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The effect of posture on diffusion into lumbar intervertebral discs*

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INTRODUCTION

The lumbar intervertebral discs are avascular structures which are so large that metabolite transport is barely adequate to supply the most inner regions (Maroudas, Stockwell, Nachemson & Urban, 1975) and it is possible that nutritional deficiency can lead to disc degeneration (Nachemson, Lewin, Maroudas & Freeman, 1970; Holm & Nachemson, 1982).

Nutrients reach the disc cells by two routes: from the blood vessels which lie around the periphery of the annulus fibrosus (the annular route) and from the vascular cavities in the central portion of the vertebral endplate (the endplate route). The transport processes are fluid flow and diffusion. Fluid flow, caused by pressure changes in the disc, is more effective in transporting large molecules, such as proteins, while diffusion is more effective for small molecules (Urban, Holm, Maroudas & Nachemson, 1982).

In a previous experiment, the authors have shown that postural variaton (the way you sit and stand) influences the fluid flow component of the nutrient supply: postures involving flexion of the lumbar spine cause a larger outflow of fluid from the disc, especially the nucleus pulposus, than do erect or lordotic postures (Adams & Hutton, 1983). Any outflow of fluid is reversed when the spine is unloaded (at night, for example), so flexed posture has the overall effect of increasing fluid exchange in the disc and hence improves the transport of nutrients. The other transport process, diffusion, may also be influenced by posture. This seems possible, since changes in posture alter the shape of the annulus fibrosus and may therefore affect diffusion by the annular route.

This possibility was investigated using a radioactive tracer technique. Cadaveric lumbar motion segments were loaded to simulate flexed or lordotic postures while immersed in a saline bath containing a radioactive tracer. The tracer could freely diffuse into the disc through the annular route but was not expected to diffuse through the endplate route because of the greater path-length from the bath, and because of blood-clotting postmortem. After a set loading period, the disc was cut up into small pieces and the radioactivity of each piece measured using a gamma-counter. By this technique, it was possible to measure the concentration of tracer that had diffused into various locations in the disc, and to see how these concentrations were affected by postural variation.

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MATERIALS AND METHODS

Cadaveric material and setting procedure

Nineteen lumbar spines were removed at routine necropsies from subjects, aged between 12 and 57 years (mean 36 years), who had been fully mobile prior to death. The spines were stored at −20 °C in sealed plastic bags for up to four weeks until required. They were then thawed in their bags in a refrigerator for twelve hours.

Each spine was dissected into two motion segments each consisting of two vertebrae and the intervening soft tissue. The upper and lower surfaces of each motion segment were scraped clean of all soft tissue and the contents of the neural arch and intervertebral foramina were removed with forceps. The anterior longitudinal ligament was removed with a scalpel, but that part of the posterior longitudinal ligament that could not be removed by forceps was left adhering to the disc. The effect of this is discussed in Appendix I. The motion segment to be tested was then set in two cups of dental plaster with the midplane of the disc parallel to the ends of the cups. Screws in the spinous processes and exposed facet joint surfaces ensured that there could be no relative movement between specimen and plaster. During setting, which took 40 minutes, the specimen was covered with thin polythene film to prevent dehydration.

The other motion segment from the same spine was wrapped in polythene film and stored in a sealed polythene bag at +2°C for up to 24 hours before testing. Of the 38 motion segments tested 3 were at L5-S1, 7 at L4-5, 12 at L3-4, 7 at L2-3 and 9 at L1-2.

Apparatus

The motion segments, set in their cups of plaster, were loaded on a Mayes servo-controlled materials-testing machine as shown in Figure 1. The angle plate could be adjusted to produce any desired wedging of the specimen. An X–Y plotter recorded the compressive force on the specimen and the vertical movement of the ram of the testing machine. The bath surrounding the specimen consisted of a thin polythene cylinder of the same diameter as the lower cup of plaster. The plaster held the polythene firmly against the side of the cup and formed a watertight seal with it. The bath was filled with about 75 ml of Ringer's solution containing a radioactive tracer so that the whole of the disc was immersed when the specimen was being loaded.

The radioactive tracers

The tracers used were iodine-131 (in aqueous solution as ^{131}I-sodium iodide) and sodium-22 (in aqueous solution as ^{22}Na-sodium chloride). These are both gamma-ray emitters, so the activity of the disc samples after the test could be measured directly using an automated gamma-counter (Wallac Decem-GTL).

Iodine and sodium were chosen to represent small negatively charged solutes and small positively charged solutes respectively. Since the disc matrix has a considerable negative fixed charge density, a difference in diffusion behaviour between sodium and iodine might be expected.

