracture
	10	F-23	57	5-1	1	2,000/12°	3,500/15°	6	Gradual prolapse
	11	M-25	75	4-5	2	2,000/9°	7,000/13°	6	End-plate fracture
	12	M-28	63	5-1	1	2,000/12°	4,500/16°	8	Gradual prolapse
	13	M-28	73	5-1	1	2,500/9°	6,000/14°	7	Sacrum crumbled
	14	M-34	56	2-3	2	1,500/11°	2,500/15°	5	End-plate fracture
	15	M-34	56	4-5	2	1,500/12°	3,500/16°	6	End-plate fracture
	16	M-34	52	5-1	2	1,100/13°	3,500/15°	5	Gradual prolapse
	17	M-39	85	2-3	2	1,750/10°	3,000/12°	4	End-plate fracture
	18	M-39	85	4-5	2	2,000/13°	3,750/15°	5	End-plate fracture
	19	M-40	69	2-3	2	1,750/9°	4,500/12°	6	End-plate fracture
	20	M-41	71	3-4	2	2,500/9°	4,500/13°	5	End-plate fracture
	21	M-41	71	5-1	2	2,750/11°	5,500/15°	6	End-plate fracture
	22	F-42	59	3-4	2	1,500/9°	4,000/15°	5	End-plate fracture
	23	F-42	59	5-1	2	1,500/12°	5,000/16°	7	End-plate fracture
	24	M-43	—	4-5	2	2,000/14°	3,500/15°	5	End-plate fracture
	25	M-44	—	4-5	2	2,500/8°	2,500/8°	<1	Gradual prolapse
	26	M-47	70	2-3	2	1,000/7°	4,000/13°	8	End-plate fracture
	27	M-48	73	1-2	2	1,750/9°	6,000/13°	7	End-plate fracture
	28	M-50	39	5-1	3	1,500/11°	3,500/14°	8	End-plate fracture
	29	F-53	71	3-4	2	2,000/12°	3,000/15°	4	End-plate fracture
C	1	M-40	69	4-5	2	2,000/10°	4,500/14°	5	End-plate fracture
	2	F-42	—	3-4	2	1,500/13°	2,000/15° (H)	4	End-plate fracture
	3	F-42	—	5-1	2	2,000/13°		4	End-plate fracture
	4	M-43	—	2-3	2	(H)		—	Vertebra crumbled
	5	M-45	—	5-1	2	2,500/13°	3,000/17°(H)	5	End-plate fracture
	6	M-47	71	4-5	3	(H)		—	End-plate fracture
	7	F-48	58	5-1	2	1,500/10°		6	Sudden prolapse
	8	M-50	—	5-1	3	1,500/13°	2,000/15° (H)	3	End-plate fracture
	9	F-53	71	5-1	2	(H)		—	Sudden prolapse
D	1	F-33	50	5-1	2	3,000/15° (H)		3	Sudden prolapse

other by flexion. Throughout the testing period, the specimen was immersed in a bath of Ringers solution so that the swelling pressure of the disc could operate to reduce the outflow of fluid caused by loading. Full details of the apparatus are given elsewhere.[3]

Fifty-two motion segments from 44 cadavers aged between 8 and 54 years were tested. The motion segments were separated into four groups, each of which had a different testing procedure. Groups A and B were attempts to produce gradual prolapse on unruptured discs, while Group C were attempts to produce prolapses down existing ruptures. The single unruptured specimen in Group D was tested to demonstrate that fatigue could "soften-up" a disc for sudden prolapse.

Group A: This group consisted of the first 13 motion segments that did not have ruptured discs, as revealed by the leaking of blue dye at injection. Each specimen was wedged at its physiologic limit of flexion (as determined by the elastic limit of the supraspinous and interspinous ligaments: Adams, Hutton and Stott 1980[1]) and subjected to a cyclic compressive force 40 times per minute. The compressive force oscillated sinusoidally from a minimum of 500N to a maximum value that varied between 1,500N and 4,000N depending on the age, sex, and body mass of the cadaver. The minimum force is appropriate for erect standing,[21] and the maximum simulates the action of the back muscles during heavy lifting.[16] The experiment was stopped after 5 hours or at the prior

failure of the specimen. The disc was injected with radiopaque fluid, and a second discogram was taken. (The first injection of radiopaque fluid appeared as a faint halo on this second discogram.) Then the disc was cut through along its midplane for visual examination.

Group B: The testing procedure chosen for Group A did not produce disc failure in any of the specimens, so we decided to modify it.

Group B consisted of 29 motion segments that did not have ruptured discs. Each specimen was flexed and cyclicly compressed, as before, but now the flexion angle and the peak load were increased at regular intervals throughout the testing period.

