This paper was rejected by Spine and by The Journal of Biomedical Bio-Engineering(UK) in 1987.

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The Obstetric Reason for Lordosis and the Implications for Lifting and Low Back Pain

Leading to:

A Relatively Simple Surgical Cure for Non-Specific Low Back Pain

Author; John D Gorman

MA (Cantab. 1965) (Mechanical Sciences)
C.Eng. (Chartered Engineer)
MIET (Member of the Institution of Engineering and Technology, UK)
MIMech.E (Member of Institution of Mechanical Engineers, UK)
McTimoney Chiropractor 1989 till 2014 - retired

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Abstract

A review of the reasons proposed for lumbar lordosis suggests that the obstetric reason is primary. Evaluation by scale drawing of the moment arms of erector spinae muscles shows how evolution has taken advantage of the lordosis and the shape, angle and location of the sacrum to maximise lifting strength. However, this makes the structure an "over-centre mechanism". Muscular moment arms are reduced by flexion and can be reduced sufficiently to provide the conditions of flexion and compression which may cause disc prolapse. It is proposed that prolapse is not common because a warning pain inhibits activity when either the L4-5 or the L5-S disc is flexed (or can flex) beyond parallel sided. It is further proposed that civilised sitting, with a backrest, will tend to increase the mobility of these joints and thus cause low back pain.

(Keywords: lordosis, obstetric, lifting, sitting, back pain,)

SECTION 1

The Reason for Lordosis

In the bipedal human being the primary function of the spine is to act as a column to support the upper body, head and arms above the pelvis and legs. One would expect such a column to be straight.

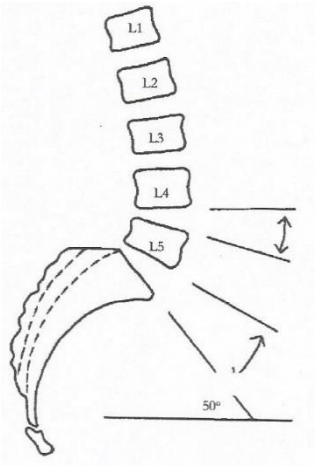

Figure 1

In fact, the lumbar spine is sharply curved at the base. The sacral endplate is frequently more than 50° from the horizontal and the lowest two discs are sharply wedge shaped.

Figure 1 is a typical shape redrawn from an X-ray tracing in a report on a population chosen because of "the rarity of the disc syndrome" (*1*) (50° sacral endplate angle as shown in figure in that paper).

There have been many attempts to explain this lordosis in the human lumbar spine.

One suggestion is that it is simply a transitional result of bringing the upper body vertical for bipedalism while the pelvis retains its orientation in the ape. (*2*) Having remained largely unchanged for four million years suggests that it is not a transitional form.

Another suggestion is that the S-shape of the spine (including the neck) acts as a spring to vertical shocks or displacements (*3*) Anyone can check in their own body that the spine does not act in this way. All parts of the spine, lordotic and kyphotic, flex to response to vertical shock such as landing from a jump.

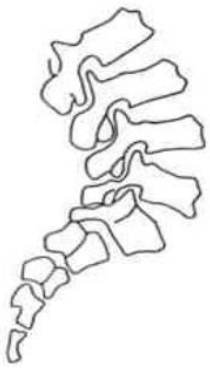

Figure 2

It is often assumed that all lumbar discs should be similar and only slightly wedge shaped in the erect position. Many diagrams, including those in technical journals, are drawn to show this (*3, 4*, 5). In fact, the lowest two discs in those free of back pain are sharply wedge shaped**.** Figure 2 (of a child) shows how extreme this can be in the population chosen "because of the rarity of the disc syndrome" (1).

In a sample of 20 from the same population obtained subsequently by this author, the mean wedge shape of L5/S was 24degrees and of L4/5 16 degrees.

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In an English group chosen because "none had suffered any back pain within 12 months or had a history of back pain" (*6*), the mean wedging of the L5-S disc was 18° and of L4-5, 16°, in the standing position". (6). Analysis was done recently (*1983-6*) by this author from the original X-ray films.

The angles from non-back sufferers in a sedentary population and the similar angles shown in the example from the more naturally living group confirm the sharpness of the lordosis and the very pronounced wedge shape of the lowest two lumbar discs.