Method

Motion segments were tested either in 'flexed posture' (such as sitting in a low chair, or on the floor with the knees up to the chest) or 'erect posture' (such as

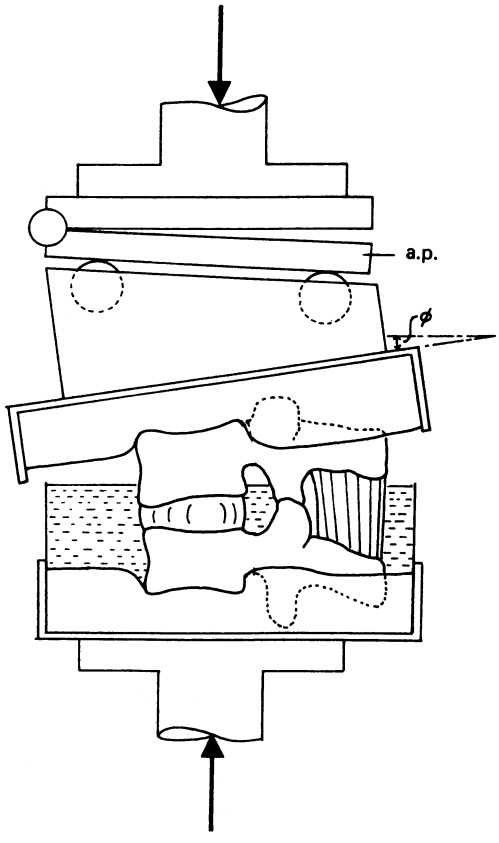

Fig. 1. The apparatus used to compress the motion segments. The adjustable angle plate (a.p.) wedges the motion segment in flexion or extension during loading. ϕ is the flexion angle.

standing or sitting upright with the lumbar lordosis intact: see Figure 6). Flexed posture was simulated by wedging the motion segment in full flexion and compressing it with a load equal to cadaver bodyweight (Adams & Hutton, 1983). The average flexion angle used was 12°. Erect posture was simulated by wedging the motion segment in 3° of extension and compressing it at bodyweight as before. This extension angle was 1° more than that used previously, because the cadaver spines used in these experiments were younger and more mobile.

Each lumbar spine yielded a pair of motion segments, one of which was tested in each posture. In half the pairs, the 'flexed' motion segment was lower in the lumbar spine, and also in half the pairs the flexed motion segment was tested first. This selection procedure ensured that exactly equivalent groups were tested in flexed and lordotic posture.

The motion segment was loaded while wedged at the appropriate angle. One ml of tracer solution with an activity of 2μCi was thoroughly mixed with 150 ml Ringer's solution, and half this mixture was used to fill the bath. The other half of the mixture was kept for the paired motion segment to be tested in the alternative posture.The solution and apparatus were equilibrated at room temperature (20 °C ± 1 °C) before the test, and then during the test the bath was undisturbed. At the end of the testing period, the bath was drained, the disc dabbed dry with cotton wool, and the load was then removed.

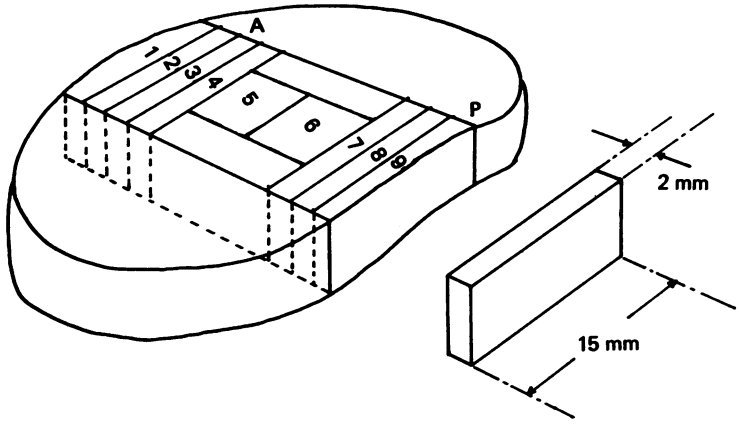

Fig. 2. Nine specimens were cut out from the central strip of each disc as shown. A, anterior margin of disc; P, posterior margin. Specimens 5 and 6 are referred to in the text as the anterior nucleus and the posterior nucleus respectively.

The disc was excised and a central strip 15 mm wide was cut out (Fig. 2). A layer of disc material and hyaline cartilage less than 1 mm thick was left adhering to the vertebral bodies. A special cutting tool (Adams & Hutton, 1983) was used to cut the strip into 2 mm thick slices from front to back. Slices 1 to 4 were counted from the anterior edge of the annulus, and 7 to 9 were from the posterior edge of the annulus. The remaining part of the strip, between slices 4 and 7, was cut up to obtain slices 5 and 6 which represented the anterior and posterior parts of the nucleus respectively.

The nine cut slices of the disc were put in nine small pre-weighed plastic bottles with tight-fitting lids.* A tenth bottle was filled with a sample of the bath solution after testing. This sample was obtained from the part of the bath furthest from the disc so that its activity would not have been substantially reduced by diffusion into the disc. The ten plastic bottles were weighed again to obtain the weight of the samples. They were then fed into the gamma-counter and counts (30 minutes) made of each. The counts were repeated once or twice with the bottles re-arranged in random order each time.