Initially, both the flexion angle and peak load were set at values within the physiologic range. After 1 hour, it was found that the disc and posterior ligaments had adapted to this wedging angle so the motion segment was flexed an extra 1° without exceeding the elastic range of these tissues. This process was repeated several times, at 1-hour intervals, until the flexion angles were higher than for Group A specimens, even though the disc and ligaments were both undamaged. (This process is physiologic, as explained in the Appendix.) Eventually, the flexion angle could not be further increased without risk of damaging the specimen, and then this final limit of flexion was kept for the rest of the test. Its value ranged from 12° and 19°.

With the flexion angle fixed, the peak compressive force was then increased by increments of 500N at half-hour intervals until failure eventually occurred. This ensured that each specimen was tested for at least half an hour (1,200 cycles) at loads near its physiologic limit.

At the end of the test, the disc was again injected with radiopaque fluid, and a second discogram was obtained. The disc was then cut through in the midplane and examined.

Group C: This group comprised nine motion segments that had disc ruptures revealed by leaking of the Conray dye mixture. They were subjected to various testing procedures to see if the rupture made them particularly susceptible to disc prolapse.

The exact procedure used on each specimen can be appreciated from Table 1. The following examples should make this clear. Specimen C1 was loaded as for Group B specimens with the peak load and flexion angle increasing from 2,000N and 10° to 4,500N and 14° during the 5-hour test. Specimen C7 was tested with a peak load of 1,500N and flexion angle of 10° throughout the test. The symbol (H) means that the specimen was hyperflexed and loaded once to failure in order to simulate a sudden prolapse. This experimental procedure is described in detail elsewhere.[2] This hyperflexion test was performed after fatigue loading in specimens C2, C5, C7, and C8 and instead of fatigue loading in specimens C4, C6, and C9.

After testing the disc was simply cut open and examined.

Group D: The single specimen in this group was not ruptured. It was fatigue loaded for 3 hours and then loaded to simulate a hyperflexion injury. We wanted to demonstrate that fatigue loading can deform the lamellae of the annulus and facilitate a sudden prolapse.

RESULTS

The outcome of each test is given in the final column of Table 1.

Of the 13 Group A specimens, five failed during the course of the 5-hour testing period, all by fracture of the vertebral end-plate. The average value of the peak load applied to these specimens was 2,240N. None of the 13 discs sustained a prolapse. Specimen 8, however, showed a complete posterolateral radial fissure on the "after" discogram that was not present before testing, and four other specimens showed severe distortion of the lamellae after testing (as described in Adams and Hutton 1983[3]).

The 29 Group B specimens were all tested to failure, and this occurred at an average peak load of 3,800N. Usually it was the vertebral body that failed, either by fracture of the end-plate or by crushing of the anterior wall.

In six specimens, there was a "gradual prolapse" of the intervertebral disc. Blue-stained nuclear pulp first appeared, rather like a bruise, just under the surface of the annulus (Figure 2). In five specimens, the pulp appeared at the posterolateral corner that was being more highly stressed, and in the other it appeared centrally. In a matter of minutes, the pulp worked its way to the surface and then oozed out. Very little was extruded from five of these discs even after an hour of further cyclic loading or when the peak load was increased until the vertebrae were crushed. Figure 3 is typical of these five. Considerably more pulp was extruded from one 23-year-old specimen (Figure 4), but practically none could be coaxed from another, aged 34. The prolapse in the 44-year-old specimen (number B-25) occurred after only 200 loading cycles. This disc must have been about to prolapse in life. Four of the

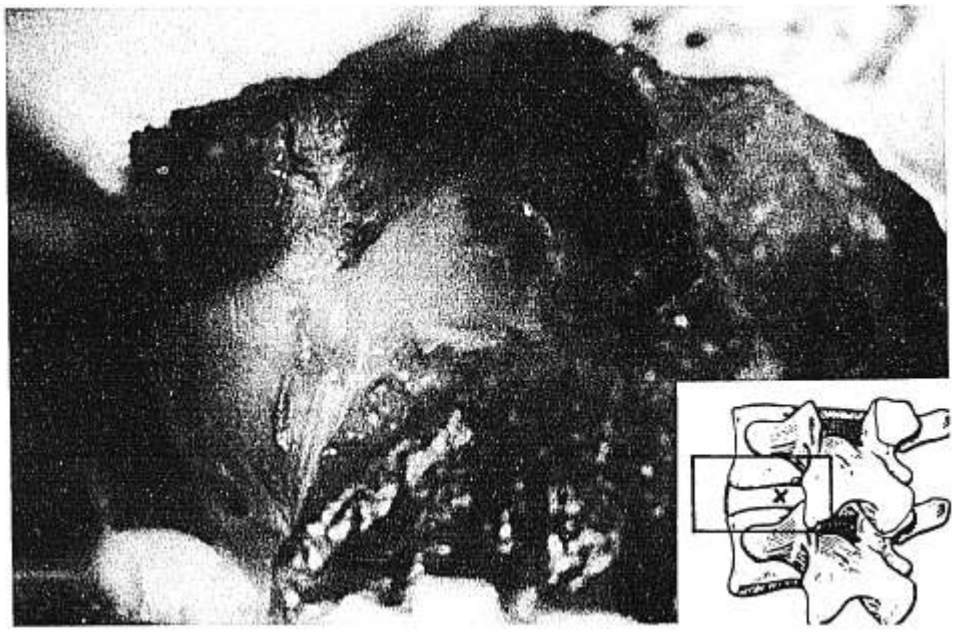

Fig 2. The first sign of gradual prolapse. The blue-stained nuclear pulp has just reached the surface at the posterolateral corner of the disc (Specimen B-8). The squared off section on the inset shows the frame of the photograph.

