Scientific basis for the treatment of low back pain

RC MULHOLLAND

Spinal Disorders Unit, Nottingham University Hospital, Nottingham, UK

Disc degeneration causes back pain because the function of the disc as a load transferring structure is abnormal, producing abnormal patterns of load which cause pain. Fusion has been unpredictable because it only fortuitously addresses loading. A fusion may be in such a position that it takes load from the disc, and this may be reflected in the way the fusion models, demonstrating a weight bearing pattern, but this is unpredictable. Recognition that we are dealing with a problem of load transfer allows us to design any surgical implant to solve this problem, without stopping movement.

The finite element studies were carried out by Dr Vinu Palissery PhD in the Biomechanical Laboratory of Nottingham University Department of Engineering, under the supervision of Dr Donal McNally PhD.

KEYWORDS

Low back pain – Disc degeneration – Spinal fusion – Surgical implant

CORRESPONDENCE TO

RC Mulholland, Emeritus Professor of Orthopaedic and Spinal Surgery, Spinal Disorders Unit, Nottingham University Hospital, Nottingham, UK
E: mulhollandrcm@aol.com

For over 75 years, spinal fusion has been regarded as an appropriate surgical treatment of so-called ‘mechanical’ low back pain due to disc degeneration. The basis for the use of fusion was the concept that the pain was due to abnormal movement; stopping movement would, therefore, cure the pain. The term instability was commonly used to describe the abnormal movement, and fusion was advocated to cure this instability. The term instability was first used by surgeons in a clinical sense, rather than a mechanical sense; indeed, the first formal definition of instability defined it as a clinical entity of symptoms and signs, and specifically excluded any specific relationship to excessive movement.¹ Bioengineers wrestled with the problem of trying to define a pattern of abnormal movement which was associated with pain, but were unable to do so.^{2,5} Despite this, surgeons, aware that results of fusion were very unpredictable, felt that the unpredictability was due to unsatisfactory fusions. Hence, internal fixation was progressively more rigid, and posterior fusion was combined with anterior fusion culminating in the 1990s with a posterior fusion, with pedicle fixation, and the disc replaced with a cage of metal filled with bone. Yet, as we reached this apogee of surgical endeavour, a Cochrane study⁴ indicated that there was no scientific basis for the use of fusion as a treatment for back pain, and two major, prospective, randomised studies^{5,6} showed in one that surgery was no better than an intensive physiotherapy programme, and the other that, although surgery showed a modest benefit in some patients, a simple posterolateral fusion without instrumentation was as good as any complex method. This disenchantment with spinal fusion has fuelled enthusiasm for the development of methods of treatment of back pain,

which allow movement. The era of the artificial disc replacement had started.

What causes mechanical low back pain?

In the early years of the last century when fusion was done for back pain, pain was ascribed to local radiographic abnormalities, such as lumbarisation, sacralisation, and acute lumbosacral angles.^{7,8} It was not until after the surgery for the herniated disc was well established, that the disc was seen as central to the causation of pain. The writings of Kirkaldy-Willis⁹ established the central role of disc degeneration as a cause of back pain. However he concluded that it caused pain because it produced abnormal movement and the mechanical concept of instability was established. He also postulated that continued pain meant continued tissue damage, thus providing support for the concept that rest and a lumbar support was appropriate treatment for back pain.

Function of the disc

The disc has two functions, to allow movement, and to transfer load. Despite the work of Nachemson and Schultz^{10,11} demonstrating the relationship of pain to load, and indeed the clinical experience of doctors that back pain is so load sensitive, little attention has been paid to the likelihood that back pain is related to a disturbance of load transfer, rather than movement (Fig. 1).

The normal, non-degenerate disc is isotropic, that is to say that whatever position the spine is in, the load is trans-

Figure 1 Relative increase and decrease in intradiscal pressure in different supine, standing and sitting postures compared to the pressure in upright standing. Reproduced by permission of Oxford University Press from Nachemson A. Towards a better understanding of low-back pain: a review of the mechanics of the lumbar disc. *Rheumatol Rehabil* 1975; **14**: 129–43.

ferred evenly over the whole endplate and vertebrae. However, in a degenerate disc, it is non-isotropic, and where the load is transferred to the vertebrae depends on the position of the patient. Hence, when they bend, their load is taken by the front of the endplate and, as they bend and lift, there will be greatly increased focal load. We know that cancellous bone is very sensitive to pressure increases (how immediately we relieve pain if we drill an osteomyelitis). If, as they bend and lift, they cough (increasing also the intervertebral pressure), the pain further increases. The success of vertebroplasty appears to be due not to correction of deformity, but because it supports the thin osteoporotic lamellae transferring load within the vertebrae.