Any hypothesis proposed as a reason for lordosis must explain this shape in the lower lumbar and sacral area of the spine. Mechanical arguments to do with columns and cantilevers will not do so. They will suggest that the spine should be straight.

The Obstetric Explanation

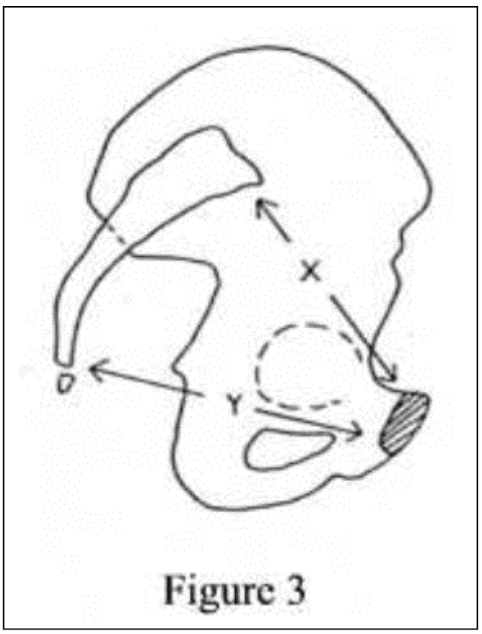


Figure 3

The shape of the lower lumbar spine is defined by the angle, shape and position of the sacrum in the pelvis. Figure 3 shows this. The ventral surface of the sacrum is the arc of a circle centred on the pubic symphysis.

This shape and angle of the sacrum is absolutely necessary to the passage of the baby's head through the mother's pelvis in childbirth. The dimensions x and y in Figure 3 are the pelvic inlet and outlet a-p diameters respectively and are used by the gynaecologist and midwife in predicting the difficulty of childbirth.

It is proposed here that this is the sole reason for the lordosis in the human spine.

This is confirmed in an analysis of the changes that took place in the evolution of one of the earliest bipeds, Australopithecus afarensis (e.g. Lucy) from the previous chimpanzee-like ape (7).

"Among primates, hominid (i.e. Australopithecus or Homo) morphology is unique. The non-hominid primate (e.g. chimpanzee) ilium is cranio-caudally elongated separating the sacroiliac and hip joints … As an adaptation to bipedality among hominids the lower iliac height shortened, thereby narrowing the distance between the sacroiliac and hip joints. However the approximation of these joints is obstetrically disadvantageous as the anteroposterior diameter of the pelvic inlet becomes constricted … The difference in sacral position between non-hominids and hominids also is obstetrically significant. Among non-hominids the sacral apex does not extend down to the level of the ischial spines; the dorsal walls of the pelvis midplane and outlet are muscular. Among hominids, the reduced distance between the sacroiliac and hip joints lowers the sacrum into the pelvic cavity; the ventral surface of the sacrum forms a prominent bony wall of the true pelvis. Obstetrically, the sacrum reduces the sagittal dimensions of the hominid birth canal, particularly at the midplane and outlet. In contrast with other primates, the position of the hominid sacrum may contribute to parturitional difficulty." (7).

Figure 4(a) shows the chimpanzee pelvis positioned as in a vertical body. The height of the pelvis ensures that the sacrum does not obstruct the birth canal. The sacrum is approximately vertical resulting in a straight lumbar spine.

Figure 4b shows the pelvis of Lucy. The overall height of the pelvis is greatly reduced. The ilia have been modified such that the iliac crest is closer to and above the hip joint. This allows the muscles from the iliac crest to control the leg laterally when walking. "As an adaptation to a bipedal gait in which the thigh passes underneath and behind the hip joint, hominid ilia rotated to a more sagittal plane … The gluteal muscles (medius, minimus and upper part of maximus) became positioned to function as abductors. These muscles are principal in controlling pelvic balance in the transverse plane during the single support phase of stride." (7).