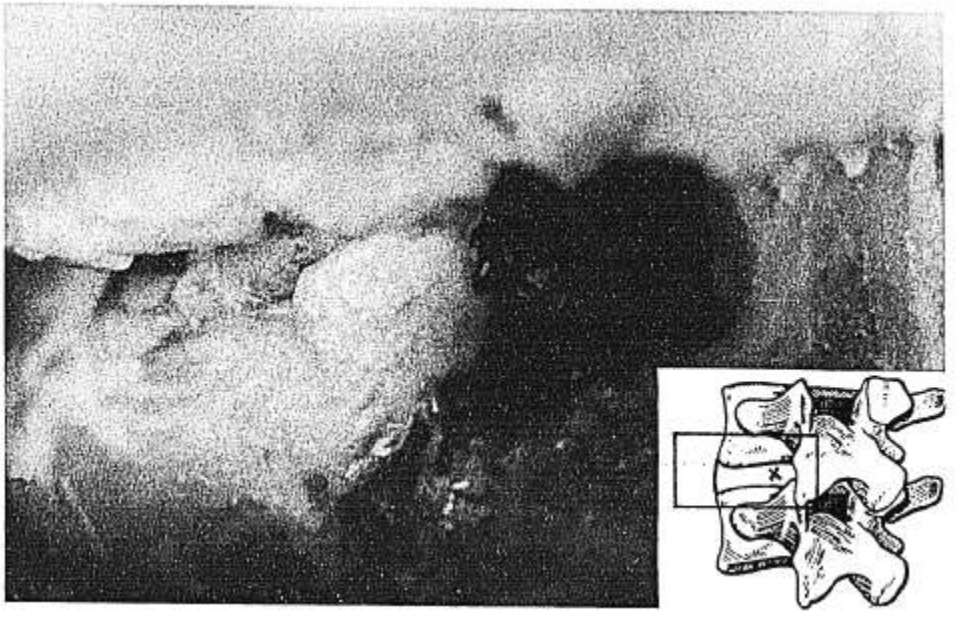

Fig 3. A gradual prolapse has resulted in the extrusion of blue-stained nuclear pulp (Specimen B-12).

gradual prolapses occurred in specimens aged 28 or under, and four were lumbosacral joints. The six discograms taken before testing usually indicated a particularly thin posterior annulus. The "before" and "after" discograms confirmed the formation of a complete posterolateral fissure during the testing period (Figure 5). Visual inspection of the bisected disc showed the lamellae distorted to form a radial fissure, and a thin line marking the final route of the nuclear pulp through the annulus (Figures 6 and 7). Nearly all Group B discs had the lamellae distorted after testing; three notable exceptions (specimens 14, 19, and 22) all had the nucleus located more anteriorly than usual.

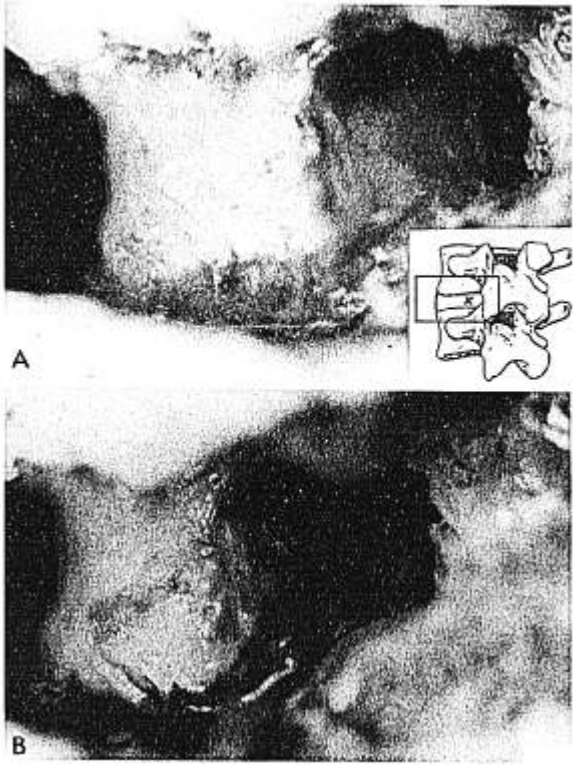

Fig 4. Specimen B-10 just before **A,** and after **B,** a nuclear extrusion.

