Why I Pursue Discogenic Pain*

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***Synopsis of the Case For, in a debate on discogenic pain conducted at the Annual Scientific Meeting of the German Pain Society, held in Bremen on 20th October, 2005;**

and reiterated at the Debate on Discography at the Annual Scientific Meeting of the Australian Association of Musculoskeletal Medicine, Melbourne, November 2008.

The fundamental reason why I pursue the diagnosis of discogenic pain is that patients have no other valid alternative. Patients with chronic back pain get caught in a circus (Figure 1). They are told that there is nothing wrong with them medically; or they are told something fallacious such as: they once did have nociception; but that has now ceased; and now they have only a "memory" of that pain. Under those conditions, medical treatment will not help; and the only prospect of treatment is behavioural and physical rehabilitation. But that treatment does not work. The patients still have pain. Yet again they are told that there is nothing wrong. They failed rehabilitation, and the only recourse is to repeat it.

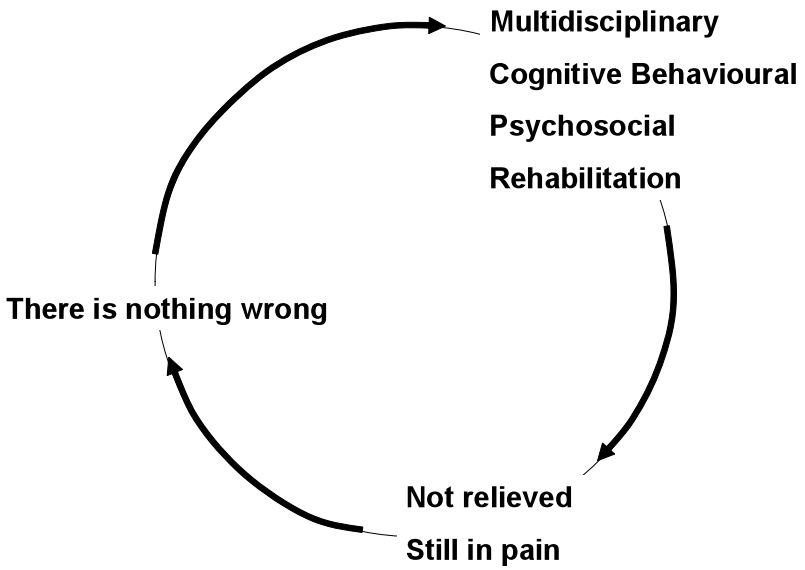

Figure 1. The back pain circus. Patients are told nothing is wrong; they must undergo rehabilitation; but rehabilitation does not work; they still have pain; but are still told that nothing is wrong.

It is politically correct to declare that multidisciplinary pain treatment is not only effective, but is also superior to medical treatment. Yet examining the literature casts doubt on this.

Systematic reviews have found that behavioural therapy may be superior, in some respects, to no treatment but it is not more effective than exercises, and if added to physical rehabilitation it does not improve outcomes [1]. A review of multidisciplinary treatment programs, i.e. functional restoration programs, found evidence that programs with less emphasis on physical domains are NOT effective; the evidence supported only those programs with an emphasis on intensive physical rehabilitation [2]. If one consults the source literature upon which the reputation of multidisciplinary therapy is based, a more sobering impression arises.

Deadorff et al [3] treated 55 patients with physical therapy conditioning, work training, psychological pain management, and operant condition, and compared their outcomes with those of 15 patients who had no treatment. The treatment group achieved an average of 15 points reduction in pain scores, from 64 to 49, at 10-13 months follow-up. But the group who had no treatment also achieved a similar reduction, from 71 to 54. Yet this is held to be a positive study. Moreover, excluded from the treatment group were Medicare and patients who were considered not appropriate fro therapy or who were not motivated. The control group was a convenience sample of patients who were denied payment for therapy by their insurance company.

The use of convenience samples is common in studies of multidisciplinary therapy. The Volvo Award-winning study of Mayer et al [4], which founded functional restoration, used a convenience sample as its control group. Thus, it appears acceptable to use convenience samples when the objective is to validate multidisciplinary therapy. This raises an intriguing comparison when, later, it comes to evaluating the literature on intradiscal therapy (see below).

A Swedish study, compared patients treated with applied relaxation, or applied relaxation combined with operant conditioning, and patients put on a waiting list [5]. In the three groups, pain scores dropped from 4.3 to 4.1, 6.0 to 4.7, and 5.6 to 5.4, respectively. Despite these clinically inconsequential changes and differences the study is considered positive.

A Norwegian study compared the outcomes of 142 patients treated with multimodal cognitive behavioural therapy with those of 81 patients who underwent usual care [6]. In the treatment group, 50% returned to work. Meanwhile, 58% of the usual care group returned to work.

A study by a prominent US proponent of behavioural therapy compared the outcomes of patients put on a waiting list with those treated with behavioural therapy, exercises, or a combination behavioural therapy and exercises [7]. The outcomes of behavioural therapy were not significantly better than those of no treatment (Figure 2). Those patients who had exercise therapy were only slightly more improved than those who were put on a waiting list.

A German study found no difference in pain scores between patients treated with cognitive behavioural therapy and those put on a waiting list [8] (Figure 3). Nor did this study find any differences if scores for depression (Figure 4).

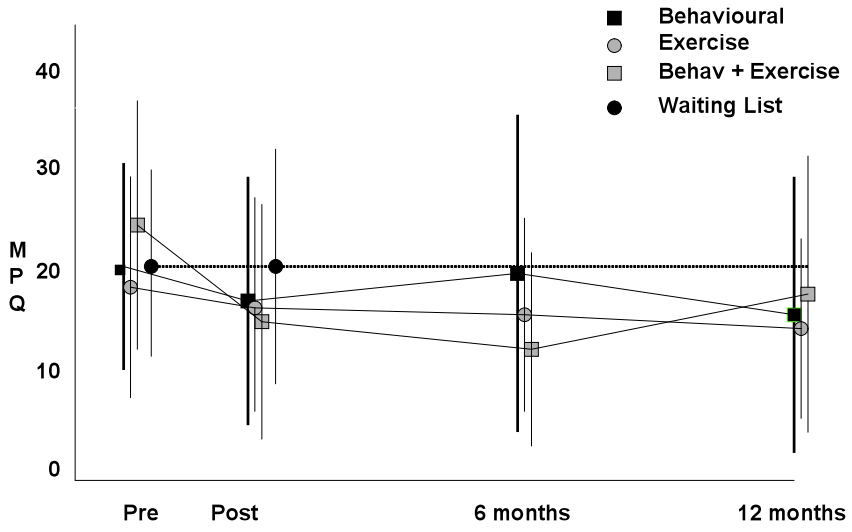

Figure 2. The outcomes of a study of behavioural therapy by Turner et al [7]. MPQ: McGill Pain Questionnaire. The graph shows mean scores and standard deviations.

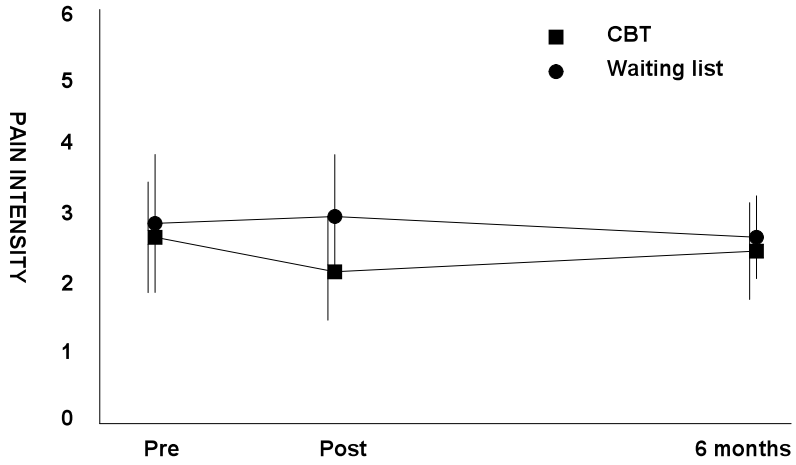

Figure 3. Pain scores in a study that compared cognitive behavioural therapy (CBT) and being put on a waiting list [8]. The graph shows mean scores and standard deviations.

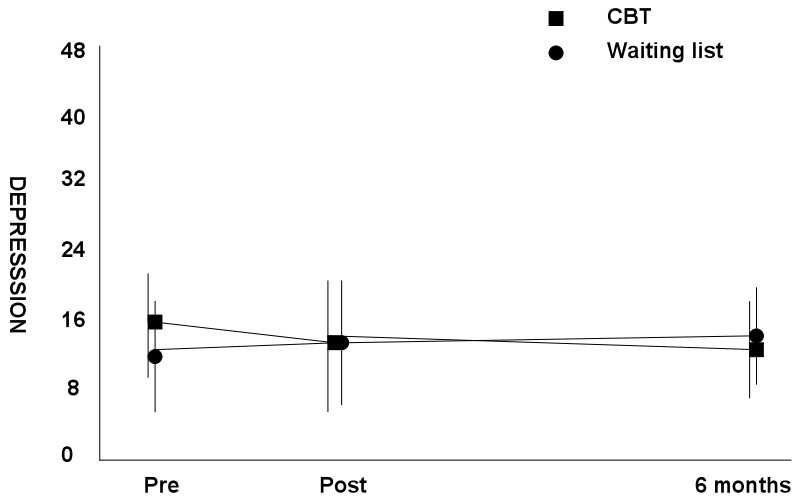

Figure 4. Scores for depression in a study that compared cognitive behavioural therapy (CBT) and being put on a waiting list [8]. The graph shows mean scores and standard deviations.