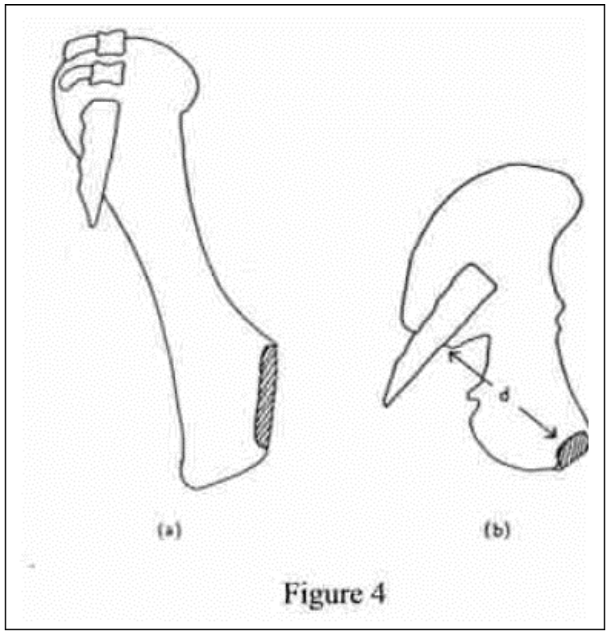


Figure 4

With the shape of the ilia thus defined by the needs of bipedalism, there is no possible configuration of a vertical sacrum and straight spine, which would not place the spine too far behind the acetabulum. Figure 4(b) shows the configuration. The sacral angulation in Lucy is less than the mean of human females but within the range of human male or female angles (7). This resulted in a lumbar spine very similar to the modern human with a marked lumbar lordosis. The sacrum probably did not have the characteristic

curvature of the human sacrum and the limiting dimension for childbirth was between "… the pubic symphysis and the centre of the sacrum" (d in Figure 4 b), (7).

In comparison with the chimpanzee, childbirth was almost certainly difficult for Lucy ("adaptations for locomotion and visceral accommodation and support functions at the expense of parturitional ease"), (7). The human adult brain is almost four times as large as that of Lucy (similar to chimpanzee (7); chimp 300-400 cc (8), human 1350 cc). As brain size grew in steps from Lucy to Homo habilis to Homo erectus and to ourselves, childbirth must have been a major problem and a limit on our evolution.

There are four main parts to the solution that has evolved;

1) We are born early with a small and incompletely developed brain. Comparing our lifespan with other primates, we should have a gestation period of about 18 months instead of 9 (9). "Among extant hominoids (includes the chimps, etc), humans have the lowest newborn-to-adult endocranial ratio" (7).

2) The human cranium is "collapsible" at birth with the four main cranial bones overlapping one another and greatly reducing the transverse and a-p diameters of the foetal cranium. This mechanism does not exist in the chimpanzee or any other ape.

3) During human parturition, both the pubic symphysis and sacroiliac joints can separate slightly to increase pelvic diameters.

4) The fourth part of the solution must be to maximise the size of the birth canal in the pelvis where this is at all possible and maybe at the expense of perfect adaptation for other purposes.

Inherent faults?

Investigations of more naturally living peoples speak of "the rarity of the disc syndrome" (*1*) among one group, and in another "primitive population" the incidence of disc narrowing was very much lower than among "North Americans and North Europeans" (10).

Numerous epidemiological papers show the increased incidence of low back pain and disk degeneration with a sedentary lifestyle particularly when driving. This was noted as early as 1972 by Kelsey and Hardy (11), was confirmed in 1986 in Spine (12) and was strongly emphasised by the work of Professor Mark Porter, Professor of Ergonomics at Loughborough University, UK, who observed "-- -- a six fold increase in -- -- low back trouble by car drivers who spend more than half the working day behind the wheel" (13)

It seems likely that low back problems are a result of some aspect of civilised life rather than an inherent fault in the spine. The human spine has evolved for at least four million years with this constraint and seems to be relatively free of problems for more naturally living peoples.

Conclusion to Section 1

Throughout our evolution as a bipedal animal, childbirth has been difficult. The need to get the increasingly large foetal head through the pelvis has defined the angle, shape and position of the sacrum in the pelvis and the concurrent evolution of the spine for bipedalism had to proceed with this as an unalterable constraint.

Thus, the lordotic shape of the lower lumbar spine including the sharp wedging of the lowest two spinal discs is a direct result of the needs of childbirth.

As far as I know, this point, critical to the understanding of the spine and low back pain, is still not recognised in medicine in 2017 despite being generally accepted in anthropology since 1986 (7,14) or much earlier.

SECTION 2

Wedge Shape and Flexion Limit of L4/5 and 5/S Disks.