The testing period (that is, the time the disc was immersed in the saline/tracer mix) was set at four hours. Several one hour tests were carried out, but these tests proved inaccurate (for reasons explained in Appendix I) and so were discontinued. Some pairs of motion segments were preloaded for three hours in a bath of pure Ringer's solution, before this was replaced with the usual Ringer's/tracer mix. In these tests the wedging angle and compressive load were constant for seven hours. This was done to see if fluid flowing out of the loaded discs had any significant effect on the diffusion of tracer into the disc. (Fluid flow is considerable in the first three hours of loading: Adams & Hutton, 1983.)

* The time interval between draining the bath and placing the slices into bottles was less than four minutes.

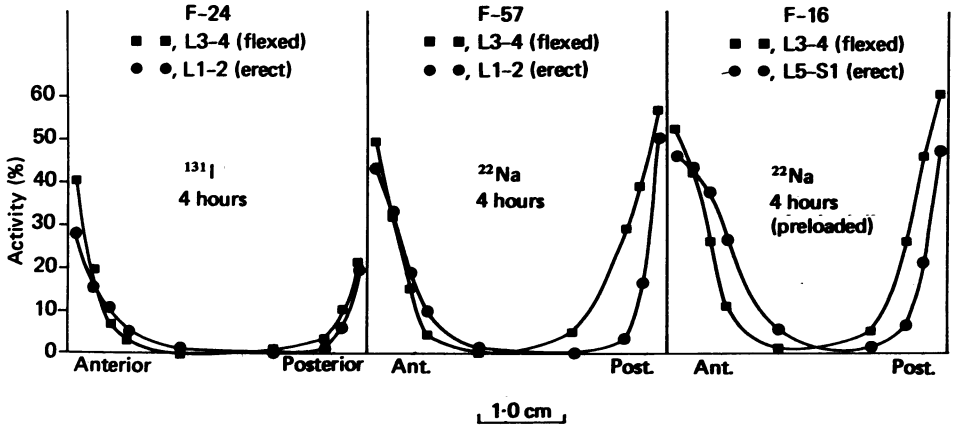

Fig. 3. Activity profiles for three pairs of discs showing how the concentration of radioactive tracer varies along the sagittal section of the discs, after testing. Each 'flexed' disc is compared with an 'erect' disc from the same spine (F-24, female aged 24 years). The tracers used were iodine (^{131}I) and sodium (^{22}Na).

RESULTS

It was convenient to express the concentration of radioactive solute in the disc slices as a percentage of the concentration in the surrounding bath. This percentage could then be termed the 'activity' of the disc slice, given by the equation:

$$\text{Activity } (A) = 100 \times \frac{N - b(c)}{w} \Big/ \frac{N(b) - b(c)}{w(b)},$$

where N = number of gamma rays counted from disc sample in given time; $N(b)$ = number of gamma rays counted from bath sample in same time; $b(c)$ = number of gamma rays counted from background radiation in same time; w = weight of disc sample; $w(b)$ = weight of bath sample.

Table 1 shows the average results for the groups of motion segments tested. Three pairs of typical results are plotted along a sagittal section in Figure 3. These show that the activity falls off rapidly away from the disc periphery. The very low values in the nucleus suggest that the quantity of tracer entering the disc through the vertebral endplate was negligible compared to that entering through the periphery of the annulus.

Activities were lower for the negative ion (^{131}I), presumably because the ion was repelled by the negative fixed charge density of the disc, whereas the positive ion, ^{22}Na, was attracted by the disc.

As expected, the one hour tests showed lower activities. There was considerable scatter in these results for the reasons discussed in Appendix I.

Discs that were not preloaded had lower activities than those that were. The results for the specimens which were not preloaded represent the diffusion that occurs in life during the first few hours of each day when fluid is being expressed from the disc. The preloaded results are applicable to the rest of the day when fluid flow is negligible.

The effect of posture on the penetration of tracer can be gauged from Table 1 but can be assessed more accurately by comparing the activity of each flexed disc with

Table 1. *The average results for the groups of motion segments tested. The numbers given are mean values* ±1 S.D.