This pattern was echoed in a seminal British study, in which cognitive behavioural therapy was compared with control intervention amounting to providing patients with attention [9]. No differences were achieved with respect to pain (Figure 5) or depression (Figure 6). Moreover, this study was based on only 9 patients.

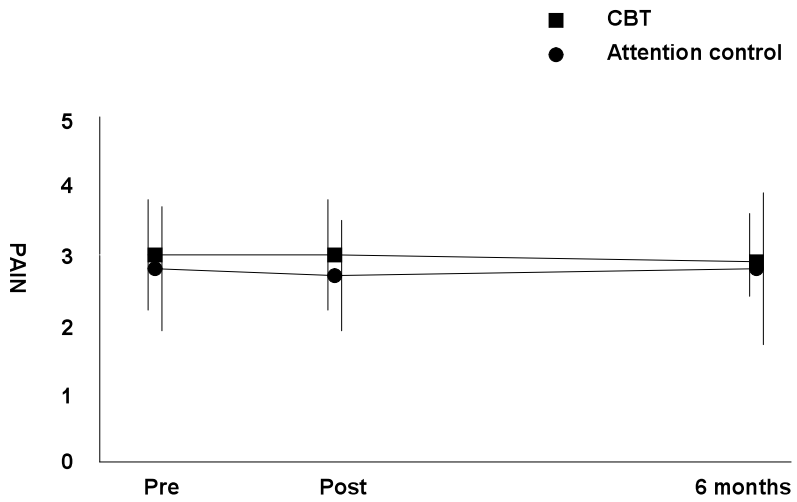

Figure 5. Pain scores in a study that compared cognitive behavioural therapy (CBT) with attention control [9]. The graph shows mean scores and standard deviations.

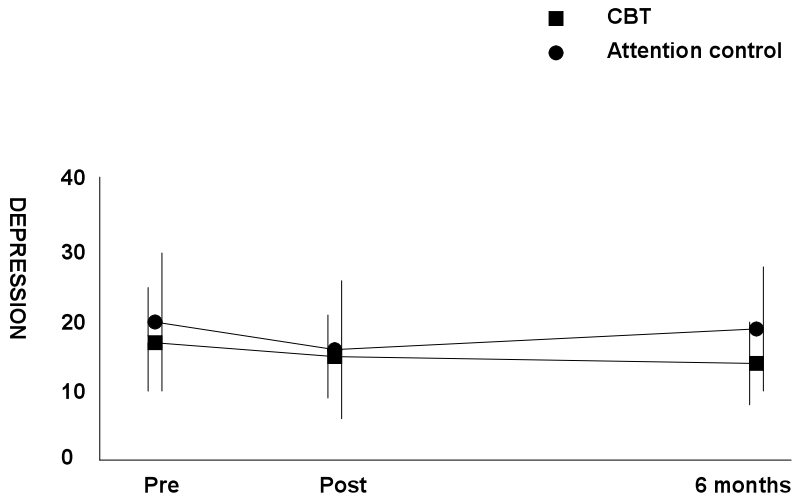

Figure 6. Scores for depression in a study that compared cognitive behavioural therapy (CBT) with attention control [9]. The graph shows mean scores and standard deviations.

Although a review of multidisciplinary functional restoration found that intensive programs do reduce pain and do improve function [2], the source literature reveals the magnitude of these supposedly beneficial effects. Functional disability improves from a score of 15.5 out of 30 to 8,5, and pain decreases from 5.3 to 2.7, at four months follow-up [10]. Yet other studies from the same investigators attest to improvements in disability 16.9 to 12.1, and reductions in pain scores from 6.1 to only 5.7 [11].

Studies such as these indicate that whatever else multidisciplinary and behavioural therapy programs might or might not achieve, they do not succeed in abolishing pain, or even substantially reducing it. Pain persists despite rehabilitation.

It is that pain that I seek to diagnose and treat. The objective is to break the circle (Figure 7). Persistent pain implies a source. Finding a source of pain refutes the accusation that nothing is wrong. Finding a source provides for a legitimate and credible medical diagnosis. That alone can bring about closure: protecting the patient from continuing to pursue a diagnosis in a futile manner, and protecting them from arbitrary applications of treatment that does not match the source and cause of their pain, and which is doomed to failure. As well, the prospect arises of providing a minimally invasive treatment directed accurately at the source of their pain.

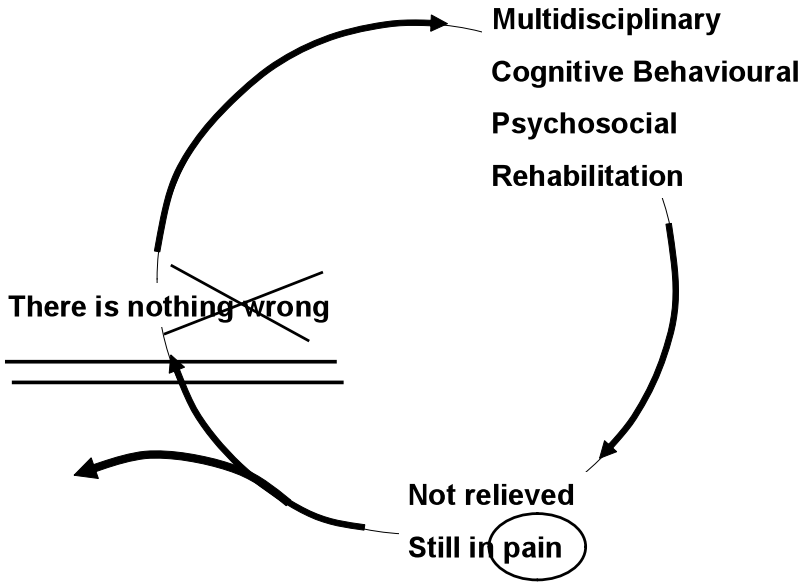

Figure 7. Breaking the back pain circus. Patients with persistent pain are withdrawn from the circus by investigating for its source, and by providing targeted therapy.

INTERNAL DISC DISRUPTION

One of the conditions that I pursue is internal disc disruption. This condition is not disc degeneration. It is a specific condition characterised by degradation of the matrix of the nucleus pulposus and radial fissures that penetrate the anulus fibrosus, but without breaching the outer lamella (Figure 8). The perimeter of the disc is intact. The disruption is totally internal. The fissures may be entirely radial, or a radial fissure may extend circumferentially around the outer anulus. The extent of fissuring may be graded according to if the radial fissure reaches the inner, middle, or outer third of the anulus [12], or if it extends circumferentially [13] (Figure 9).

The morphological features of internal disc disruption cannot be demonstrated by plain radiography or by CT. Even MRI is of limited value (see below). The features can only be shown by post discography CT (Figure 10).

A large study, using multiple regression analysis showed that age changes and degenerative changes did not correlate with the disc being painful [14]. Grade III fissures, however, correlated strongly with pain, and were not related to age changes (Table 1).

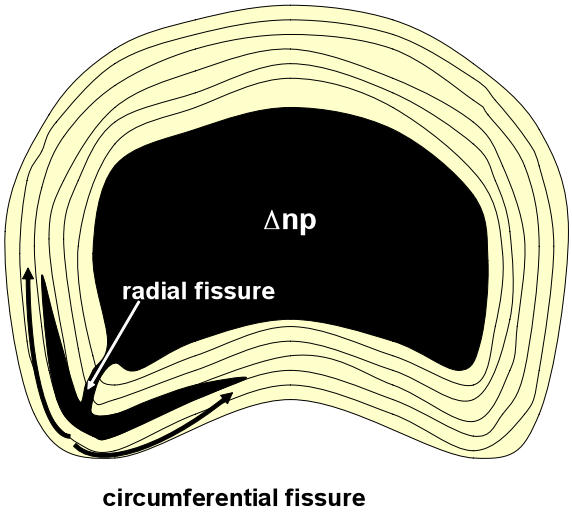

Figure 8. A sketch of a transverse section of a lumbar intervertebral disc, showing the characteristic features of internal disc disruption.