In 1992, McNally and Adams¹² developed a method of measuring stress in the disc under load, which they termed 'profilometry'. Using cadaveric spines, of varying ages and differing degrees of degeneration, they showed, graphically, the pattern of stress or load under varying degrees of load, with the spine in different positions. We subsequently used this technique, in collaboration with McNally, to carry out similar profiles on patients in Nottingham in whom we were doing discograms to identify painful segment prior to fusion.¹³ Our procedure was to pass a profilometry needle into the disc prior to the discography. The profile obtained was stored on a disc and sent to Bristol (we would not know the pattern). Immediately, at the same sitting following the profilometry, pain reproduction studies were done on each disc. The level fused was dictated by the pain reproduction studies. We found that very definite types of profile abnormalities

were present in painful discs, not present in non-painful, but degenerate, discs.

The facet joints

Facet joints become osteoarthritic, especially in the presence of disc degeneration. Clearly, they can be a source of pain. Indeed, surgeons who do total disc replacement often blame their failures on the facet joints, but have failed to provide any evidence that this is true. Repeated attempts to show that there is a facet syndrome, that is a recognisable pattern of pain due to facet arthropathy, have failed.^{14,15} Clearly, facet joint arthritis can cause back pain, and may be contributory to it. Facet degeneration will commonly be associated with disc degeneration, but does not contribute in a major way to the overall severity of the pain in the majority of patients.¹⁶ Posterior fusion, in any event, always fused the posterior facet joints, so that if they were a major cause, one would have expected fusion to be much more successful. A recent study of long-standing, treated spondylolisthesis showed no relationship between the degree of back pain and the presence of facet arthritis.¹⁷

Cage fusion

In the early 1990s, after we had used femoral ring allografts for a time as an implant for anterior fusion, we started to use cages because there were concerns about the risk of prion infection from the femoral rings. As other surgeons found, despite a near 100% fusion rate, the clinical results were no better than those we achieved 30 years earlier. We had expected that the cage, which replaced the degenerate disc, would produce a normal loading pattern. However, from the work of Polikeit *et al.*¹⁸ in Bern, and later Kumar *et al.*¹⁹ from our own department, it was clear that loads under the cages were very abnormal and very high (Fig. 2).

More recently, a prospective, randomised study comparing the use of femoral ring allografts with a titanium cage containing bone showed the superiority of the former in terms of clinical result, not related to a higher fusion rate. It seems likely that this may be related to the fact that transfer of load was through bone remodelling, not through a cage, and that, although integrated to the adjacent vertebrae by bone, the contained bone was not load bearing.

Relevance to disc replacement

The prospective US FDA studies on disc replacement have indicated that the results are no better than fusion at a time when, as has been indicated, the effectiveness of fusion for back pain is questioned. Many advocating the use of disc replacement, accepting that it seems little better than fusion, now claim that its principal value is that it allows

Figure 2 Reproduced with the kind permission of Polikeit A, Ferguson SJ, Nolte L-P, and Orr TE. First used in a poster presentation entitled ‘The importance of the endplate for interbody cages in the lumbar spine: finite element analyses’ at the Proceedings of the 28th Annual Meeting of the International Society for the Study of the Lumbar Spine, Edinburgh, UK, 2001.

movement and protects the disc above from degeneration, an argument not supported by any evidence.

Although over 80 types of disc replacement have been patented, the ones in clinical use all rely on a plate that sits on the vertebrae. Various ‘protrusions’ on the plate maintain its position. However, load is transferred onto the vertebrae through the plate. The vertebral/plate interface is very variable. The endplate is thin, and it is partly surgically removed or thinned, to produce bleeding bone, prior to introduction of the plate. It is never a perfectly smooth flat surface. Is it not likely that the disappointing results of disc replacement, to date, are due to the abnormal pattern of loading beneath the plate?

Finite element study was carried out using a 2-D axysymmetric model of two adjacent vertebral bodies with a disc in between. Four cases were considered:

Case 1 Vertebral body with a natural disc.

Case 2 Vertebral body with intact bony endplate and natural disc replaced by metal disc.

Case 3 Vertebral body with metal disc, and endplate with bony and calcified fibrous tissue.