The ruptured discs of Group C specimens did not prove to be particularly susceptible to prolapse. Fatigue testing did not cause any nuclear material to "leak," and there were only two sudden prolapses in the hyperflexion tests.* One of these was a nuclear extrusion (Specimen C-9, flexion angle 17°, compressive load 5,480 N), and one was an annular protrusion (Specimen C-7; flexion angle 16°, compressive load 5,100 N).

The nonruptured Group D specimen was subjected to fatigue loading followed by a hyperflexion test, and it sustained a sudden prolapse by nuclear extrusion down a radial fissure (flexion angle 17°, compressive load 5,900 N).

DISCUSSION

This experiment shows how cadaveric lumbar discs can gradually prolapse. The experimental procedures required to produce this type of prolapse are examined in the Appendix. We conclude that they are not unphysiologic except in one regard, and that is the absence of diurnal fluid flow in the disc. In life, this would retard the process of fissure formation so that prolapse may occur over days or months, instead of hours, as in the experiment. Despite uncertainty over the time scale, we suggest that the following description of the mechanics of gradual prolapse can be applied to living people. Five stages can be recognized.

Stage 1

Self-selection of the disc. We could only induce gradual prolapse in discs that had a soft, pulpy nucleus, and a posterior annulus that was much thinner than the anterior annulus. In effect this meant young discs at the L5–S1 and L4–5 levels with discograms showing "posterior position" of the nucleus as described by Jayson, Herbert, and Barks (1973)[18]. This position of the nucleus might be natural or it might be acquired, perhaps as a consequence of lordotic posture causing "pressure atrophy" of the posterior annulus.[17,19]

Stage 2

Distortion of the lamellae of the annulus. Activities such as repeated bending and lifting distort the lamellae of the annulus as shown in Figure 8.[3] The lamellae become tightly curved and

*In our previous hyperflexion tests, 14 of 17 discs between the ages of 39 and 51 prolapsed.