In my 1987 paper, I referred to two papers on the shape and mobility of the lumbar spine. I have since found another important paper. Fortunately, the three are of different populations living very different lifestyles;

A) White male English men in sedentary occupations. (6)

B) Male and Female people in Iran of presumably Arab ethnicity. (15)

C) Male and Female black South Africans. Some were nurses. Some were tribal living (1)

The similarity in the shape of the lower lumbar spine of each group allows the following generalisations to be made;

L5/S	*Angle of wedging when standing*	*20°*	*(18-23°approx)*
	Mobility in Flexion	*10°*	*(8-15°)*
L4/5	*Angle of wedging when standing*	*16°*	*(14-18°)*
	Mobility in Flexion	*14°*	*(14-20°)*

In both joints the angle of wedging when standing is greater than the mobility in flexion so it can generally be stated that, for these three very different populations:

The lowest two spinal joints, L4/5 and L5/S, do not flex beyond the endplates being parallel even in full flexion. *(This point is covered in more detail in Section 3.)*

The information available is good enough to make this generalisation even though it is not perfect.

A) The angle information was not available in the British or South African papers. I had to work from the original UK x-rays and from new x-rays of tribal living Africans obtained for me by the South African authors. The British paper (6) did include x-rays of the flexed shape.

B) Mobility information was not included in the Iranian paper. I used the figures from the English group.

C) The mobilities of the South African group were full range extension to full flexion. The figures for the English group were standing shape to full flexion.

D) The full extension to full flexion figures for the young black South African group were very high at 20°plus at L4/5. However, the wedge shape of this joint in Fig2, from the same paper is 30° so this still maintains the wedge shape of the disk wide end anterior even in full flexion.

Conclusion to Section 2

In the lowest two joints of the human spine, the wedge shape should always be maintained, wide end anterior even in full flexion.(This, of course, refers to those free of back pain.)

The normal range of mobility of these lowest two joints of the lumbar spine is all on the extension side of what might be assumed to be the centre of the range for a spinal joint, the shape when the endplates are parallel to one another.

It would not be surprising therefore if flexion outside this "normal" range resulted in some level of abnormal feeling or pain. This however does not mean that the pain needs to be severe and disabling as back pain so often is. There needs to be a reason for Low Back Pain.

SECTION 3

Strength when Lifting.

This section explained the mechanical problem that exists in the spine for which the evolved solution is low back pain (LBP). The section extended to twelve pages originally.

Unfortunately, in the subsequent thirty years I have only met one medic, of any kind including orthopaedic surgeons, physicians, physios, chiropractors and osteopaths, who understood this mechanical argument and she (it would be a she) was an engineer in her first career.

So, on the basis that "everything should be made as simple as possible, but not simpler" much of this section has been rewritten to emphasise just the one vitally important point. (If, after re-reading, you still don't understand it, please ask an engineer, not another medic!)

Walking on two legs requires balance and control but does not put great mechanical stress on the spine. In bending and lifting, however, the forces are multiplied by ten or more (*16*). Many articles in technical journals confirm that lifting can stress the spine close to its limits (17, *18*, *19, 20*, 21, *22*).

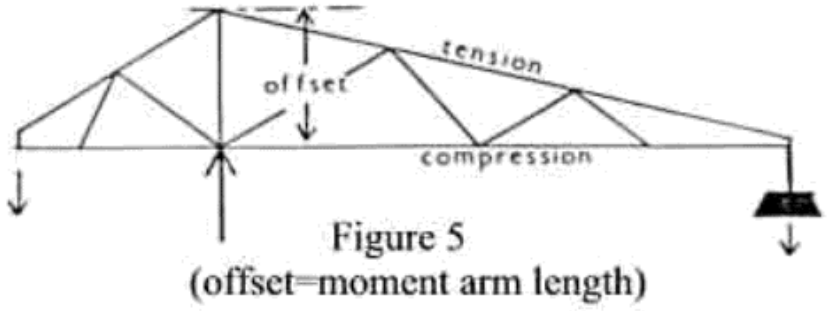


Figure 5
(offset=moment arm length)

In evaluating the forces on the spine in lifting, the body must be seen as a cantilever like the jib of a crane (Figure 5) There is a compression component and a tension component and the offset between them is critical to the strength.

The main compression component is the column of vertebral bodies and discs. There are several different tension components and there is considerable disagreement in the technical literature as to which are the most important.