Testing conditions					Activity of the disc slices								
Tracer	Duration (hours)	Preload (hours)	Posture	Number of discs	1	2	3	4	5	6	7	8	9
^{131}I	4	—	Erect	4	30·1 ±4·6	14·1 ±1·2	7·4 ±2·5	3·2 ±1·5	0·4 ±0·3	0·2 ±0·2	2·2 ±0·8	6·5 ±1·5	15·9 ±3·4
^{131}I	4	—	Flexed	4	35·9 ±4·8	16·8 ±3·8	6·9 ±1·2	2·5 ±0·8	0·3 ±0·3	0·9 ±0·4	6·4 ±2·8	13·2 ±3·6	25·3 ±8·9
^{22}Na	1	—	Erect	1	17·1	4·6	0·9	0·3	0·2	0·1	2·7	10·8	27·5
^{22}Na	1	—	Flexed	1	33·9	11·5	1·2	0·1	0·0	0·2	2·5	10·5	27·6
^{22}Na	1	3	Erect	2	23·6	5·5	0·8	0·1	0·0	0·1	0·3	2·3	18·2
^{22}Na	1	3	Flexed	2	29·6	11·9	5·2	0·6	0·1	0·1	2·9	12·4	30·8
^{22}Na	4	—	Erect	6	39·5 ±8·8	28·9 ±7·4	16·7 ±5·1	8·0 ±2·9	1·7 ±2·4	0·9 ±0·6	9·9 ±3·9	24·9 ±8·5	48·5 ±9·8
^{22}Na	4	—	Flexed	6	42·4 ±8·8	27·9 ±8·8	12·8 ±4·4	4·6 ±1·5	0·6 ±0·5	3·9 ±2·0	25·9 ±7·0	36·9 ±5·6	48·9 ±7·5
^{22}Na	4	3	Erect	6	54·5 ±6·8	44·4 ±6·7	28·4 ±7·4	16·6 ±7·2	2·5 ±1·8	1·2 ±0·6	10·1 ±4·9	27·6 ±10·7	57·8 ±12·1
^{22}Na	4	3	Flexed	6	59·7 ±18·3	39·8 ±16·1	22·1 ±13·8	7·2 ±4·2	0·5 ±0·3	6·2 ±3·6	30·6 ±10·5	47·9 ±11·4	56·6 ±6·0

Table 2. *The effect of posture on the penetration of tracer into the discs*

For each disc slice (1...9) the tracer activity in the flexed disc was divided by the activity in the paired erect disc, to get the ratio of the activity $r_1 \ldots r_9$. The values shown are the mean values of $r_1 \ldots r_9$ for the groups of specimens tested. The overall mean ratios (±1 S.D.) are shown at the bottom of each column.

			Ratio of activity (flexed/erect)								
Tracer	Preload (hours)	Number of disc pairs	r_1	r_2	r_3	r_4	r_5	r_6	r_7	r_8	r_9
^{131}I	—	4	1·26	1·20	0·89	0·70	0·71	5·12	3·00	2·05	1·70
^{22}Na	—	6	1·13	0·95	0·76	0·59	0·44	4·15	3·86	1·70	1·02
^{22}Na	3	6	1·10	0·92	0·82	0·46	0·22	5·82	3·06	1·72	1·01
Overall mean ratios (n = 16)			1·15* ±0·26	1·00 ±0·26	0·82*** ±0·27	0·57*** ±0·19	0·43*** ±0·35	5·02** ±2·71	3·34** ±2·17	1·80** ±0·46	1·19* ±0·43

* Ratio significantly greater than 1 ($P < 0·05$); ** Ratio significantly greater than 1 ($P < 0·01$); *** Ratio significantly less than 1 ($P < 0·01$).

the activity of the erect disc from the same cadaver spine. The ratios of these activity values were calculated for each disc slice for each pair of discs. For example,

$$r_6 = \frac{\text{activity of slice 6 from a flexed disc}}{\text{activity of slice 6 from the erect disc from the same spine}}.$$

The average values of these ratios for the four hours tests are shown in Table 2.

An analysis of variance revealed that these ratios were not significantly altered by either the tracer used (^{131}I or ^{22}Na) or by the presence of preloading, so the results for the three tests could be pooled. A further analysis of variance then showed that the overall mean ratios varied significantly for different disc slices ($P < 0.0001$). A *t*-test showed that the overall mean ratios were significantly greater than one for slices 1, 6, 7, 8 and 9 and were significantly less than one for slices 3, 4 and 5 ($P < 0.05$ or better). In plain words, flexed discs had significantly more tracer in slices 1, 6, 7, 8 and 9, but less in slices 3, 4 and 5, and this result did not depend on the type of tracer used, or on the presence or absence of preloading.

In order that diffusion into the posterior and anterior parts of the annulus might be compared, the areas under the activity curves (Fig. 3) were measured to a distance of 17 mm from the posterior and anterior borders respectively. The areas were proportional to average solute concentration in the posterior and anterior parts of the disc. These were measured for the four hours ^{22}Na (preloaded) tests and normalised relative to the value for the anterior annulus in erect posture. The mean normalised values are shown in Figure 6. The concentration of solute in erect posture was greater in the anterior disc ($P < 0.01$) but this was reversed in flexed posture ($P < 0.005$).

The values shown in the tables have limited significance since they refer to diffusion for set time periods down artificially induced concentration gradients. In order to generalise the effect of posture on diffusion into the disc it is necessary to use diffusion theory.

Interpretation of results using diffusion theory

In this section, a simple model is proposed to explain how the annulus deforms in flexed and erect posture. Then, diffusion theory is used to calculate the effect this deformation should have on the activity values discussed above. Finally, the model is tested by comparing calculated and experimental values of activity.