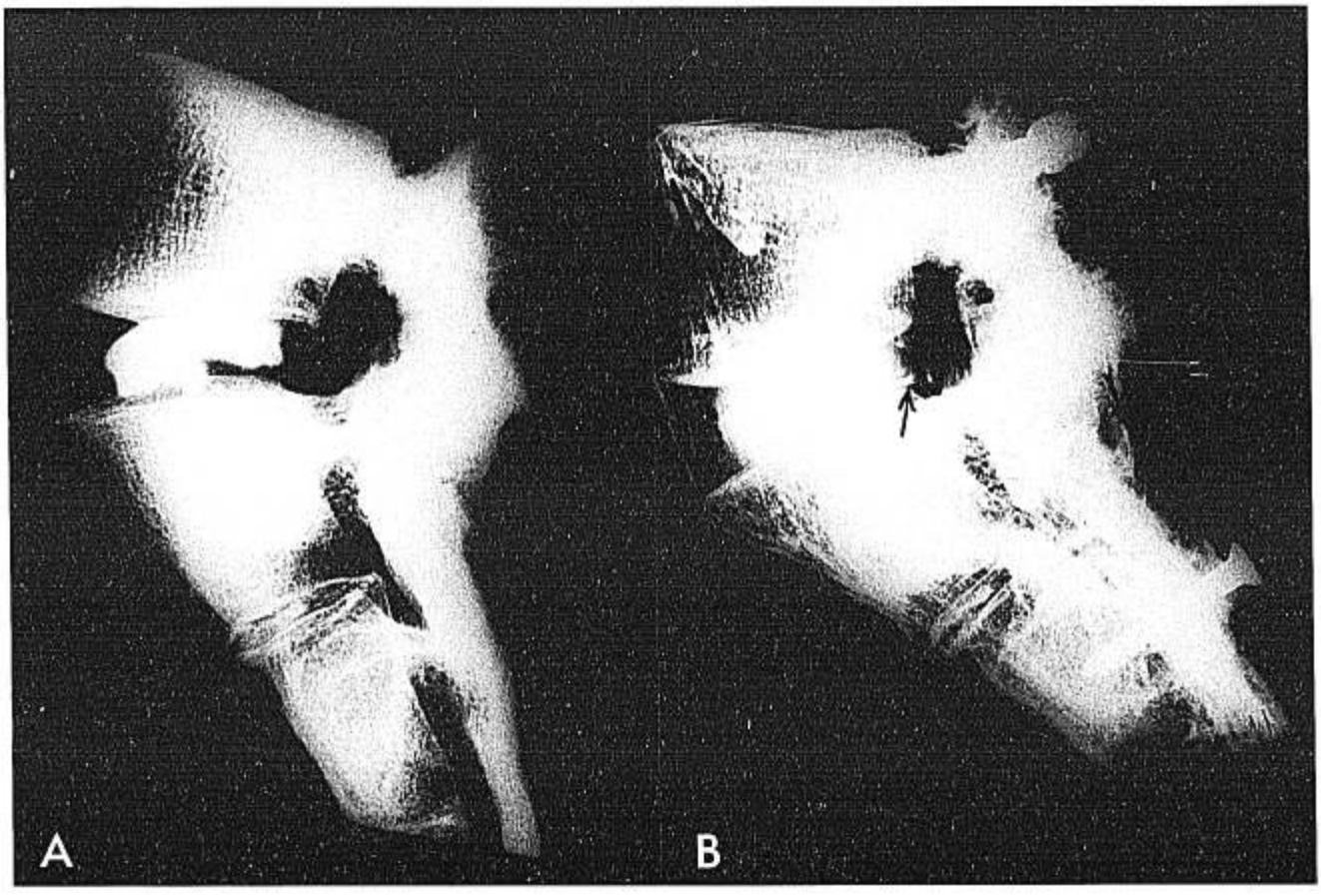

Fig 5. Discograms of Specimen B-10 before **A,** and after **B,** testing. The arrow indicates the fissure produced by testing. Some radiopaque dye from the second injection has leaked down the fissure and can be seen on the anterior surface of the specimen in B.

packed together in the posterolateral corners, and the nucleus migrates down these developing posterolateral radial fissures.

If there is a regular tendency to load the spine asymmetrically by bending forward and to one side (perhaps because of a dominant hand) then the radial fissure will become more developed in the contralateral corner of the disc.

Stage 2 was reached by the great majority of Group A and B discs. In the upper lumbar spine, where the posterior annulus tends to be thicker and stronger the discs rarely pass this stage, but in the lower lumbar spine they can progress to Stage 3.

Stage 3

Breaking through the lamellae. The nuclear pulp must penetrate the tightly packed lamellae of the posterior annulus to escape from the disc. The distance may be only two or three millimeters in the posterolateral corners (rather more centrally) but the collagen "fences" still present a formidable barrier. This can be demonstrated by cutting away the outermost layer of annulus during a test: this was done twice, and in both cases there was an immediate large "prolapse" (Figure 9). Without the aid of such intervention,

Fig 6. The disc of Specimen B-8 cut through in the midplane after testing. A complete posterolateral fissure produced by testing is shown in the lower left-hand corner.

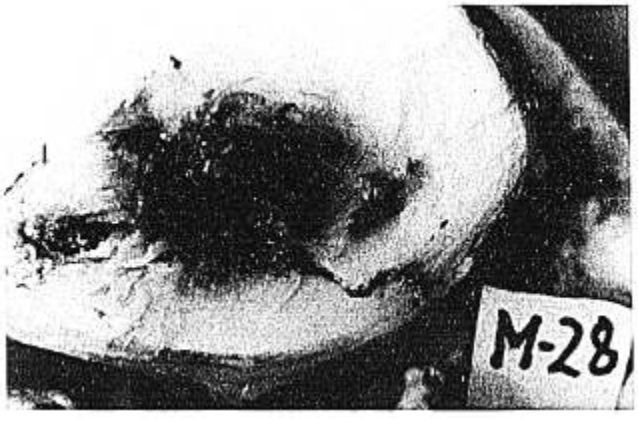

Fig 7. The disc of Specimen B-13 cut through in the midplane after testing. A complete posterolateral fissure produced by testing is shown in the lower right-hand corner.

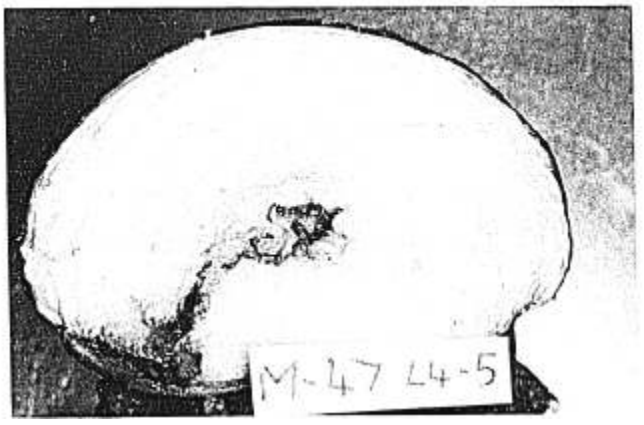

Fig 8. Specimen C-6 cut through after testing. The complete posterolateral radial fissure was already present in the disc before testing. Note the distortion of the lamellae. No nuclear material was extruded from this fissure during the test.

the nuclear pulp creates a narrow, often tortuous, channel through the lamellae (Figures 6 and 7).

The final barrier consists of the outermost lamella and adhering posterior longitudinal ligament. Being unsupported on the dorsal side, it can stretch and bulge outwards in response to the pressure of the pulp accumulating behind it. In our tests, this bulging tended to be slight, but in life it can be quite marked, and the result is the familiar annular protrusion (Figure 10). In mature discs with a fibrotic nucleus, this may be the final stage (Figures 8 and 10). Younger discs, however, can progress to the next stage.

