Eur Spine J (2008) 17:619–625
DOI 10.1007/s00586-008-0612-2

REVIEW

The myth of lumbar instability: the importance of abnormal loading as a cause of low back pain

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Received: 15 December 2007 / Accepted: 22 January 2008 / Published online: 27 February 2008

Abstract Spinal fusion became what has been termed the "gold standard" for the treatment of mechanical low back pain, yet there was no scientific basis for this. Operations of fusion for low back pain were initially done at the beginning of the last century for back pain thought to be related to congenital abnormalities or for past spinal infection. The recognition of the disc as a cause of sciatica, commonly associated with back pain, and the recognition that a degenerate disc led to abnormal movement suggested the concept that this abnormal movement was the cause of pain, and this abnormal movement came to be called "instability". Much biomechanical expertise confirmed the fact that degenerate discs led to abnormal movement, there were many hypothesis as to why this caused pain. However clinical results of fusion for back pain were unpredictable. The failure of pedicle screws and cage fusion to improve the clinical results of fusion despite near 100% fusion success, and the introduction of "flexible stabilization" and artificial discs, which demonstrated that despite the often unpredictable movement permitted by of these devices, clinical success was similar to fusion, directed attention to the other role of the disc, that of load transfer, which these devices also affected. Abnormal load transfer was already known to be critical in other joints in the body and had led to the use of osteotomy to realign joints. The relevance of load transfer to the future design of spinal implants used in the treatment of low back pain is discussed, and some finite element studies are reported demonstrating the likely effect of abnormal loading beneath an incompletely incorporated plate of an artificial disc, perhaps explaining in part the somewhat disappointing clinical results to date of the implantation of artificial discs.

Keywords Back pain · Instability · Load transfer · Spinal fusion

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Introduction

The cause and hence the best treatment of "mechanical" low back pain remains unsolved, despite nearly a century of endeavour. It is now generally accepted that some form of failure of the intervertebral disc is central to causation. In the latter half of the twentieth century, failure of the disc leading to abnormal movement, popularly called instability, legitimised the use of fusion as treatment. However the unpredictable results of fusion, which did not improve despite progressively more rigid methods of fusion cast doubts on the concept that back pain was movement related and that stopping movement was central to its treatment. Is it possible that back pain is load related and not movement related? Is instability as a cause of back pain a myth?

Spinal fusion and instability

Spinal fusion as a treatment for back pain was in vogue from the beginning of the last century, but little thought was given to what the pain source might be. None of the papers dealing with fusion until the nineteen-fifties mention abnormal movement as a cause of pain [5, 14, 22, 32, 49, 50].

Then how did the term "instability" become used as a diagnostic term? Barr in 1950 [1] in a review article stated that "It is becoming evident that backache is often

associated with mechanical instability of a degenerate disk lesion". This is the first reference to instability as a cause of back pain that i have been able to find.

In 1954 Harris and MacNab [11] in a paper entitled "Structural changes in the lumbar intervertebral discs and their relationship to low back pain and sciatica," fully addressed the central role of the disc in causing low back pain and sciatica. Although the term instability is used in the paper, it is not suggested that excessive movement is present, and indeed translational movement is deemed to be unusual. The term unstable is used to mean a disc whose movement is irregular.

In 1962 Harmon [10] presented a review paper at the western orthopaedic association meeting in San Francisco, in which the term "Instability" appears.

However, Harmon's description of what he meant by instability (unfortunately in a footnote) is revealing

"Spinal instability refers to a low back-gluteal-thigh clinical triad of symptoms that may be accompanied (overt cases) by incapacitating regional weakness and pain. This is the effect of disk degeneration with or without disc hernia. Some may be asymptomatic or slightly symptomatic when instability is compensated by muscle or ligament control. It does not refer to spinous process or laminal hypermobility which some surgeons like to demonstrate at the operating table nor does this clinical concept parallel the common spinal hypermobility, which is the product of intervertebral disc degeneration, demonstrable in flexion-extension filming of the region, since the anatomic hypermobility is not always productive of symptoms".

Sadly this description of instability appears to have been ignored, and the concept of mechanical instability as a cause of back pain was progressively accepted. Harmon's view of the effect of fusion was that it cured pain by reducing the irritation of the neural contents produced by movement. His paper was influential as he emphasized the importance of appropriate investigations prior to fusion and the segmental nature of back pain but unfortunately his use of the term instability was interpreted as supporting the view that segmental abnormal movement was the cause of the pain.