Measurements on X-rays of healthy adults (Adams & Hutton, 1982) show that, in full flexion, the posterior annulus is stretched vertically by about 50% and the anterior annulus compressed by about 30% compared to erect standing posture (Fig. 4). The posterior annulus is sufficiently thin and far from the centre of rotation that it can be considered to be stretched uniformly. To maintain constant volume it must therefore be thinned (along an anteroposterior axis) to about 0·67 of its thickness in the erect posture. In fact, fluid flow can slowly decrease the volume so that after several hours the posterior annulus will be about 0·63 of its (anteroposterior) thickness in lordotic posture.* The anterior annulus is thicker and extends almost to the centre of rotation. In flexed posture, it becomes less wedged and bulges markedly, but its deformation cannot be described as simply as in the case of the

* After four hours of flexed posture, the fluid content of the annulus is reduced by 15%, while after four hours of erect posture, it is reduced by only 10% (Adams & Hutton, 1983). Because the annulus is only 80% water, this will cause volume reductions of 12 and 8% respectively, i.e. a relative reduction in flexion of 4%.

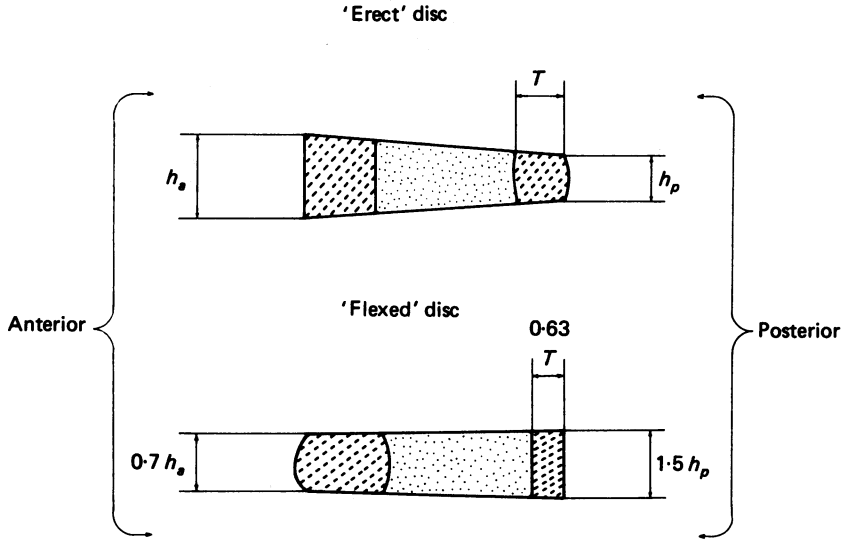

Fig. 4. Typical deformation of a lumbar disc in erect and flexed postures as inferred from X-rays on healthy subjects. T, thickness of posterior annulus; h_a, h_p, heights of anterior and posterior annulus.

posterior annulus. Qualitatively, flexion will stretch and thin the outermost layers (because of bulging) whilse some of its bulk will be displaced posteriorly, increasing its overall thickness.

The large changes in thickness of the posterior annulus can now be used to calculate the effect of posture on the diffusion of tracer into the disc from the surrounding bath.

The activity of a disc slice at a distance x from the posterior border of the disc is given by

$$\text{Activity} = \frac{k}{1+k(D_2/D_1)^{\frac{1}{2}}}\, erfc\, \frac{x}{2(D_2 t)^{\frac{1}{2}}}, \qquad (1)$$

where t = time since diffusion started; k = equilibrium partition coefficient of the tracer between bath and disc; D_1, D_2 = diffusion coefficients of tracer in bath (D_1) and disc (D_2); $erfc(x) = 1-2/\sqrt{\pi}\int_0^x e^{-t^2}\,dt$.

In Appendix II, the use of this equation is justified, and it is shown that for a 'flexed' disc, x must be replaced by

$$x_f = 0{\cdot}68x,$$

and for an 'erect' disc, x must be replaced by

$$x_e = 1{\cdot}08x,$$

to account for the changes in annular thickness from the neutral position.

Figure 5 shows equation 1 evaluated for 'flexed' and 'erect' discs, using values of t, k, D_1 and D_2 appropriate for the four hours ^{22}Na tests at 20 °C. The values used were k = 1·8 and 2·2 (Urban & Maroudas, 1979), $D_1 = 11{\cdot}7\times10^{-6}$ cm^2/s and $D_2 = 4{\cdot}0\times10^{-6}$ cm^2/s (calculated from Urban (1977) using $D_2 = D_1 0{\cdot}63E$ where E = volume fraction of water in the disc = 0·74). The experimental values for the four hours ^{22}Na tests (with preloading) are also shown. The experimental result for slice 9 in the flexed posture is significantly different from the theoretical values