Stage 4

Extrusion of nuclear pulp. The pulp may appear at the mouth of the fissure, but quite commonly it migrates behind the final barrier and emerges somewhere else. Little pulp was extruded in our experiments, but this may not be the case in life when it may continue to leak over a period of days or months. It is possible that nucleus regeneration could lead to recurrent prolapse.

Stage 5

Ruptured but stable. A ruptured disc does not continue to leak nuclear pulp indefinitely. In our Group C tests on ruptured discs aged over 40, we could not get any leaking of nuclear material in response to fatigue loading. This is probably because the nucleus of these discs consists of fibrous lumps,[5] and these are too large to pass down a fissure, except under the extreme loading of a hyperflexion test. Even then, the self-sealing properties of the annulus (Markolf and Morris 1974[20]) ensure that prolapse is just about as difficult as in a nonruptured disc.

The 5 stages are depicted in Figure 11. It is likely that some of them are symptomatic. Stages 3 and 4 could give rise to pain from a variety of sources. Stage 3 is equivalent to the "annular herniation" described by Fraser et al,[11] and this is the type of disc that most frequently gives symptomatic pain reproduction when injected with fluid.

Now, what evidence is there that the disc lesions shown in Figure 11 actually do occur outside our laboratory? Let us consider the Stages 2–5 separately. The radial fissures of Stage 2 occur in cadaveric lower lumbar discs of all ages.[14] The annular protrusion that sometimes marks the end of Stage 3 is often seen at surgery and is more common than bulk nuclear extrusion, especially in young patients.[23] A slight annular protrusion in an older disc (Figure 8) is probably the "concealed rupture" described by Dandy.[8] The leaking nucleus of Stage 4 would be difficult to observe directly, but its existence may be inferred from the frequent occurrence, in young people, of sciatica without neurologic deficit,[12] which suggests that a nerve root is being irritated without being compressed. Discography shows ruptures in young, non-degenerated discs,[5] and they are more likely to be symptomatic than ruptures in older discs.[13] Finally, Stage 5 is confirmed by the high incidence of disc ruptures in (presumably asymptomatic) discs from cadavers aged over 34[13] and by the ruptures found in asymptomatic volunteers.[15]

The "gradual prolapse" described above is quite distinct from the "sudden prolapse" simulated previously.[2] The main differences are set out in Table 2. Note however that a sudden prolapse can occur to a disc in the various stages of gradual prolapse, as demonstrated by the Group C and D tests described above.

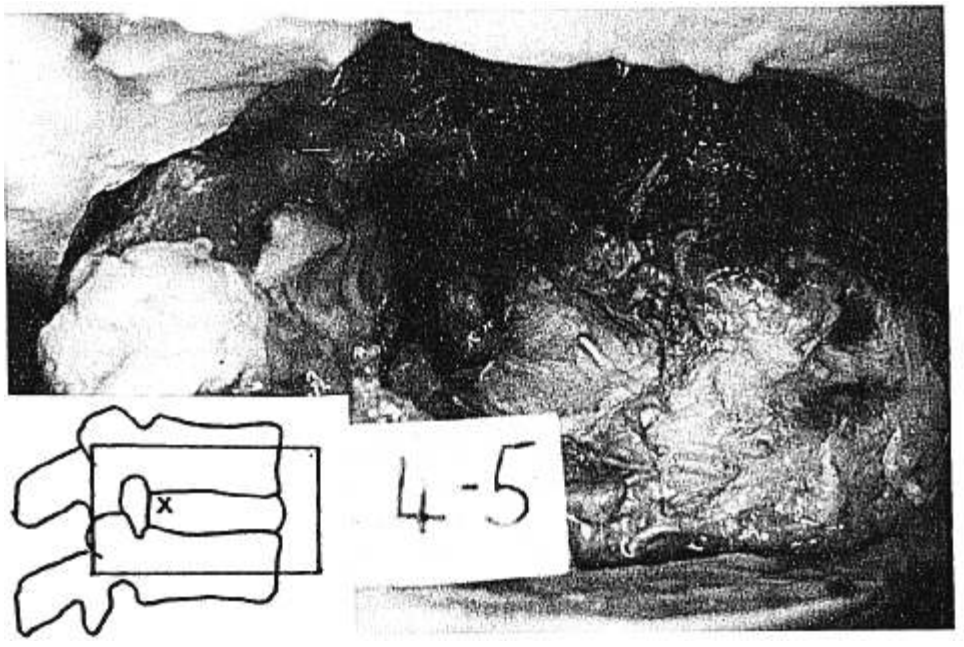

Fig 9. Specimen C-1 had a pre-existing complete radial fissure. However no nuclear material could be forced down it by fatigue loading until a slice of annulus 1 1/2 mm thick was shaved off the posterolateral corner. There was then an immediate sudden extrusion of blue stained nuclear pulp.