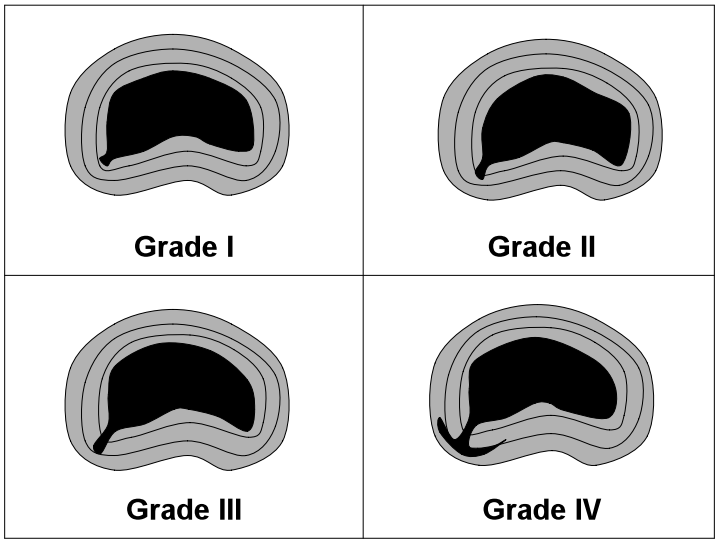

Figure 9. The grading of internal disc disruption according to the extent of fissuring of the anulus fibrosus.

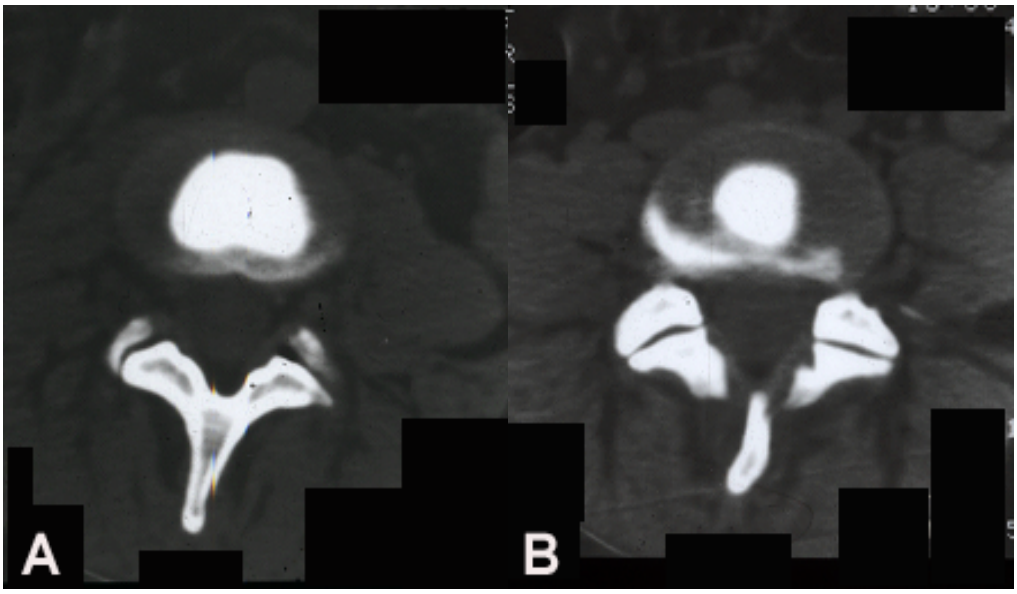

Figure 10. The appearance of discs on CT-discography. **A:** normal disc. The nucleus is rounded and contained within an intact anulus. **B:** Internal disc disruption. A radial fissure at 6 o'clock spreads circumferentially around the anulus.

Pain	Anular Disruption Grade			
Reproduction	III	II	I	0
Exact	43	29	6	4
Similar	32	36	21	8
Dissimilar	9	11	6	2
None	16	24	67	86

Table 1. The correlation between anular disruption and reproduction of pain from the affected disc. Based on Moneta et al [14].

Internal disc disruption also exhibits biophysical features which cannot be faked. Stress profilometry is a technique whereby the internal stresses within a disc, across its diameter, can be measured. Normal disc exhibit a uniform distribution of stress across the anterior anulus, the nucleus pulposus, and the posterior anulus [15] (Figure 11). In discs affected by internal disc disruption, two abnormalities are evident. Within the nucleus, the stresses are irregular and reduced, and may be zero in some

discs, or in some regions of the nucleus (Figure 12). In the posterior anulus, the stresses are raised above normal (Figure 12).

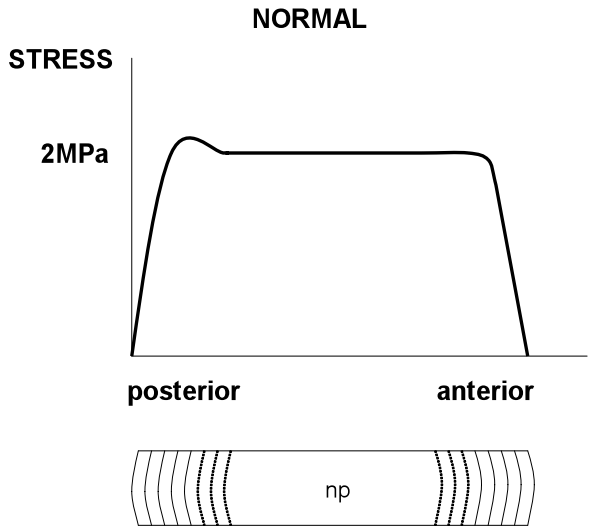

Figure 11. Stress profilometry of a normal disc. The stress is uniform across the anterior anulus, nucleus, and posterior anulus.

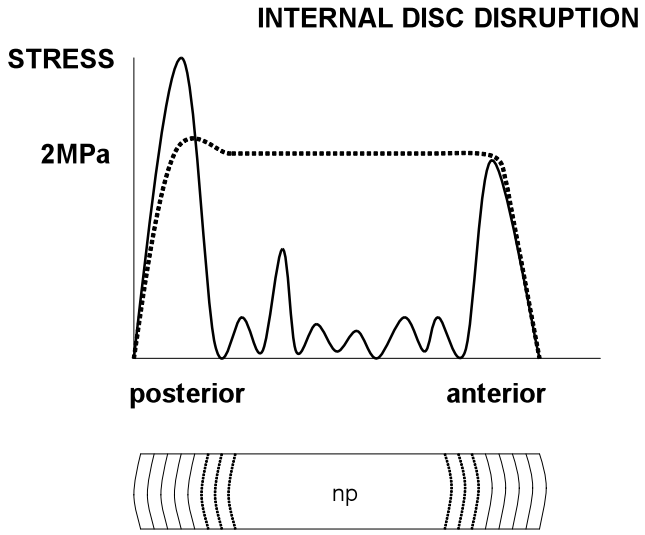

Figure 12. Stress profilometry of internal disc disruption. Nucleus stress is reduced and irregular. Posterior anulus stress is increased.

The depressurisation of the nucleus reflects the degradation of the nuclear matrix, which can no longer retain water efficiently, in order to sustain axial loading. This results in extra load having to be borne by the posterior anulus.

Each of these biophysical features correlates with the disc being painful [16] (Table 2). Discs with increased posterior anulus stress are likely to be painful; discs with normal anulus stress are uncommonly painfully. Discs with a depressurised nucleus are highly likely to be painful; discs with normal nuclear pressure may or may not be painful. Painful discs are likely to exhibit increased anulus stress and a depressurised nucleus. Painless discs will have normal pressure in both the anulus and the nucleus.

	PAIN	NO PAIN	Fisher's exact test
ANULAR STRESS			
Stressed	17	2	
Normal	1	11	p = 0.001
NUCLEAR STRESS			
Depressurised	11	0	
Normal	7	13	P = 0.017

Table 2. The correlation between pain and each of increased anular stress and decreased nuclear stress.

The aetiology of internal disc disruption has been established. Biomechanics experiments have shown that the vertebral endplate is subject to fatigue failure [15]. Subject to loads of 37-50% ultimate tensile strength, endplates can fracture after 2,000 or 1,000 repetitions. Subject to loads of 50-80% ultimate tensile strength, they can fail after as a few as 100 cycles [17,18]. Such loads and repetitions are well within the ranges encountered during moderately heavy work activities.

When subjected to repeated compression loading, discs exhibit mechanical failure. If examined morphologically the failure coincides with the presence of an endplate fracture. Furthermore, upon fracture of the endplate the disc exhibits the onset of the biophysical features of internal disc disruption: the nucleus is depressurised and posterior anulus stress abruptly increases (Figure 13).

The biochemical features of internal disc disruption have also been induced in live animal models [19]. Experimental fracture of an endplate causes de-aggregation of proteoglycans in the nucleus, a reduction in water content, and depressurisation of the nucleus, as well as delamination of the anulus.

Internal disc disruption is the most comprehensively understood cause of low back pain (Figure 14). The condition is characterised morphological by a degraded nuclear matrix and radial fissures through the anulus. These morphological features correlate with the disc being painful. Affected discs exhibit specific biophysical features. These, too, correlate with the disc being painful. The mechanical aetiology of internal disc disruption is fatigue failure of the endplate, which precipitates the biophysical features of the condition. The biochemical features have been produced by endplate fractures in animal models.