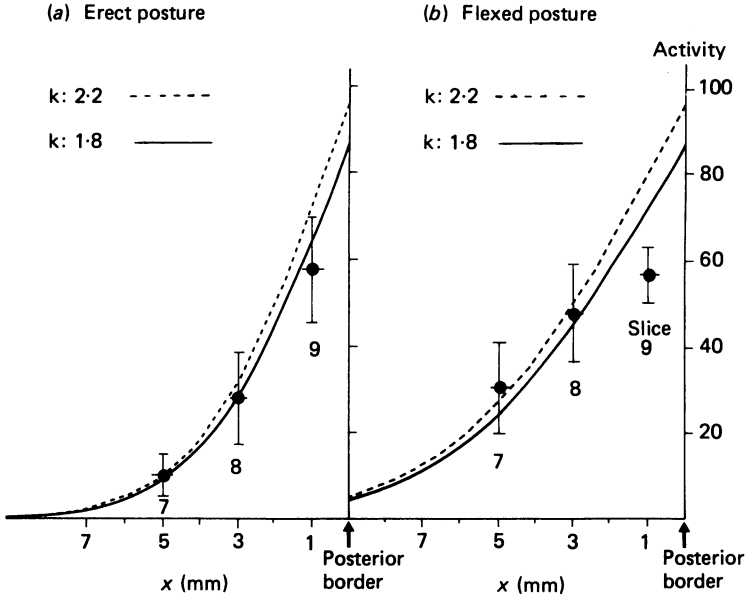

Fig. 5. Tracer activity in the disc falls off with distance (x) from the posterior border. Experimental results Φ (± 1 S.D.) are compared with calculated predictions of the model using diffusion theory and assuming $D_a = 4 \times 10^{-6}$ cm/s. The results shown are the average values for four hours ^{22}Na tests (with preloading), taken from Table 1. k, equilibrium partition coefficient.

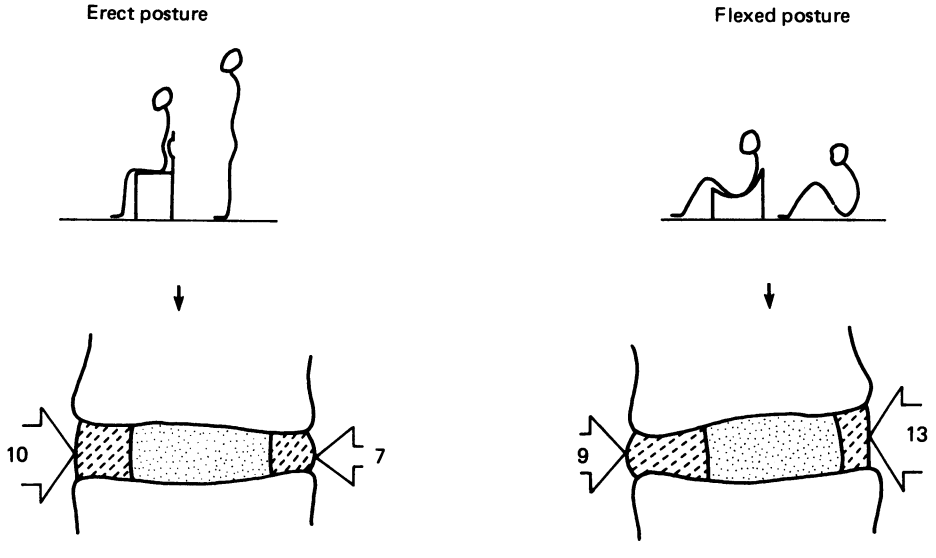

Fig. 6. The effect of posture on diffusion into lumbar discs. The numbers are proportional to the average concentration of ^{22}Na in the anterior and posterior disc, after 4 hours in erect and flexed posture. Note that flexion reverses the imbalance seen in erect posture.

Table 3. *The effect of posture on the penetration of tracer into disc slices* 7, 8 *and* 9: *comparison between experimental results and the predictions of the model*

	Ratio of activity (flexed/erect)		
	r_7	r_8	r_9
Experiment (4 hour ^{22}Na tests with preloading)	3·06 ± 1·21	1·72 ± 0·46	1·01 ± 0·17
Theory ($D_2 = 4 \times 10^{-6}$ cm^2/s)	2·66	1·56	1·11
Theory ($D_2 = 3 \times 10^{-6}$ cm^2/s)	3·46	1·74	1·13

($P < 0{\cdot}05$) but for the other results there is no significant difference between experiment and theory.

A more exacting test of the experiment is to compare the increase in activity caused by flexion (Table 2) with the increase predicted by equations 1–3. This is done in Table 3 for disc slices 7, 8 and 9 for the four hours ^{22}Na tests (with preloading). The theoretical ratios were calculated using $D_2 = 4 \times 10^{-6}$ cm^2/s and $D_2 = 3 \times 10^{-6}$ cm^2/s. This latter value for D_2 is lower than previously reported values for the whole disc, but may be more appropriate for the outer annulus after it has lost 15% of its water content during loading. Differences between experiment and theory are not significant at the 5% level.