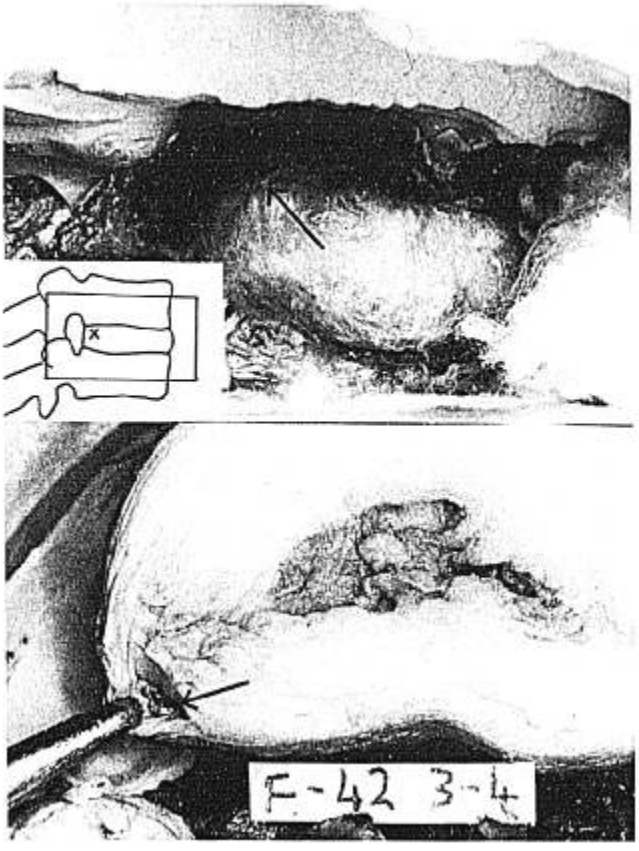

Fig 10. A, Specimen C-2 had a pre-existing complete radial fissure leading to a distinct annulus protrusion (arrowed). No nuclear material was extruded from this protrusion during testing. **B,** When the disc was cut through after testing, nuclear pulp could be removed from behind the bulging outermost lamella (arrowed).

CONCLUSION

"Gradual prolapse" is a slow, progressive injury that occurs more readily in nondegenerated discs. It can be caused by activities such as frequent bending and lifting. The injury starts with the lamellae of the annulus being distorted to form radial fissures. Nuclear pulp then breaks through the distorted lamellae and can cause the outermost lamella, and the adhering posterior longitudinal ligament, to protrude. In the final stage, the pulp is extruded from the disc and leaks into the spinal canal.

Ruptured discs in older age groups seem stable and do not leak nuclear pulp.

Table 2. The Difference Between Gradual and Sudden Prolapse

Gradual Prolapse	*Sudden Prolapse*
Young discs most at risk.	Mature discs most at risk.
Caused by fatigue compressive loading of a flexed disc.	Caused by a single high compressive load on a flexed disc.
Occurs slowly, probably over days or months.	Occurs in less than one second.
Lamellae of the annulus are distorted.	Lamellae are not noticeable distorted.
A protruding annulus often precedes extrusion of nuclear pulp.	A centrally protruding annulus can be the final stage.
Nuclear pulp leaks in small quantities.	Nuclear pulp can be extruded in bulk.