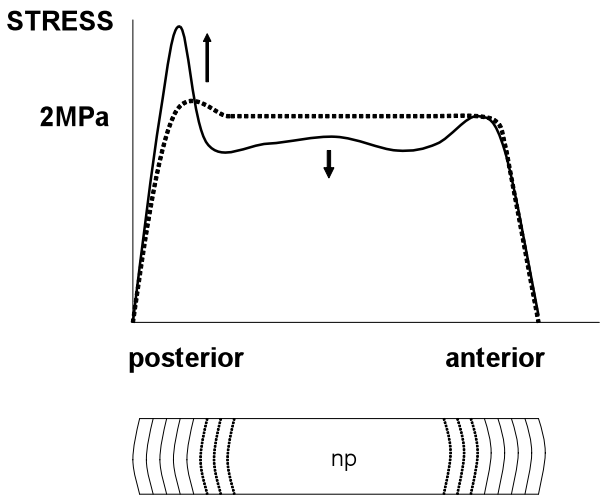

Figure 13. Stress profilometry of a disc immediately after the onset of a fatigue fracture of its vertebral endplate. The nucleus is depressurised and the posterior anulus stress increased markedly.

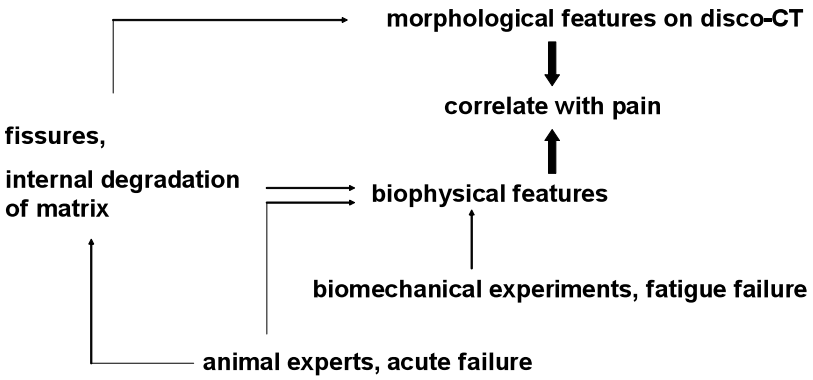

Figure 14. A synopsis of the correlates of internal disc disruption.

Clinical studies have determined that internal disc disruption is the basis for pain in as many as 40% of patients with chronic low back pain [20]. This estimate of prevalence is a worst-case estimate. It excluded two-level disease. The prevalence of internal disc disruption may be considerably higher than 40%; but 40% itself amounts to a

considerable proportion of patients in whom a patho-anatomic diagnosis can be established.

DIAGNOSIS

The diagnostic criteria for internal disc disruption [21] are:

- reproduction of the patient's pain by stimulation of the affected disc (Figures 15 and 16),
- such that the evoked pain has an intensity of at least 7 on a 10-point scale, and
- pain is reproduced at a low pressure of stimulation: 15 psi (1 kg cm $^{-2}$),
- provided that of adjacent discs does not reproduce pain, and
- post-discography CT demonstrates a grade III or IV fissure (Figure 17).

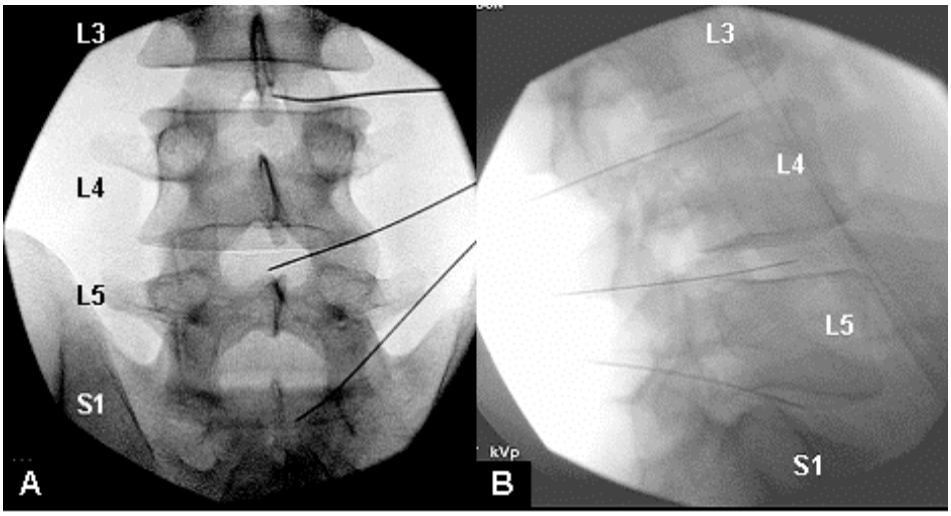

Figure 15. Placement of needles into the three lower lumbar discs, prior to disc stimulation. Reproduced from the ISIS guidelines [21].

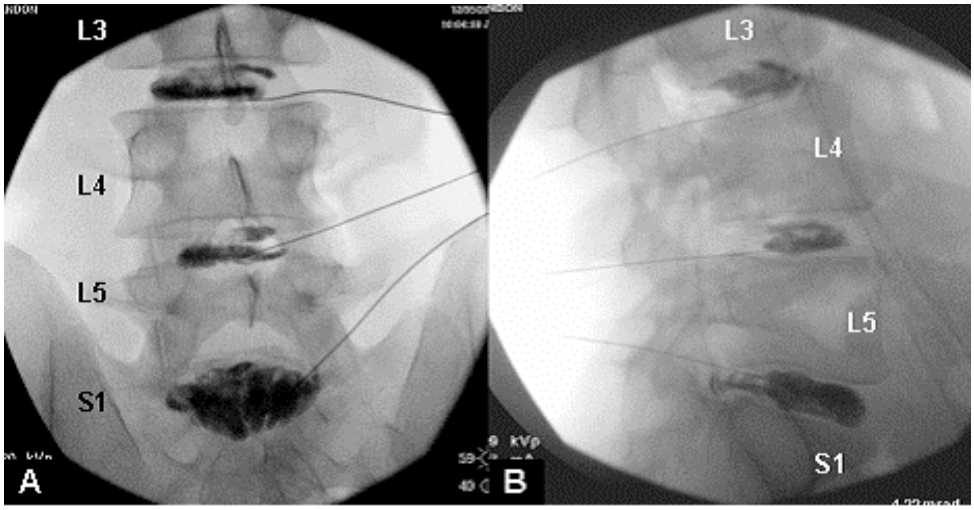

Figure 16. The appearance of the three lower lumbar discs, after injection of contrast medium into the nucleus. Reproduced from the ISIS guidelines [21].

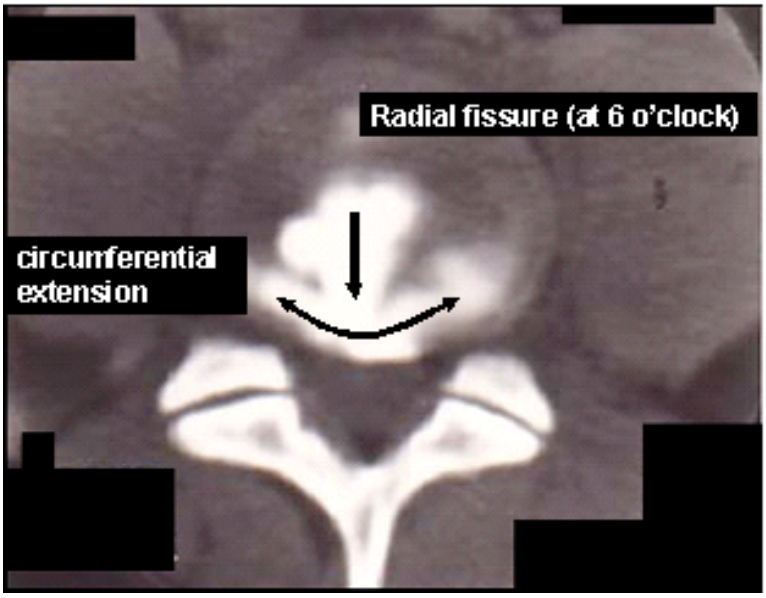

Figure 17. The diagnostic features of internal disc disruption on CT-discography.

The guidelines of the International Spine Intervention Society [21] provide instruments to assist practitioners in the conduct of lumbar disc stimulation. One indicates the information that should be obtained at the time of disc stimulation (Appendix 1). The other provides a scoring system by which to determine if a patient's response is positive or not (Appendix 2). I use these both to establish a record of the procedure and its interpretation, and to ensure that my interpretations remain consistent and, therefore, reliable.

CONTROVERSY

Some investigators have warned that disc stimulation may produce false-positive responses. They based this warning on the responses to disc stimulation of sets of patients who had no symptoms, who had chronic pain but not back pain, and who had been diagnosed as having a somatization disorder [22,23]. Explicitly they imputed false-positive rates of 10%, 20%, and 75% in these groups respectively. However, those percentages were based on sample sizes of only 10, 10, and 4 patients respectively (Table 3). These small sample sizes result in wide confidence intervals of the estimated rates, which renders them poorly representative. Other considerations modify the estimates as well.

The cited estimates did not adhere to the recommended criteria for disc stimulation. They were not subject to anatomic controls, which require that adjacent levels be not painful. They were not subject to manometric criteria.