In 1965 Newman [37] in an editorial concerning lumbosacral arthrodesis refers to the need to stabilize the lumbar spine in patients with back pain after discectomy for a lumbar root entrapment.

At the beginning of the seventies the perception was that disc degeneration led to abnormal translational movement, and this was painful.

McNab in 1971 [26] who had done much work on the disturbance of movement in the degenerate disc described what he termed the "traction spur," a particular type of anterior osteophytes which he said was related to an abnormal pattern of translational movement. This view again supported the concept of instability. He added the important caveat that it "was impossible to establish the clinical significance of the traction spur as a statistically valid investigation The traction spur was revisited in the late eighties and was shown to be no different to claw osteophytes, and often both would be present in the same patient [12, 43]. It was not related to abnormal movement".

Although McNab used the term instability, he used it in the sense that the spine was vulnerable to acute episodes of pain, because the degenerate disc rendered it more easily injured. He did not view it as a cause of chronic back pain.

Kirkaldy Willis set out his views on instability in 1982. In "Instability of the Lumbar Spine" [18] he described the process of disc degeneration as passing through a stage of dysfunction, (intermittent pain), instability which caused more persistent pain but then with time stabilising to a painless state. This was his explanation for the observed fact that many very degenerate discs were painless. However, he at that stage was somewhat unhappy with an entirely mechanistic view for pain. Hence he writes "Instability can be defined as the clinical status of the patient with a back problem who with the least provocation steps from the mildly symptomatic to a severe episode". Further he writes "Detectable increased motion does not always solicit a clinical response, and that abnormal motion may be abnormal increase or abnormal decrease". He further writes "It is insufficient to detect the abnormal increased motion, but the mechanism by which it precipitates the symptomatic episode must also be identified". Indeed in the seven cases he reported only one patient had backache alone, the others were all radicular problems. His paper shows that identifying abnormal movement establishes the fact that the segment is disordered, but he does not in that paper indicate that movement itself is the cause of pain.

Subsequently in his very influential book "Managing Back Pain" in 259 pages just one page is devoted to the rationale of lumbar fusion. The only reason for fusion appeared to be that, other treatments had failed, that it was reasonable from the psychological viewpoint, and that instability was present. Instability is defined elsewhere in the book as increased abnormal movement, and this is illustrated by x-rays purporting to show abnormal rotations and various types of abnormal tilt. He accepts that such appearances may be entirely painless, but in the patient with back pain they identify the causative level, and fusion is justified.

However, in a joint paper with Depuis in 1985 [4] entitled "Radiological Diagnosis of Degenerative Lumbar instability" they write "A lumbar motion segment is considered unstable when it exhibits abnormal movements. The movement is abnormal in quality (abnormal coupling patterns) or in quality (abnormal increase of movement...)

Pain is a signal of impending or actual tissue damage-and when present it indicates that a mechanical threshold has been reached or transgressed. Repeated transgressions will damage the stabilizing structures beyond physiological repair, thus putting abnormal demands on secondary restraints".

Hence from being a method of identifying an abnormal degenerate disc, abnormal motion itself became the injurious agent.

Instability and the basic scientists

In the eighties there was an increasing realization that basic science must play a major role of we were to advance our understanding of spinal function. As the term instability seemed to be such a major aspect of back pain, help was sought from bioengineers. Between 1980 and 1995 some 83 papers were published dealing with instability and associated biomechanics.

In 1985 Pope and Panjabi [45] in a paper entitled "Biomechanical definition of spinal instability" wrote "Instability is a mechanical entity and an unstable spine is one that is not in an optimal state of equilibrium. (...In the spine stability is affected by restraining structures that if damaged or lax will lead to altered equilibrium and thus instability. Instability is defined as a loss of stiffness". Panjabi's views were generally accepted by basic scientists interested in this field.

Subsequently Panjabi concluded that increased movement was not necessarily a feature of what he termed instability, but reduction in the neutral zone was [41]. However, in a more recent paper he has abandoned the concept of instability altogether, and ascribes chronic back pain as being caused by ligament sub-failure injuries leading to muscle control dysfunction [42].