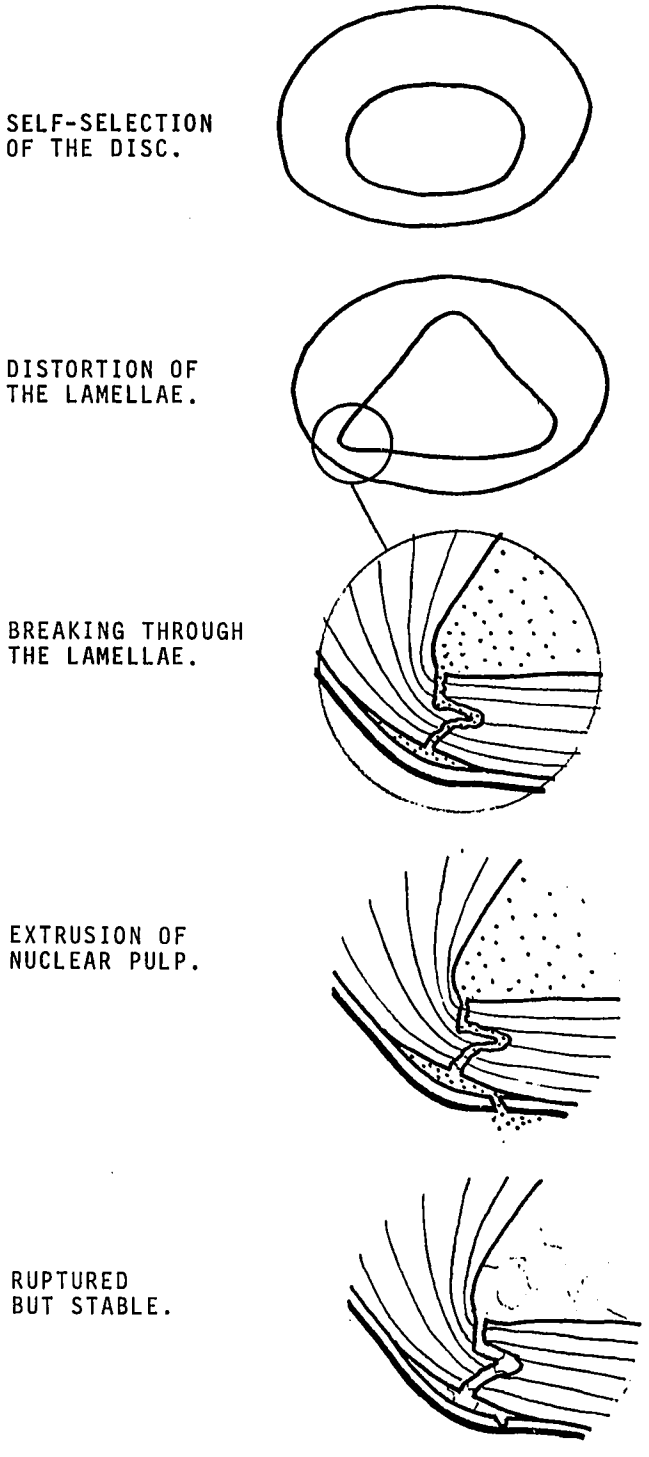

Fig 11. The five stages of gradual disc prolapse.

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APPENDIX

Appraisal of the Testing Procedure

In this Appendix we examine several aspects of the testing procedure to see how physiologic they were.

The Fluid Injections. It is unlikely that the mechanism of gradual prolapse in Group B specimens was significantly affected by the fluid injections since the same distortion of the lamellae to form radial fissures can be induced in noninjected discs.[3] Also, by the time the nuclear pulp worked its way down through the lamellae at the end of the testing period, its fluid content would have been normal, as the following argument shows.

When a small quantity of aqueous solution is injected into the nucleus of a young disc, it is incorporated into the proteoglycan-water gell.[5] This is shown when the discs are cut open at the end of the test—there is no free solution in the nucleus. A typical injection of 0.6 ml will increase the water content of a nucleus by about 12%, since typical nuclear dimensions are 3 cm wide by 2 cm deep by 1 cm high, and about 80% of this volume is water. This extra water, however, will be squeezed out again by fatigue loading. The nucleus of a flexed disc loses 10% of its water content after four hours of loading at bodyweight.[4] Therefore at the higher load levels employed in this test, it is likely that the fluid content of the nuclear pulp would have been reduced to normal after the first few hours of loading. It is only in the later stages of testing that the loading regime in the Group B tests become severe enough to damage the disc. It is worth noting that it is young discs that sustain gradual prolapse, and these are the ones that accept less fluid at injection.[5] The Group A motion segments were loaded severely from the start, before the injected fluid had been expelled. The injected fluid raises the pressure in the nucleus pulposus for the same applied load, and this could explain why some of these specimens sustained end-plate fracture at low loads.

The Compressive Loads and Flexion Angles in the Group B Tests. The gradual prolapses obtained in Group B discs depended on the technique of increasing both the flexion angle and the peak compressive load during the course of the test. This technique is entirely physiological in the sense that neither the bones nor the ligaments of the motion segment sustain any irreversible damage before prolapse occurs. The peak compressive loads are well within the scope of the back muscles. When bending forwards and making heavy lifts the back muscles can generate an extensor moment of about 300 Nm[6] and this will cause a compression force of about 6000N to act on the lumbar spine.[16] The compressive forces applied in this experiment were much lower than this (see Table 1). The final flexion angles are only marginally higher than those measured in life on healthy young volunteers.[22] If these volunteers had remained bent over for a long period, they would have 'rested on their ligaments'[10] and this would inevitably have caused creep in the posterior ligaments and an increase in flexion. The extra flexion is not entirely due to ligament creep but is also caused by the disc losing height during loading as fluid is expelled from it[4]. Height lost by the disc means the posterior ligaments gain some slack and can allow more flexion. This process can be appreciated by trying to touch your toes first thing in the morning, and then again last thing at night, when the exercise will be found much easier.

The Number of Loading Cycles. It is hard to make 40 lifts per minute nonstop for up to 8 hours. Our intention was to condense several days work into one afternoon. The drawback with this is that it does not allow time for any repair processes to act, as they may do in life. Cellular mechanisms of repair are so slow that their effect over several days, or even months, can be disregarded. This point is discussed in Adams and Hutton 1983.[3] However, there is a physical 'repair' mechanism that may be effective over a 24 hour period, and this is the absorption of fluid by the disc from surrounding tissues when the spine is unloaded during sleep.[4,9] The resulting fluid redistribution may reverse or hinder the deformation of the lamellae that occurs in Stage 2 of the gradual prolapse. This means that, in life, Stage 2 will progress to Stage 3 only if the rate of fatigue damage outstrips the nightly repair process. For this reason, we must be vague over the time scale and severity of loading required to produce gradual prolapse in life.