DISCUSSION

The main conclusion from these experiments is that erect posture favours diffusion into the anterior half of the disc compared to the posterior half. Flexed posture reverses this imbalance because it stretches and thins the posterior annulus and compresses and thickens the anterior annulus. Can these results be applied to living people?

In principle there should be no difficulty since diffusion is a physical and not a metabolic process. Previous work by Urban, Holm, Maroudas & Nachemson (1977) has shown good correspondence between the measured rate of solute penetration into the discs of living dogs and the predictions based on *in vitro* work and diffusion theory. The calculations of Urban *et al.* assumed that the disc is effectively surrounded by a 'bath' of tissue fluid, as in the present experiment. In practice, experimental error could limit the applicability of the results. However, the experiment has been designed as a 'difference experiment' so that most errors will be randomised and have little effect on the mean values (see Appendix I). It should be acknowledged that posture may affect disc metabolite transport by many other mechanisms which are entirely outside the scope of this experiment. For example, posture may alter the blood supply to the lumbar spine. Such mechanisms are left for others to investigate.

The relevance of the results to different parts of the disc will now be examined in more detail. The nucleus receives most of its nutrients through the vertebral endplates so that changes in the supply through the annulus will not be of great significance. However, the penetration of solute into the posterior nucleus of flexed discs observed here is not negligible and may be important for negatively charged solutes which tend to be discouraged from the endplate route by the high negative fixed charged density of the nucleus. (Urban (1977) has estimated that two thirds of such solutes enter the disc by the annular route.) In life, this increased nutrient supply to the posterior nucleus of a flexed disc by the annular route will be offset by a decrease

in the supply by the endplate route. This is because flexion increases the distance from the endplates to the midplane of the posterior nucleus and so will decrease the nutrient supply by this route. In the case of the anterior annulus, the effect of posture on solute penetration is not great and is unlikely to be significant since, overall, the anterior annulus is better supplied than the posterior annulus (Table 1). This is probably because, except in extreme flexed posture, disc height decreases from front to back, so that solutes diffusing in from the anterior margin are 'funnelled' into a decreasing area. This might explain why degenerative changes are seen less frequently in the anterior annulus. The posterior annulus, in flexed posture, receives much higher solute penetration into its inner portion and clearly, this is the most significant result of this investigation. Calculations have shown that, in erect posture, diffusion of glucose through the posterior annulus can supply cells only to a depth of 4·4 mm (if a cell glycolytic rate of $5{\cdot}2 \times 10^{-11}$ m-mol/cell/hour* is assumed) and that there is a region, 4·4 mm to 6 mm from the posterior border, which has a deficient supply from all sources (Maroudas *et al.* 1975). Now, the present experiment has shown that flexion reduces the thickness of the posterior annulus by 37% on average. This would bring the whole of the 'deficient' region to within 4·4 mm of the surface and ensure a sufficient supply of glucose to all cells in the posterior annulus.

Diffusion into the disc is influenced by posture as discussed above, but it is also influenced by fluid flow. When discs are loaded at body weight, 10–15% of their fluid is expelled in the first few hours (Adams & Hutton, 1983) and this outward fluid flow reduces the inward diffusion of solute in those specimens which have not been preloaded (Table 1). There are two reasons for the reduction. Firstly, solute diffusion within the disc is slowed down because it is moving against the outward flow of fluid. Secondly, fluid expressed from the disc dilutes the bath immediately surrounding it and hence reduces the tracer concentration in the disc. The second effect is an artefact due to the use of an unstirred bath, and so no comment can be made on the scale of the interaction between fluid flow and diffusion within the disc in life. This artefact does not affect the results from preloaded specimens, while its effect on the results from specimens that were not preloaded is to lessen the increase in the tracer concentration in the disc caused by flexed posture.

Returning to the most important result, this experiment has shown that flexed sitting posture increases diffusion of small solutes into the posterior annulus of lumbar intervertebral discs and so redresses the imbalance resulting from erect posture. Flexion also increases the transport of large molecules by the agency of fluid flow (Adams & Hutton, 1983), so there can be little doubt that lumbar disc nutrition is improved by alternating flexed postures with the more usual erect posture. The improvement may be significant since inadequate metabolite transport has been linked with disc degeneration (Holm & Nachemson, 1982; Nachemson, Lewin, Maroudas & Freeman, 1970). This may explain why populations that adopt a flexed sitting posture have a lower incidence of lumbar disc disease (Fahrni & Trueman, 1965).

However, the conclusion that 'flexed sitting posture is best', should not be reached without considering that wedging the lumbar discs in flexion increases the intra-discal pressure by about 50% compared to lordotic posture (calculated from Nachemson, Schultz & Berkson, 1979). This extra pressure may be important if the

* This is the higher of the two rates considered by Maroudas *et al.* (1975). More recent experiments indicate that the other rate was too low (Holm *et al.* 1981).

disc is injured, but is it likely to have serious consequences for a healthy disc, bearing in mind that compressive loads in these postures are only about 10% of the compressive strength of the disc (Hutton & Adams, 1982)?