Case 4 Vertebral body with metal disc and endplate with bony and soft fibrocartilage tissue

Figure 3 Details of finite element model with material properties of various regions for different cases. Finite element analysis was carried out for the region shown in mesh.

Figure 4 Compressive stress distribution in the vertebral body for various interface conditions.

A pictorial representation of the finite element model with material properties is given in Figure 3. Figure 4 shows comparison of compressive stress distribution in the vertebral body for an applied axial load of 1 MPa. The results indicate that there is a stress distribution pattern on changing the natural disc with a metal one. It also shows that, depending on the quality of the soft tissue, there is a variation in the local stress distribution at the interface.

Localised stress points are observed with the presence of non-uniform endplate, even in a simple model used here. It indicates the need for a better understanding of the local stress distribution patterns for the artificial disc vertebral unit, with more realistic geometric and material property distribution.

Implications for artificial disc design

If it is accepted that the artificial disc must transfer load normally and evenly over the endplate, then the structure of the disc should be as near as possible to the isotropic nature of the normal disc, and the disc/vertebral interface should transfer load evenly over the whole endplate. This may mean that the plate must either have the flexibility of the endplate (we know that, under load, the endplate normally deforms) or the plate must be so well integrated to the bone that all parts of the plate transfer load to bone integrated to it, irrespective of the position of the patient.

Flexible stabilisation

With the increasing disillusionment with fusion, and the recognition that stopping movement might not be central to curing back pain, a number of other devices have been in use which have been grouped under the general term 'flexible stabilisation'.

The two that have been used most extensively are the Graf and the Dynesys. Both essentially reduce the range of movement, both in fact specifically stop flexion at the disordered segment. Pressure profilometry has been done in both the Graf and the Dynesys and show that, in the former, load is transferred to the back of the disc, and the overall load though increased is flattened. One feature of profilometry in a painful disc, which we demonstrated was the occurrence of areas of very high load, the Graf may flatten these out. It is of course in the bent position (both bending to lift and sitting down) that most chronic back pain is aggravated; both these systems prevent this anterior overload, so it was not unusual that, after operation, there was an initial favourable result. However, neither system unloads the disc; overall, both increase the overall load.

Both the above systems and a third, the Wallis ligament, were introduced as a surgical treatment for back pain. They have seemingly not fulfilled their promise. The Graf ceased to be used once its creator lost enthusiasm. The Dynesys is currently being evaluated in the US, in an FDA study, but it is being assessed as

an 'add on' to a spinal decompression for spinal stenosis. Its value as a treatment for back pain has been demolished in a recent, prospective, randomised study.²⁰ The Wallis ligament is being used for back pain by surgeons though its inventor, Senegas,²¹ principally advises its use as an adjunct to disc surgery for root entrapment when there has been a major protrusion, repeat surgery for second protrusions, for degenerative disease at a level adjacent to a previous fusion and for an isolated Modic 1 lesion causing back pain. However, he has not reported on its use for low back pain and no prospective studies have demonstrated its value even in the limited areas its inventor has suggested.

The FASS design

In the belief that disc unloading was central to the treatment of back pain, we believed that it would be possible to design a flexible stabilisation system which did unload the disc.²² We placed a fulcrum in front of the posterior ligament, which meant as the ligament was tightened, the segment did not go into extension, which would have transferred load to the back, but the disc height was increased, and the disc was unloaded. The more the disc was unloaded the stiffer the system was, and it was clear that if we attempted to unload the disc fully, then it would have the stiffness of a rigid construct and one could expect that screw loosening, or breakage would occur. Clearly, for such a system to succeed clinically, it must be load sharing, that is under increased load the disc takes load, but is protected from excessive load by the fulcrum. This particular system has not been clinically developed as the design and the nature of the fulcrum was not solved. However, a system that embodies the concept of unloading the disc has been used with apparent initial success in a simple cohort study.

Conclusions

Disc degeneration causes back pain because the function of the disc as a load transferring structure is abnormal, producing abnormal patterns of load which cause pain. Fusion has been unpredictable because it only fortuitously addresses loading. A fusion may be in such a position that it takes load from the disc; this may be reflected in the way the fusion models, demonstrating a weight bearing pattern, but this is unpredictable. Recognition that we are dealing with a problem of load transfer allows us to design any surgical implant to solve this problem, without stopping movement.

Acknowledgement

This Hunterian Lecture was delivered in September 2006 Glasgow at the British Orthopaedic Association Meeting.