However, throughout the period from the fifties to the nineties, the Panjabi view held sway, and the term instability evolved from being a useful term to denote a segment that was abnormal due to a degenerate disc, to a term denoting a diagnosis of an abnormal, (usually increased) pattern of movement with a translational component. The abnormal movement was thought to be the cause of the pain and clearly fusion or stopping movement was a logical treatment.

However, the inability to show that abnormal or increased movement was a feature peculiar to the painful degenerate disc, combined with the fact that despite more rigid fusions using pedicle fixation, the clinical results of fusion had not improved, was increasingly casting doubt on the concept of instability. The paper by Murata [35] combining MRI examination with flexion and extension films in patients with back pain, showed that increased angular and translational movement was a feature of the normal or mildly degenerate disc, not of the markedly degenerate disc, where movements were reduced. In 1998 Kaigle et al. [17] demonstrated that comparing patients with normal subjects there was always less movement present in the degenerate spine. It was therefore generally accepted that the effect of disc degeneration was to reduce movement [6, 53] not to increase it, as the term "instability" would imply. It may be argued that, unfortunately, this reduction of movement is associated with abnormal patterns of movement, and this is the meaning of "instability". However despite considerable efforts over many years, using flexion/extension films, no clear relationship has been established between pain and such abnormal movements. In other words, patients with degenerative disc disease may exhibit abnormal patterns of movement, yet have no pain. The one new movement that may be present in some patients with degenerative disc disease is excessive translational movement, described by Fujiwara [7, 8] as been seen in patients with degenerative spondylolisthesis. Dealing with this observation, two recent papers concerning back pain and translational movement are of importance. Igunuchi et al. [15] examined flexion and extension movement in symptomatic patients, and found that whereas flexion and extension films were not of value, if translation occurred, then it was greater in the more symptomatic, if 3 mm or more. However the paper by Ochia et al. [39] examined asymptomatic subjects, and showed that translation movement was often greater than had been shown to be present in the symptomatic patients reported by Iguchi, or by Fujiwara, being up to 5.4 mm.

By the mid nineties, instability was still the term used to describe the disorder that we treated by fusion, but the failure to improve results by the introduction of pedicle fixation, caused many surgeons to question the concept of instability, but surgeons were all aware that fusion although unpredictable in terms of clinical result, was the best surgical treatment for chronic low back pain. It was well recognized that clinical success was unrelated to the success of the fusion, pseudarthrosis was as common amongst successful patients as in those who had failed [46]. Was there anything else that a fusion did to the intervertebral disc unrelated to the fact that it stopped movement?

The intervertebral disc

It is recognized that morphological changes in the disc play a major role in low-back pain, although it is also recognized that there is no correlation between the degree of degeneration and the severity of back pain. The disc has two biomechanical roles, it must transmit load and it must allow a controlled range of movement, so that such

movement does not compromise the adjacent neural elements.

The disc is well designed to transmit load. The normal disc is isotropic that is to say that it behaves liked a fluid filled bag, and transmits load uniformly across the surface of the disc and to the endplate [27]. This has a number of important biomechanical consequences. Key among these is that in any position of the spine, be it flexion, extension or lateral-bending, the load is transmitted uniformly over the endplates. There is no high spot loading related to different positions of the spine. It may be recalled that this is the case in a normal diathrodial joint. Here, the design of the joint ensures an even pattern of load transmission. Disturbance of anatomy of the joint, such as a menisectomy in the knee, or destruction of the cartilage by disease (infection/arthritis) in other joints, leads to a disturbance in the normal weight transmission, producing pain. It was recognized many years ago that an appropriately planned osteotomy, which altered the load transmission, might result in pain relief. We are all familiar with radiographs showing the high spot-loading in a subluxing hip, and are also aware that an appropriate osteotomy, which reduces spot-loading by containing the hip, relieved pain. Similarly, in a varus or valgus deformity of knee joint, abnormal distribution of load may lead to unicompartmental osteoarthritis, and an appropriate osteotomy [16, 51] especially if done early with minimal cartilage loss, would produce a satisfactory long-term result. We emphasize that the biomechanical effect was to alter the loading pattern, and the resultant clinical effect was pain relief. Prior to the development of hip replacement, much thought was given to what was the pain source. The capsule was thought to be causative of pain, yet when we replace hips now, we do not remove the capsule, but we do create a loading pattern over as wide an area as possible, and we know if we get stem point loading we expect pain. If we accept that in load-bearing joints overall an altered loading pattern produces pain, then we can more easily accept that his concept also applies to the disc. This load transfer is to the underlying vertebrae. The vertebrae are well innervated, and also sensitive to excessive pressure, as shown by the severe pain of an osteomyelitis relieved immediately if the bone is decompressed. Another important consequence of the uniformity of distribution of load transmission across the surface of the disc is that it transmits load to the annulus, producing a tension in the annulus, and converting it into a load-bearing structure. It is established that disc degeneration alters the isotropic nature of the disc [19, 27] and, as a consequence, load transmission over the endplate becomes irregular, leading to high spot-loading, particularly associated with certain positions. It will be appreciated that the degree of disc degeneration itself will not affect to the loading pattern, as the anatomy of the degenerate disc is so varied.

