

This was my covering letter to Science Advances in 2016.

I submit the attached paper which was rejected by "Spine" and "The Journal of Biomedical Engineering" (UK) in 1987. (*This updated paper was rejected by Science Advances by return of post!*)

After submission in 1987 I remained in this field for the rest of my career. I am now 73 and retired. I qualified as a chiropractor in 1989 and practised till 2014, twenty-five years. During this time, I used McKenzie (a physiotherapy system) for most cases of acute low back pain. I trained with the McKenzie Institute UK in 1989 or 1990. McKenzie is entirely compatible with this argument.

I also manufactured office chairs (as Pelvic Posture Ltd 1985 – 2002) and replacement car seats (as Gorman Design Ltd.) These chairs and car seats were based, not on lumbar support, but on pelvic support as described in my US patent number 4911502. This patent was granted in about 1985 and expired in 2007.

Although my paper is twenty-nine years old, the points I make are still entirely new to the literature of orthopaedic medicine and low back pain. These are:

1. The sole reason for the lordosis in the human spine is the birth canal. This defines the position, angle and shape of the sacrum. This point does not appear in any of the medical literature of low back pain. The most common erroneous explanation is to liken the S shape of the spine to an S-shaped spring as mentioned and referenced in my paper. The same erroneous explanation appears as late as 2013 in Spine. The obstetric reason for the evolution of lumbar lordosis is however fully accepted in the field of anthropology as I referenced in 1987. More recent papers include "The Obstetric Dilemma" by a professor of anthropology from Cambridge UK. (My ref 14*)
The alternative reasons for lordosis published in Spine, and other medical literature over the years, are interesting only in their almost incredible "pseudo-engineering" content.
2. The angle of the sacral end-plate from the horizontal, and the resulting lordosis within the pelvis, mean that the lowest two discs have such a pronounced wedge shape (around 20deg) that they cannot flex beyond parallel sided even in full flexion. (By "parallel sided" I mean the angle of flexion of the disk when the superior and inferior endplates are parallel. This limitation applies, of course, to the natural or asymptomatic spine.)
3. The prominent sacrum, necessary to accommodate a birth canal, is the origin of the most important tension structure when the body is a cantilever in lifting. However, this origin is below the lowest joints of the spine which makes the whole structure an "over-centre mechanism" and therefore liable to collapse. This collapse i.e. hyper-flexion of lowest discs, would result in instantaneous prolapse as shown by the in-vitro experiments of Adams and Hutton. (Spine 1984 My ref 17*)
4. At the time of writing in 1987, the majority of papers blamed the facet joints as the source of LBP. This opinion certainly prevailed through the nineties. This was the motivation behind the surgical interventions performed by Dr Markwalder in Switzerland from 1990 till 1995. (See below)
By the time of Bogduk's** 4th edition in 2007, research consensus was that the nerves in the posterior annulus of the disk were the source of LBP and that radial cracks in the postero-lateral sectors of the disk allowed access of the nucleus to these nerves.
These cracks however are always viewed as a fault or deficiency in the spine. My hypothesis, in my 1987 paper, was that this is a pain that has evolved to be very severe in order to avoid the prolapse described in (3) above. An instantaneous prolapse would result in direct impingement on the spinal cord or nerve roots and would be disabling.
Over most of the five million years of our bipedal evolution this would have been fatal to the sufferer and dependent offspring. This is strong evolutionary pressure to find a solution. Low back pain is that solution.
5. I suggested in 1987 that the nucleus of a disk would migrate posterior on flexion through the parallel sided shape. Since the relevant disks should not flex beyond parallel sided, the nucleus in these two disks would always remain anterior and would not reach the nerves in the posterior annulus even if there were cracks in the annulus. (This migration was confirmed in 2007 by the MRI studies at Aberdeen, Scotland. My ref 31)

6. In the natural lifestyles during the last five million years, of our various ancestors, natural selection would ensure that it would only be in rare cases that this “warning pain” would be triggered. However, the mechanical situation when sitting with a backrest is completely different from natural slumped sitting. The effect of a backrest is always to flex the lowest joints while hyperextending some upper lumbar or lower thoracic joints. This effect is only accentuated by shaping the backrest to provide “lumbar support”. This was clear in the tabulations in Nachemson’s 1979 paper in Spine (my ref no.5) but was not recognized by the authors. Thus all backrests, particularly if shaped for lumbar support, will distort the shape of the lumbar spine. This will, in many cases, take the lowest joints into that range of flexion, beyond parallel sided, which will trigger the warning pain; Low Back Pain.



I emphasize that the above points constituted the argument of my 1987 paper and that none of the points 1 thru 6 above appear, or have been accepted, in the literature of low back pain in the subsequent thirty years.

If the above mechanical argument is correct, it should be obvious that the ubiquity of sitting with a backrest in our lifestyle will make low back pain almost universal, as indeed it is! It should also be obvious that the lumbar support car seat provides the worst mechanical situation for the longest periods of time and should be expected to correlate with the highest incidence of back pain. This is also found to be the case.

I suggested thirty, and more, years ago that surgical intervention to limit the flexion of the L4/5 and L5/S joints would probably “switch off” the warning pain and provide a real solution to low back pain in the early stages before any actual damage had occurred. I found by chance very recently that this operation had been done by Dr Markwalder in Switzerland between 1990 and 1995 with almost 100% success post op, and over 75% at five year follow up; an unknown success rate in surgery for low back pain.

Dr Markwalder’s intention was to immobilize the facet joints, which he did in the extended spinal shape of the relevant spinal joints with a Graf ligament. It was hoped that this would allow these joints to recover from whatever trauma the capsules had suffered.

As mentioned above, the facet joints are no longer seen as being the source of low back pain. Instead the posterior migration of the nucleus is now seen as the source (Bogduk 4th edition 2007) so it is very likely that the success of this surgical intervention was because it limited flexion to the natural level (or less) and ensured that the joints did not flex beyond parallel sided. This ensured that the nucleus remained anterior in the disk and that the warning pain was therefore not triggered.

The same limitation of flexion could be achieved without using the invasive Graf ligament. Simply extending the supraspinous ligament from the spinous process of L4 to the sacrum would achieve the necessary limitation. This prosthetic ligament should be able to stretch elastically but not plastically. A preliminary proposal is at www.naturaljointmobility.info/prostheticsupraspinousligament.htm This would be a very superficial surgical intervention which might become an almost universal solution to low back pain in the early stages and avoid the frequent progression to real disk damage.

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* References are in the paper at www.naturaljointmobility.info/theobstetric.pdf

** The Clinical Anatomy of the Lumbar Spine and Sacrum. Nikolai Bogduk (Book now in 5th edition.)