References

1. Harmon P. Indications for spinal fusion in lumbar diskopathy, instability and arthrosis. I. Anatomic and functional pathology and review of literature. *Clin Orthop* 1964; **34**: 73–91.
2. Panjabi MM, White 3rd AA. Basic biomechanics of the spine. *Neurosurgery* 1980; **7**: 76–93.
3. Panjabi MM. Clinical spinal instability and low back pain. *J Electromyogr Kinesiol* 2003; **13**: 371–9.
4. Gibson A, Grant IC, Waddell G. The Cochrane review of surgery for lumbar disc prolapse and degenerative lumbar spondylosis. *Spine* 1999; **24**: 1820–32.
5. Fritzell P, Hagg O, Wessberg P, Nordwall A. 2001 Volvo Award Winner in Clinical Studies: Lumbar fusion versus nonsurgical treatment for chronic low back pain: a multicenter randomized controlled trial from the Swedish Lumbar Spine Study Group. *Spine* 2001; **26**: 2521–32.
6. Fairbank J, Frost H, Wilson-MacDonald J, Yu LM, Barker K, Collins R. Randomised controlled trial to compare surgical stabilisation of the lumbar spine with an intensive rehabilitation programme for patients with chronic low back pain: The MRC Spine Stabilisation Trial. *BMJ* 2005; **330**: 1233.
7. Hibbs R, Swift W. Developmental abnormalities at the lumbosacral junction causing pain and disability. *Surg Gynecol Obstet* 1929; **48**: 604–12.
8. Fiske E. Mechanical influences in sciatica. *Am J Orthop Surg* 1921; **19**: 563.
9. Kirkaldy-Willis WH. *Managing Low back Pain*, 1st edn. Edinburgh: Churchill Livingstone 1983, 259.
10. Nachemson A. Towards a better understanding of low-back pain: a review of the mechanics of the lumbar disc. *Rheumatol Rehabil* 1975; **14**: 129–43.
11. Schultz A, Andersson G, Ortengren R, Haderspeck K, Nachemson A. Loads on the lumbar spine. Validation of a biomechanical analysis by measurements of intradiscal pressures and myoelectric signals. *J Bone Joint Surg Am* 1982; **64**: 713–20.
12. McNally DS, Adams MA. Internal intervertebral disc mechanics as revealed by stress profilometry. *Spine* 1992; **17**: 66–73.
13. McNally D, Shackleford IM, Goodship AE, Mulholland R. *In vivo* stress measurement can predict pain on discography. *Spine* 1996; **15**: 2580–7.
14. Jackson RP, Jacobs RR, Montesano PX. 1988 Volvo Award in Clinical Sciences. Facet joint injection in low-back pain. A prospective statistical study. *Spine* 1988; **13**: 966–71.
15. Bogduk N. A narrative review of intra-articular corticosteroid injections for low back pain. *Pain Med* 2005; **6**: 287–96.
16. Dreyer SJ, Dreyfuss PH. Low back pain and the zygapophysial (facet) joints. *Arch Phys Med Rehabil* 1996; **77**: 290–300.
17. Remes V, Lamberg T, Tervahartiala P, Helenius I, Osterman K, Schlenzka D *et al*. No correlation between patient outcome and abnormal lumbar MRI findings after posterior or posterolateral fusion for isthmic spondylolisthesis in children and adolescents. *Eur Spine J* 2006; **14**: 833–42.
18. Polikeit A, Ferguson SJ, Nolte LP, Orr TE. Factors influencing stresses in the lumbar spine after the insertion of intervertebral cages: finite element analysis. *Eur Spine J* 2003; **12**: 413–20.
19. Kumar N, Judith MR, Kumar A, Mishra V, Robert MC. Analysis of stress distribution in lumbar interbody fusion. *Spine* 2005; **30**: 1731–5.
20. Grob D, Benini A, Junge A, Mannion AF. Clinical experience with the Dynesys semi-rigid fixation system for the lumbar spine: surgical and patient-oriented outcome in 50 cases after an average of 2 years. *Spine* 2005; **30**: 324–31.
21. Senegas J. Mechanical supplementation by non-rigid fixation in degenerative intervertebral lumbar segments: the Wallis system. *Eur Spine J* 2002; **11** (Suppl 2): S164–9.
22. Sengupta DK, Mulholland RC. Fulcrum assisted soft stabilisation system: a new concept in the surgical treatment of degenerative low back pain. *Spine* 2005; **30**: 1019–30.