Category of Subject	Imputed False-Positive Rate	95% confidence intervals
Asymptomatic	1 / 10 = 10%	0% - 29%
Chronic pain	4 / 10 = 40%	10% - 70%
Somatization	3 / 4 = 75%	33% - 100%

Table 3. The imputed false-positive rate of disc stimulation in three categories of subjects, based on Carragee et al [22].

If the original data are analysed, and if the criterion for anatomic controls are applied, the imputed false-positive rate in asymptomatic subjects remains 10%, but that for patients with chronic pain reduces to 20%. The rate for subjects with somatization remains 75% (Table 4). The confidence intervals remain wide.

Category of Subject	Imputed False-Positive Rate	95% confidence intervals
Asymptomatic	1 / 10 = 10%	0% - 29%
Chronic pain	2 / 10 = 20%	0% - 45%
Somatization	3 / 4 = 75%	33% - 100%

Table 4. The imputed false-positive rate of disc stimulation in three categories of subjects, if the criterion for anatomic controls is applied.

Manometric criteria are essential for disc stimulation, for it is a provocation test. In principle, any disc, even a totally normal one, might painful if it is stressed strongly enough. The pressure limits beyond which disc should not be stimulated can be derived from data on normal volunteers. Such data exist [24].

If asymptomatic volunteers, or volunteers who have experienced back pain only occasionally, underdo disc stimulation, a pattern of responses emerges. In some subjects, some discs are not painful even if the disc is stressed to 100 psi (6 kg cm $^{-2}$). Otherwise, however, there is a two-fold trend. The chances that a disc is painful increase as the pressure of stimulation is increased, but if the disc is painful the intensity of pain tends to be low; the pain is unlikely to be severe (Table 5).

Across such data a boundary can be identified: at pressures below which pain does not occur in normal volunteers, or at which the intensity of pain does not exceed certain prescribed values (Table 5). For example the chances are effectively zero that a

normal volunteer will perceive pain if their discs are stimulated up to a pressure of 20 psi. Alternatively, the chances are zero that they will perceive pain of intensity 6/10 or greater if their discs are stimulated up to a pressure of 70 psi.

These data vindicate previously invoked, ad hoc, operational criteria [25]. At pressure of injection up to 50 psi normal subjects should be very unlikely to experience pain whose intensity exceeds 6/10. Up to 15 psi, no normal subject should experience any pain. Applying these manometric criteria reduces the imputed false-positive rate of disc stimulation.

If the criterion of 50 psi is applied, the false positive rates in asymptomatic subjects and in subjects with chronic pain fall to 10% (Table 6), which are clinically tolerable levels. If the criterion of 15 psi is applied, the false-positive rates become zero in asymptomatic subjects and in subjects with chronic pain. In patients with somatization they fall to 25% (Table 7).

PRESSURE (psi)		VAS 0	1	2	3	4	5	6
100	Occ	0.30	0.40	0.25	0.25	0.25	0.10	0.00
	No	0.17	0.48	0.30	0.22	0.09	0.04	0.04
90	Occ	0.35	0.40	0.25	0.25	0.25	0.10	0.00
	No	0.22	0.43	0.30	0.22	0.09	0.04	0.04
80	Occ	0.55	0.30	0.25	0.25	0.25	0.10	0.00
	No	0.22	0.43	0.30	0.22	0.09	0.04	0.04
70	Occ	0.55	0.30	0.25	0.25	0.25	0.10	**0.00**
	No	0.52	0.30	0.17	0.13	0.04	0.00	**0.00**
60	Occ	0.65	0.30	0.25	0.25	0.25	0.10	**0.00**
	No	0.65	0.30	0.17	0.12	0.04	0.00	**0.00**
50	Occ	0.75	0.20	0.15	0.15	0.15	0.05	**0.00**
	No	0.83	0.17	0.09	0.06	0.04	0.00	**0.00**
40	Occ	0.80	0.15	0.10	0.10	0.10	**0.00**	**0.00**
	No	0.96	0.04	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
30	Occ	0.95	0.05	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
	No	1.00	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
20	Occ	1.00	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
	No	1.00	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**

Table 5. The responses to disc stimulation of subjects with no history of back pain (No) and subjects with a history of occasional back pain only (Occ), according to the pressure of stimulation and the intensity of pain evoked. The tabulated figures are the cumulative frequency of responses, which reflect the chances of pain of a particular intensity occurring at a particular pressure of injection. The line indicates the boundary below which normal volunteers do not experience pain. From Derby et al [24].

Category of Subject	Imputed False-Positive Rate	95% confidence intervals
Asymptomatic	1 / 10 = 10%	0% - 29%
Chronic pain	1 / 10 = 10%	0% - 29%
Somatization	2 / 4 = 50%	1% - 99%

Table 6. The imputed false-positive rate of disc stimulation in three categories of subjects, if the criterion for anatomic controls is applied together with the manometric criterion of 50 psi.

Category of Subject	Imputed False-Positive Rate	95% confidence intervals
Asymptomatic	0 / 10 = 10%	0% - 28%
Chronic pain	0 / 10 = 10%	0% - 28%
Somatization	1 / 4 = 25%	0% - 69%

Table 7. The imputed false-positive rate of disc stimulation in three categories of subjects, if the criterion for anatomic controls is applied together with the manometric criterion of 15 psi.

These considerations indicate that the threat of false-positive responses to disc stimulation have been exaggerated. In asymptomatic individuals and in patients with chronic pain, the imputed false-positive rate is effectively zero, provided that the stringent operational criteria for disc stimulation are satisfied. Only in patients with somatisation might concern about false-positive responses be justified. What the false-positive rate might be in such patients is not clearly evident, because of the small sample size that has been studied; but it does appear to be non-zero.

IMAGING

Certain features, evident on MRI increase the likelihood that the affected disc has internal disc disruption and is painful. They are Modic lesions and high-intensity zones.

Modic type I lesions occur in the spongiosa of the vertebral bodies adjacent to the affected disc. They appear as a high-intensity signal on T2-wieghted images. They indicated oedema of the spongiosa. Modic type II lesions appear as a high intensity signal in the spongiosa on T1-weighted images. They reflect fatty infiltration of the vertebrae. These lesions have a strong correlation with the disc being painful on stimulation (Table 8). The low sensitivity reflects the fact that not all patients with

discogenic pain exhibit these features. The high specificity, however, indicates that when Modic changes are present they are nearly always associated with a painful disc.

Sensitivity	Specificity	Likelihood Ratio	Reference
0.23	0.97	7.7	26
0.22	0.95	4.4	27

Table 8. The strength of relationships between Modic changes and discogenic pain.

High intensity zones (HIZ) are a very bright signal contained within the posterior anulus fibrosus, as seen in sagittal sections on MRI. They are sagittal sections of circumferential fissures (Figure 18). Not all fissures or grey spots on an MRI constitute an HIZ, however (Figure 19). To constitute an HIZ, the zone must have a very bright signal on heavily T2-weighted scans; the brightness should rival or exceed that of the cerebrospinal fluid.

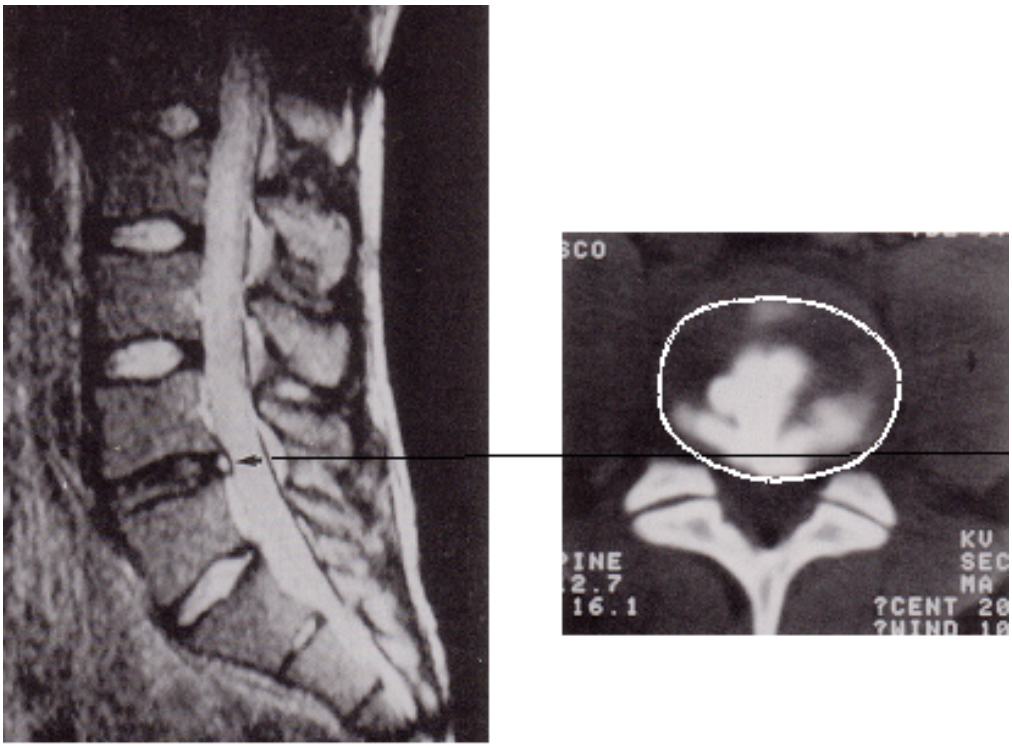

Figure 18. The anatomy of high intensity zones (HIZ). The HIZ seen on sagittal MRI of an L4 disc (arrowhead) constitutes a sagittal section of the transversely widest length of a circumferential fissure, as shown in the CT discogram.