Biomechanics of the disc and relation to load bearing

It is curious that instability or movement abnormalities are blamed for chronic back pain. Whenever a history is taken of chronic back pain, it is clear that the problems experienced are postural, rather than related to the process of movement. It is pain whilst bent down, it is pain whilst leaning forward, and it is pain whilst sitting. As the loads on the back are mostly produced by muscle action rather than body weight, activities that involve strong muscle action, such as lifting, are associated with pain. Standing perfectly still with a heavy load is a painful experience for some patients with a painful back, quite unrelated to any movement. We are all aware that lying down flat and reducing the load relieves pain. Nachemson [36] in his classic work, showed very clearly the close relationship of posture and stress in the disc, and later Schultz [48] demonstrated the important effects of muscle action on these stresses.

Was there any way we could demonstrate how disc degeneration could affect load transfer, and could we find a connection between disc degeneration and abnormal loading?

At that time we were routinely doing discography prior to fusion to identify the appropriate level to be fused, identifying the painful disc, which involved injecting a number of degenerate discs in each patient. Using the technique developed by McNally [27], it was therefore possible for us to carry out a profilometry immediately before the discography at each level examined. Discs which demonstrated that the nucleus was taking no load, that is, it was all annular load, or discs that had very focal areas of high load were the painful discs. Other patterns of profilometry of a degenerate disc were not painful [28].

Consequences of abnormal load bearing being a continuous source of pain despite use of pedicle fixation

It had been recognized by O'Brien [38] that pain could continue despite a structurally successful posterolateral fusion, and to an extent the re-use of anterior fusion in the nineties was consequence of this. However with the introduction of pedicle fixation and the recognition that despite the great rigidity of this, clinical results did not improve, surgeons spoke of "vertical instability", a concept rather akin to abnormal loading. However, disappointment with pedicle fixation, and a wish to dispense with interbody

fusion using bone, with its uncertain biomechanical properties led to the introduction of cages.

Cages

However, despite the fact that in terms of stopping movement, cages were immensely successful, again the clinical success much the same as older methods of fusion, even if combined with a posterior fusion as well [24, 25]. Clearly the cages stopped any disc loading, as they replaced it. However, loading occurred through the cage, and how it affected the underlying vertebrae, and transmitted load to it was dependant on two factors.

First, the "foot print size" was clearly critical, if the entire load was transmitted through a small area, then clearly the load per square millimetre of underlying bone would be very high (Polikeit [44] and Kumar [20]).

Second, the nature of the cage–bone interfaces. The principle behind the use of cages was that the cage provided mechanical support, and then bone placed within the cage and around it, joined across the gap between the two vertebrae. This had the effect of producing a fusion by bone. However whilst that fusion may be very effective in stopping movement, it was deficient in relation to load transfer. When the cage was subjected to load, load was transferred to the vertebrae by the stiffer metal, so that although fusion by bone would occur, stopping movement, bone was not transferring load. The early cages (Bak) had two defects, they had a small "footprint" and also because of their design, the contained bone never became a significant factor in load transfer. As a result it was an unsatisfactory cage [21]. McAfee [24] pointed out that a feature seen radiologically in patients with a good clinical result was the formation of bone, outside the cage which by its appearance was organized to take load. Cages which have a very large footprint covering the whole of the vertebrae, and whose contained bone became wholly weight bearing, the cage itself taking no load would create a pattern of load transfer as achieved by those fusions where there is organized bone load-bearing over the whole space. The recent paper by McKenna [25] demonstrating the clinical superiority of femoral ring allographs compared with a metal cage supports the view that load transfer by bone is superior to load transfer by a cage as the work of Polikeit (op cit) would suggest.