SUMMARY

The diffusion of small solutes into the intervertebral discs of cadaveric lumbar motion segments was measured using a radioactive tracer technique. The motion segments were wedged and loaded to simulate erect posture and flexed sitting postures. The results show that erect posture favours diffusion into the anterior half of the disc compared to the posterior half. Flexed posture, by deforming the annulus fibrosus, reverses this imbalance.

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APPENDIX I

Experimental error

The greatest source of experimental error was the procedure for cutting the disc into slices. The 'activity' of the slices decreased rapidly away from the disc periphery, so if the outermost slice was cut too thick or too thin, the activity of the next (inner) slice would be too low or too high, respectively. A comparison of the weights of the slices suggested that the standard error in slice thickness was about 0·5 mm,

leading to an error in the 'activity' of about 10% in the case of the four hours tests, and about 30% for the one hour tests. For this reason the latter test was discontinued. A second cutting error arose from the inability to remove all the posterior longitudinal ligament before testing. Any scraps left adhering to the disc surface were included in slice 9 (Fig. 2). This would affect the activity of slice 9 because ligamentous tissue had a lower fixed charged density than the disc and so would have a different attraction for the charged tracer ions. However, the error involved could not have been great because in the four hours preloaded ^{22}Na tests, the activities of slices 1 and 9 were similar.

Error in the measurement of the 'activity' of disc slices was small. A radioactive count of N has a standard error of $N^{-\frac{1}{2}}$, so the lowest counts of about 800 in the present study should have had an error of $\pm 3{\cdot}5\%$. In practice, the detection system was not perfect and repeated counting of the same disc slices in different plastic bottles and in different sequence showed the accuracy of the 'activity' to be, at worst, $\pm 6\%$.

Error was introduced by changes in the water content of the discs. The rate of solute penetration was decreased by any decrease in the water content and the 'flexed' discs would have had about 5% less water in them than the 'erect' discs, after the loading period of four hours (Adams & Hutton, 1983). This would have caused the increase in diffusion into 'flexed' discs to be slightly under-estimated. The water content of all the discs would have been reduced by about 2% by the freezing and thawing cycle (Köller, Funker & Hartmann, 1981), but this would not have influenced the postural effect on diffusion.

In addition to the above sources of error, there were variables that increased the scatter of experimental results. These included variations in the wedging angle used for the 'flexed' discs, and variation in the size, chemical composition and water content of discs from different spines.

Errors arising from the assumptions implicit in diffusion theory are discussed in Appendix II.

APPENDIX II

Equation 1

This equation is for plane wave diffusion from one medium into another in a direction perpendicular to the boundary (Crank, 1975). This corresponded, in the experiment, to the tracer entering the central strip of disc (Fig. 2) via the posterior face and diffusing in towards the nucleus.

The equation assumes that there are no convection currents in either medium. This was a fair approximation as far as the bath was concerned, because it was equilibrated at room temperature before testing and then left undisturbed. However, it applied to the disc only in the preloaded tests where there was little or no fluid flow in the disc during the testing period.

Equation 1 also assumes that the partition and diffusion coefficients are constant across the posterior annulus. This was a considerable oversimplification (Urban & Maroudas, 1979). Also, in a more rigorous treatment, equation 1 would require the activity values to be corrected for the varying amounts of (non-radioactive) Na^+ already present in the disc before testing. The concentration of this Na^+ falls off in the outer annulus and will have the effect of lowering the experimental activity values near the disc periphery, as can be seen in Figure 5. Similarly the distance x in Equation 1 should be replaced with an 'effective distance' that takes account of

varying water content across the annulus. However, elaborating the diffusion theory in this way was considered to be not worth the considerable effort involved, because it would not substantially alter the comparison between 'flexed' and 'erect' discs.

Calculation of x_f and x_e

Let the height and thickness of the posterior annulus of an unstressed disc be y and x respectively, and assume that in flexed and erect postures the disc is deformed as shown in Figure 4.

Now, in these experiments, the average flexion angle used (12°) was four times the extension angle (3°), so it can be assumed that the change in thickness in the flexed posture ($x-0{\cdot}63T$) is four times the change in thickness in the erect posture ($T-x$), so

$$x-0{\cdot}63T = 4(T-x),$$

therefore

$$T = 1{\cdot}08x \quad \text{and} \quad 0{\cdot}63T = 0{\cdot}68x.$$

Therefore, in the erect posture, the thickness of the posterior annulus is effectively $1{\cdot}08x$ and in flexed posture it is $0{\cdot}68x$, where x is the thickness in an unstressed disc.

Hence, in equation 1, x must be replaced by

$$x_e = 1{\cdot}08x, \tag{2}$$

and

$$x_f = 0{\cdot}68x, \tag{3}$$

to account for the penetration of tracer into 'erect' and 'flexed' discs respectively.