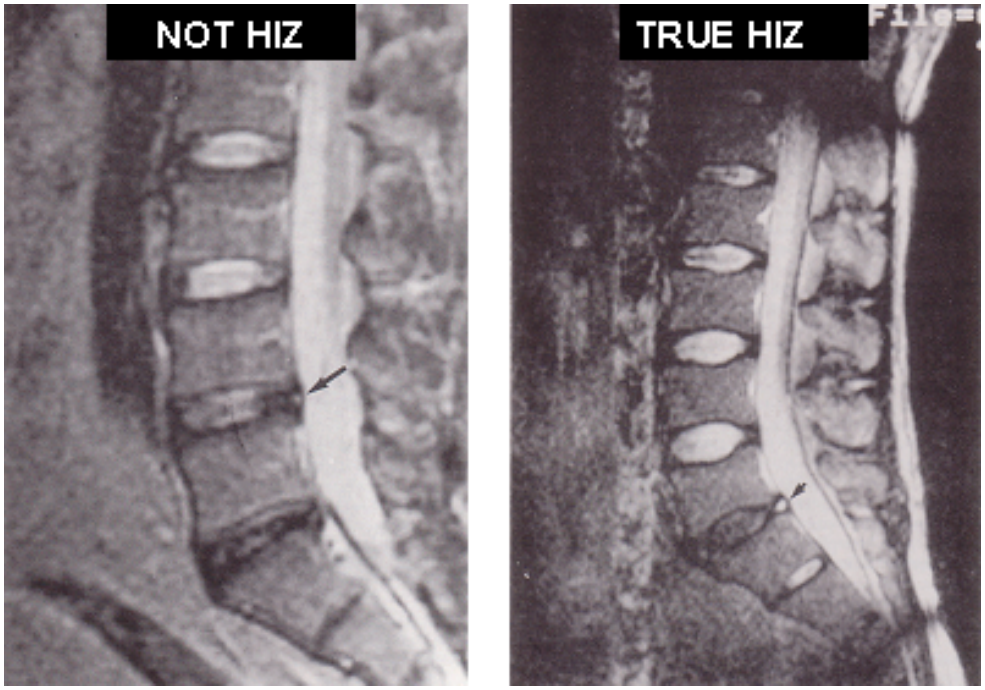

Figure 19. Not all spots in an anulus fibrosus constitute an HIZ. Grey spots may represent a fissure in the anulus, but they are not high intensity signals. In an HIZ the signal intensity exceeds that of the CSF.

The original study of HIZs found that their presence in patients with low back pain correlated strongly with the affected disc being painful on disc stimulation [13]. In this regard it is important to appreciate what was demonstrated. The study did not state that HIZs distinguished subjects with pain from those without pain. Explicitly it found that if present *in patients with back pain* the HIZ strongly implicated that disc as the source of the patient's pain. The correlation was not absolute, but was nonetheless very strong. An HIZ does not prove that the disc is definitely the source of pain, but it increases the odds that the disc is the source of pain by a factor of 6.5.

Several studies have reinvestigated this association. Although the specific statistical variables differ, the same pattern recurs (Table 9). HIZs do not occur in all patients. This is reflected by the low sensitivity of the sign as a predictor of pain. However, all studies, including the one detracting study [31], consistently show high specificity. That feature indicates a double negative: that if present, it is very uncommon for an HIZ to occur in a disc that is not painful. This results in a high positive likelihood ratio: that the presence of an HIZ strongly implies that the affected disc is the source of pain. A likelihood ratio of 5 increases the likelihood that internal disc disruption is the cause of pain from a pre-test probability of 0.4 to a post-test probability of 0.77. Even a likelihood ration of 3 provides a post-test probability of 0.67.

Sensitivity	Specificity	Likelihood Ratio	Reference
0.71	0.89	6.5	13
0.52	0.90	5.2	27
0.27	0.95	5.4	28
0.78	0.74	3.0	29
0.31	0.90	3.1	30
0.09	0.93	1.3	31

Table 9. The strength of relationships between a high intensity zone and discogenic pain.

Some investigators [32] have ventured to discredit the HIZ. They claimed that the sign was not diagnostic because HIZs occur in subjects without back pain. However, their data nevertheless indicate that HIZs significantly correlate with pain (Table 10). HIZs occur nearly three times more frequently in patients with pain than in subjects with no pain. The 95% confidence intervals of the respective proportions do not overlap (Table 10). If the subject was less controversial and emotional such a statistical difference would be considered incontrovertible.

Furthermore, the criticism of HIZ is misdirected. The HIZ was never advocated as a sign of pain. It is a sign in patients with back pain that the affected disc is the source of pain. In this regard, even the disparaging study [32] provides data to this effect. The sign has a high specificity and reasonable likelihood ratio (Table 11).

	Asymptomatic	Symptomatic
HIZ Present	13	25
HIZ Absent	41	17
Prevalence	0.24	0.60
95% CI	0.13 – 0.35	0.45 – 0.75

Table 10. The prevalence of high intensity zones (HIZ) in samples of asymptomatic and symptomatic subjects, based on Carragee et al [32].

HIZ	Disc	
	Painful	**Not Painful**
Present	24	9
Absent	29	47
Sensitivity: 0.45 Specificity: 0.84 Likelihood ratio: 2.8		

Table 11. The strength of relationships between high intensity zone lesions and disc pain, in the study of Carragee et al [32]

Notwithstanding these arguments concerning MRI, detecting an HIZ does not provide for a final diagnosis. Its presence renders it more likely than not that the affected disc is the source of pain. For conservative purposes, this level of confidence may be enough. However, if target-specific therapy is to be undertaken, the putative diagnosis needs to be confirmed by disc stimulation.

TREATMENT

There is no evidence that any form of conservative therapy is effective for proven internal disc disruption. No study of exercise, physical therapy, drugs, or other non-invasive intervention has been performed in patients in whom a diagnosis of internal disc disruption has been established. Nor have any controlled studies been reported of surgery for internal disc disruption.

The only intervention that has been studied is minimally invasive intradiscal therapy in the form of intradiscal electrothermal therapy (IDET). The procedure involves threading a flexible electrode into the painful disc, and using it to heat and coagulate the posterior anulus in the region affected by radial and circumferential fissures [33] (Figure 20). The outcomes of this treatment are limited but nevertheless encouraging.

One study compared IDET with rehabilitation [34]. Both groups of patients commenced with similar pain scores. After treatment and at follow-up 12 months and two years after treatment those scores were significantly better in those patients treated with IDET (Table 12). Cumulative proportions showed that more patients treated with IDET achieved large reductions in pain, such that the number needed to treat for an outcome of complete reduction in pain was 5; for 50% reduction the number needed to treat was 3 (Table 13). When composite criteria were applied, 54% of patients treated with IDET achieved at least 50% reduction of pain with return to work and no need for opioids, compared to only 10% of patients treated with rehabilitation (Table 14).

This study has been criticised because it was not randomised, and instead used a convenience sample of patients whose insurers denied treatment. It is ironic, if not

hypocritical, that this same criticism is not levelled at studies of multidisciplinary therapy which used the very same procedure.

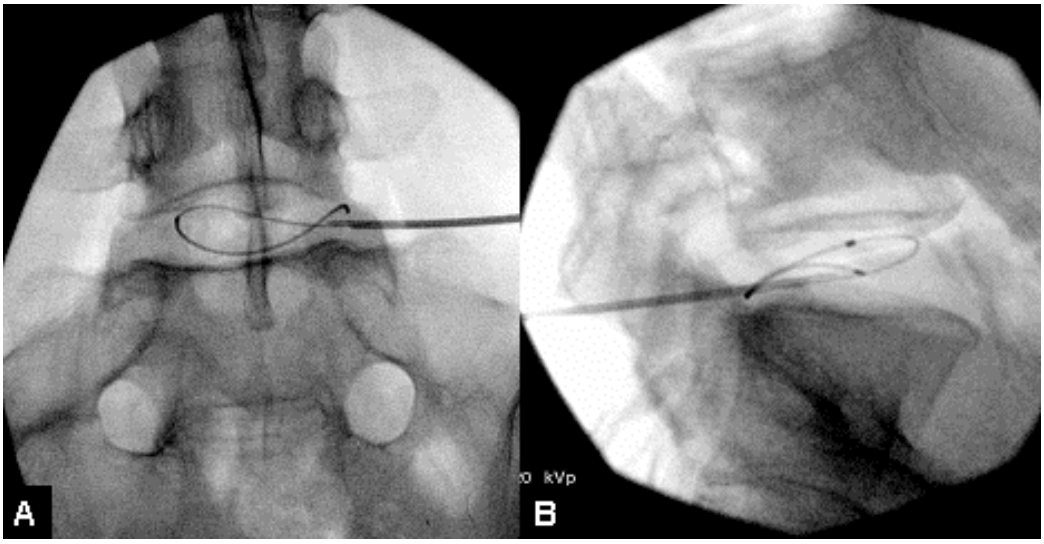

Figure 20. An electrode introduced into an L5-S1 intervertebral disc for the conduct of intradiscal electrothermal therapy. Reproduced from the ISIS guidelines for electrothermal therapy [33].