Flexible stabilization

The various devices that have recently been introduced may be discussed under the term flexible stabilization. They do relieve back pain, although whether their overall clinical success rate will justify their continued use has yet to be determined by appropriate prospective randomized trials. They restrict movement in all planes, other than translational; they do not produce the rigidity of fusion or stop movements that were regarded as evidence of instability. Despite this the reported early results were similar to fusion. The devices in clinical use all restrict the range of flexion and extension, and hence in the non-isotropic disc alter the loading pattern. The Graf ligament stopped flexion, and created a lordosis, increasing overall load in a neutral position, and concentrating it posteriorly [9]. The Dynesys in cadaveric studies, transferred load posteriorly [29], but in clinical use it restricted both flexion and extension, in some patients increasing load slightly anteriorly, but preventing this increasing, as it stopped further flexion [2]. The Dynesys has been shown to reduce the effect of loading on the disc overall, measuring pressure in the centre of the disc, so it would reduce any high spot loading associated with a particular position [47]. The Wallis ligament has had no loading studies done, but is likely to be similar to the Dynesys, restricting both flexion and extension. Clearly none of these devices produce rigidity of a fusion, and the clinical efficacy would seem likely to be due to the fact that they prevent the spine from moving into a position where pain is experienced, (especially flexion) they certainly alter the loading patterns of the segment. The association of sitting pain (essentially flexion pain) with loading of the front of the disc is well shown in the paper by Maigne [23] where he demonstrated that patients who got pain sitting, relieved when they stood up, showed marked loss of the anterior disc space.

Artificial disc

The introduction of the artificial disc, producing a clinical success similar to fusion provides strong support for the hypothesis that loading is central to back pain. The artificial disc does not create a pattern of normal movement, and is not designed to do so. It does not necessarily require the removal of the posterior innervated annulus, often regarded as a source of back pain, which continues to be moved and stressed after operation. The one mechanical effect it has is to alter load transmission, whilst allowing movement.

The interface between the implant and the underlying vertebrae is critical. It is through this interface that load must be transmitted. Bony integration must not only stop movement, but it must be sufficiently extensive that the area of bone transmitting load is adequate. Our experience with cages demonstrated that a small area of bony integration sufficient to stop movement, but insufficient to transfer load over a wide footprint was associated with continued pain.

With the artificial disc with a larger foot print transfer of load is through plates resting on the vertebrae—a universal feature of currently used disc replacements. This load transfer may be abnormal if the bone–plate interface is a mixture of fibrous tissue and bone, producing an irregular pattern of load transfer. This clearly could be one of the reasons that disc replacement is so far not a better procedure than fusion in prospective randomized trials comparing the two techniques [3, 54].

Over the last year we have been examining the effects of interface between an artificial disc and the vertebrae using the technique of finite element modelling [33, 40].

The results of these studies indicate that if the load bearing function of the artificial disc is transmitted through a rigid plate, then unless that plate is joined by bone throughout all or most of its surface, so that it transmits load evenly over the endplate, then high spots of loading may well occur... Diagnosis of such incomplete union will not be easily diagnosable, as there will be no movement, and identifying what proportion of the plate is transmitting load through fibrous tissue, or not transmitting load at all will be impossible. Design of an artificial disc must be focussed on its role as a transmitter of load, not an enabler of movement. It seems probable that one cause of clinical failure of an apparently satisfactory artificial disc may be due to failure to establish a normal loading pattern at the plate bone interface.

Conclusion

Abnormal movement of a degenerated segment may be associated with back pain but is not causative. The concept of instability as a cause of back pain is a myth. The clinical results of any procedure that allows abnormal disc loading to continue are unpredictable.

If it is accepted that load transfer disturbance is the central issue in mechanical back pain, then treatment can be directed to remedy this. Fusion will only do this if it reliably takes over the loading function of the disc. Movement preserving procedures such as "flexible stabilization" or an artificial disc are compatible with preserving motion but with an artificial disc bony integration between plate and vertebrae would appear to be essential, not just to stop movement, but to transfer load normally.

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