VAS for Pain	Rehab	IDET	P value
Inception	8 (5-8)	8 (7-9)	0.07
3 Months	8 (7-8)	3.5 (1-5)	0.00
6 Months		3 (1-6)	
12 Months	7.5 (5-8)	3 (1-7)	0.01
24 Months	7.5 (4-8)	3 (1-7)	0.03

Table 12. Median pain scores and interquartile ranges from a study that compared the rehabilitation and intradiscal electrothermal therapy (IDET) for internal disc disruption [34].

ΔVAS	Number		Cumulative Proportion		
	IDET	Rehab	IDET	Rehab	NNT
100	7		0.20	0.00	5
90	0		0.20	0.00	5
80	3		0.29	0.00	5
70	3	1	0.37	0.11	4
60	2	0	0.49	0.11	4
50	5	1	0.57	0.22	3
40	0	0	0.57	0.22	
30	4	0	0.69	0.22	
20	2	3	0.74	0.56	
10	2	1	0.80	0.67	
0	7	1	1.00	0.78	
Worse	0	2		1.00	

Table 13. The number of subjects and the cumulative proportion of subjects who achieved selected percentage improvements in pain scores (ΔVAS) after two-years follow-up in a study that compared rehabilitation and intradiscal electrothermal therapy (IDET) for internal disc disruption [34].

OUTCOME	TREATMENT GROUP	
	IDET	Rehab
50% reduction of pain + RTW + no opioids	0.54	0.10
100% reduction of pain + RTW + no opioids	0.20	0.00

Table 14. The proportion of subjects who achieved the composite outcomes indicated, at two-years follow-up, in a study that compared rehabilitation and intradiscal electrothermal therapy (IDET) for internal disc disruption [34]. RTW: return to work.

OUTCOME	TREATMENT GROUP			
	IDET		SHAM	
	n		n	
ΔPain (0-100)				
worse	2	6%	8	33%
same	5	16%	5	21%
better <20	7	22%	2	8%
better > 20	18	56%	9	38%
	P = 0.037			
ΔPain (%)				
<0	2	6%	8	33%
0-24	11	34%	6	23%
25-49	6	22%	2	8%
50-74	5	16%	7	29%
75-99	5	13%	9	0%
100	3	9%	1	4%
	P = 0.027			

Table 15. The number and proportion of patients who achieved selected absolute and percentage changes in pain scores, at six months follow-up, in a study that compared intradiscal electrothermal therapy (IDET) with sham therapy for internal disc disruption [35].

A placebo-controlled study [35] warned that placebo-responses could occur in patients undergoing intradiscal therapy. However, IDET was significantly more effective than placebo for the reduction of pain (Table 15) and for the improved of physical function in disabled patients.

Both of these studies, however, show that IDET is an incomplete treatment. Some 50% of patients do not benefit at all. Other, observational studies show variable success rates (see Bogduk et al [36] for review).

Among the reasons for variable success rates are differences in patient selection and technique used [36,37]. When originally described, the procedure required placement of the electrode at the interface between the nucleus and inner anulus (Figure 21). Those studies with better outcomes placed the electrode in the outer anulus. The optimum position requires crossing the radial fissure and lying parallel but peripheral to any circumferential fissure (Figure 22). If such a peripheral placement cannot be achieved, a more central placement, inside the circumferential fissure but nevertheless parallel and as close as possible to it, is preferred (Figure 23). If the radial fissure cannot be

crossed using a single insertion of the electrode, the fissures are addressed by bilateral placements (Figure 24).

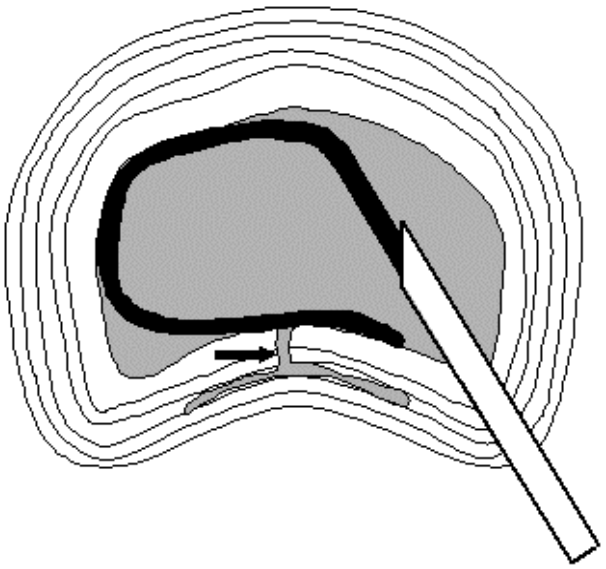

Figure 21. The recommended placement of an electrode for intradiscal electrothermal therapy, as originally described [38].

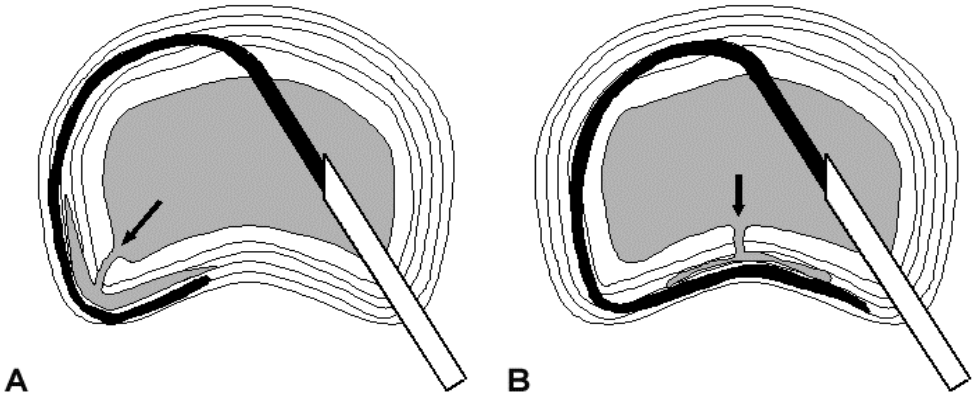

Figure 22. Suggested, optimal placement of electrodes for intradiscal electrothermal therapy. The electrode crosses the radius of a radial fissure and lies parallel but peripheral to the circumferential fissure. **A:** for a radial fissure at between 7 o'clock and 8 o'clock. **B:** for a radial fissure at 6 o'clock.

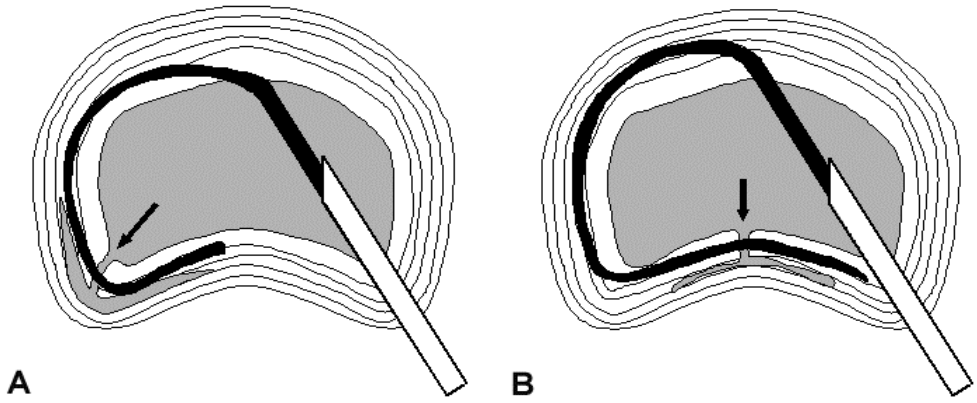

Figure 23. Alternative placement of electrodes for intradiscal electrothermal therapy. If the electrode cannot be placed peripheral to the circumferential fissure, it should be placed across the radial fissure and parallel to the circumferential fissure but internal to it. **A:** for a radial fissure at between 7 o'clock and 8 o'clock. **B:** for a radial fissure at 6 o'clock.

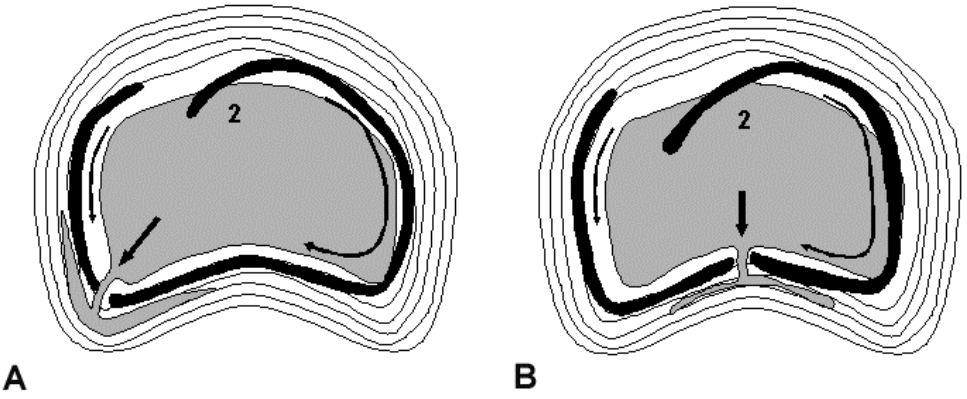

Figure 24. If the radial fissure cannot be crossed using a single insertion, bilateral placements are required to address the entire length of a circumferential fissure. **A:** for a radial fissure at between 7 o'clock and 8 o'clock. **B:** for a radial fissure at 6 o'clock.

These various consideration, however, address only two of the three dimensions of possible technical limitations for IDET. They address how far out and how far across the electrode is placed. They do not address how high or low the electrode is placed in the disc. The latter has been raised as a basis for incomplete effects of treatment [36].

The IDET electrode has only a small field of influence. It coagulates tissues in a region within about one electrode width of the electrode. For some fissures, this field of influence might be enough, i.e. the electrode crosses the fissure and completely coagulates it. In other cases this might not occur. The electrode might pass only

partially through a fissure, or may pass entirely below or above the fissure (Figure 25A). In those instances coagulation, and hence the therapeutic effect, will be incomplete or nil.

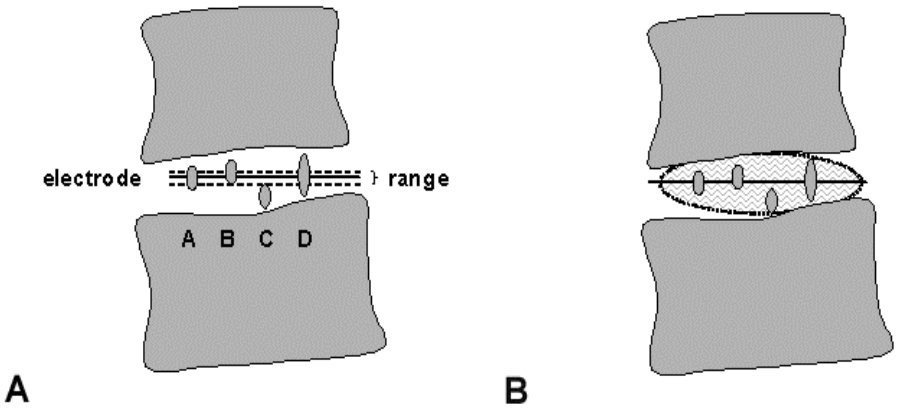

Figure 25. Considerations of the height of placement of the electrode in intradiscal electrothermal therapy. **A:** The dotted lines indicate the effective range of an electrode. If the electrode passes through a fissure it will coagulate the fissure, but the electrode may pass through only part of a fissure, or pass entirely below or above a fissure. In which case the electrode will fail to coagulate the fissure completely. **B:** What is required is an electrode whose lesion encompasses the range of possible heights of fissures.

For this limitation to be overcome an electrode is required that produces a lesion that encompasses all the possible heights of fissures (Figure 25B). To this end, an emerging technology is cold radiofrequency. This technology uses bipolar electrodes. If electrodes are inserted into each posterior corner of the target disc, a lesion is made that spans between them across the entire height of the disc (Figure 26). This technology is currently being evaluated.

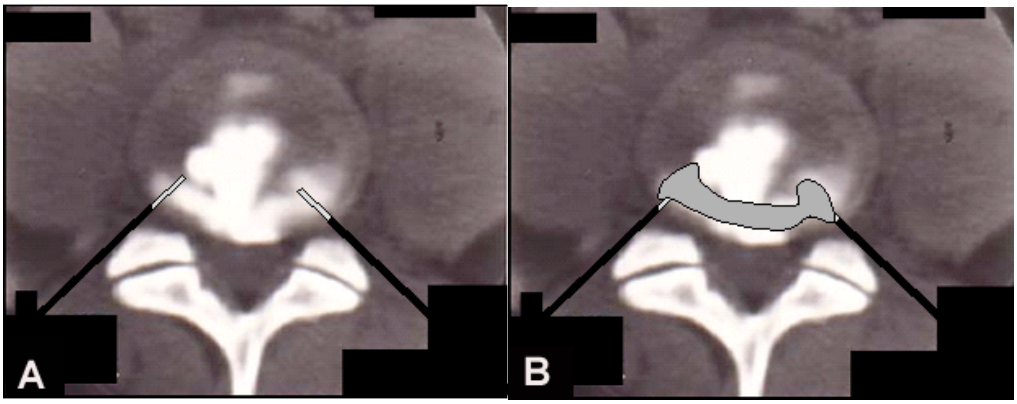

Figure 26. CT scans illustrating the principles of cold RF. **A:** bipolar electrodes are placed into the posterior corners of the disc, bracketing the target fissure. **B:** the lesion produce arches between the electrodes and fully encompasses the target fissure.

ETHICS

Where I and the colleagues in my Department differ from most other practitioners is the context in which we provide intradiscal therapy. We do so only with the approval of an ethics committee. The terms of approval allow us to evaluate the efficacy of such interventions and the efficacy of adaptations, such as multiple placements of electrodes, designed to improve efficacy. In consideration of this approval we undertake to monitor and report our outcomes. Under these conditions we invite patients to participate in studies of emerging technology. That way we offer them the possible benefit of these procedures but without pretending that they will work.

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APPENDIX 1. ASSESSMENT SHEET FOR DISC STIMULATION

Patient's name: …….. ID number:……

Date of Procedure: …… Name of Operator:……

Pre-procedural VAS: ……

SEGMENT STUDIED **RESPONSE**
(Circle appropriate entries)

L…… Not Done Reason: Unnecessary Fusion Inaccessible

PAIN None Dissimilar Concordant VAS:……

PRESSURE Opening:…… Pain:…… Final:……

Remarks: ……

L…… Not Done Reason: Unnecessary Fusion Inaccessible

PAIN None Dissimilar Concordant VAS:…….

PRESSURE Opening:…… Pain:…… Final:……

Remarks: ……

L…… Not Done Reason: Unnecessary Fusion Inaccessible

PAIN None Dissimilar Concordant VAS:…….

PRESSURE Opening:…… Pain:……. Final:……

Remarks: ……

L…… Not Done Reason: Unnecessary Fusion Inaccessible

PAIN None Dissimilar Concordant VAS:…….

PRESSURE Opening:…… Pain:……. Final:…….

Remarks: ……

DIAGNOSTIC CONCLUSION:

Negative Indeterminate Positive Positive Levels……..……

Signed: …… Date: ……..

APPENDIX 2: SCORING SYSTEM FOR RESPONSE TO DISC STIMULATION

VARIABLE		SEGMENTS STUDIED				Sum of
		L2-3	L3-4	L4-5	L5-S1	Rows
CONCORDANT LEVELS	**points**					
Concordant Pain	30					
Pain > 5/10	5					
Pain > 7/10	5					
Pressure < 50psi	10					
Pressure < 15 psi	10					
					SUBTOTAL	
Divide subtotal by number of concordant discs. Enter result in this row.						
CONTROL LEVELS	**points**	L2-3	L3-4	L4-5	L5-S1	
No Pain	30					
Pain at < 50psi	- 10					
Pain at < 15 psi	- 10					
					TOTAL of Sums of Rows below the double line	
Interpretation: > 70 points = POSITIVE 40-60 points = INDETERMINATE < 40 points = NEGATIVE						

1. For each disc studied (see columns), enter the appropriate score for each of the variables indicated (rows).

For discs with CONCORDANT PAIN,

Enter 30 if the concordant pain is produced
Enter 5 if the pain produced is greater than 5/10
Enter another 5 if the pain produced is also greater than 7/10
Enter 10 if the pressure at which pain occurred is anything less than 50 psi
Enter another 10 if the pressure is also less than 15 psi

For discs at CONTROL LEVELS, i.e. not concordant pain,

Enter +30 if the disc was painless
Enter –10 if pain occurred at a pressure less than 50 psi
Enter another –10 if pain occurred at a pressure also less than 15 psi

2. For the CONCORDANT DISCS, add up the scores in each row, and record the sum of each row in the column labeled Sum of Rows.

3. Add up the sums of the rows for all concordant discs, i.e. all scores above the double line. Divide this total by the number of concordant discs, and record the quotient in the cell indicated, immediately below the double line, in the column labeled Sum of Rows.

4. For the NON-COCORDANT DISCS, add up the scores in the rows, taking heed of any negative numbers, and record the sum of each row in the column labeled Sum of Rows.

5. Add up the total of the Sums column below the double line, taking care to heed negative numbers.

6. Interpret the result.