There was disagreement at the time of writing in 1987 but the papers of Nikolai Bogduk (23.24) were already available making the mechanical structure entirely clear. The content of these papers is now included in his book, Clinical Anatomy of the Lumbar Spine and Sacrum (25,26). This book is now in its 5th edition.

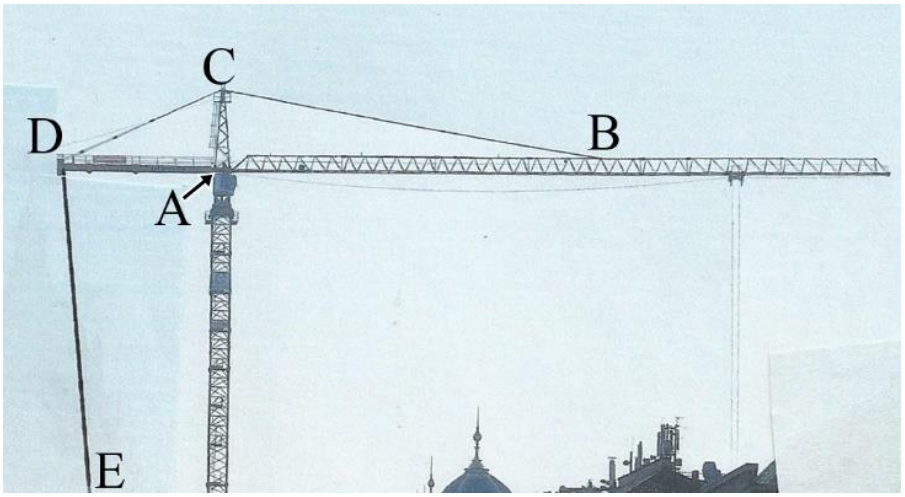
In the 1987 version of this paper, all spinal muscles were included in the analysis. This section of the original paper is now Appendix 1. Unfortunately, the detail did not clarify the problem for most people and is not necessary to the understanding. This is much clearer if we look just at the erector spinae aponeurosis (ESA). This is obviously the most important tension component of the structure in lifting because:

1) It is the most posterior and therefore has the greatest mechanical advantage.

2) It has by far the widest connections both at origin and insertion and is therefore the strongest.

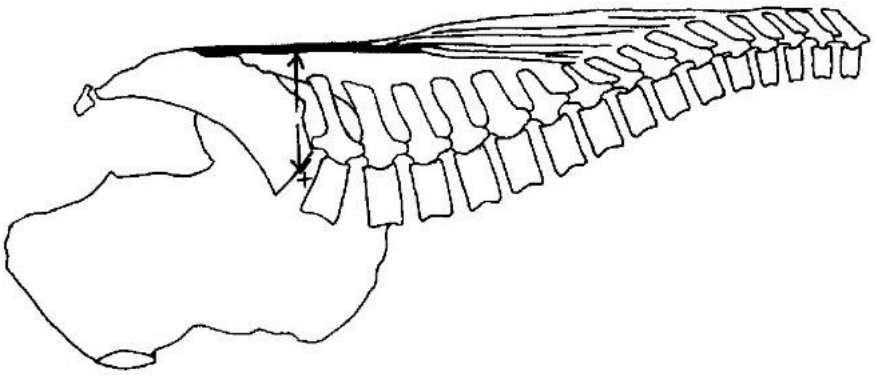
"Strength in lifting requires a tension member offset as far as possible behind (posterior to) the vertebral bodies. The prominence of the sacrum, necessary for the birth canal, is ideally placed and the erector spinae apponeurosis, (ESA), has its origin here, extending to a total length of 5 or 6 inches (12-15 centimetres) from one posterior iliac spine, down and across the most prominent part of the sacrum and up to the other iliac spine. In addition, the fibres are integrated with ligaments and tendons of the hip and thigh for direct transfer of forces. All beautiful design, as indeed it is at the superior (top) part of this structure where muscles connect to every thoracic vertebra and almost every rib. This spreads the load to the whole of the thorax.

Figure 6



The purpose of these two pictures is to show the location of the erector spinae aponeurosis (ESA) and the parallel with the tension part of a crane jib (the top cable, BC).

It is not suggested that lifting should be done with the standing shape of the spine. Nor am I suggesting that the other parts of the spinal musculature are not important. However, the ESA is obviously the most important part of the structure in lifting because it is the most posterior and has the widest connections at origins and insertions.



Evolution has cleverly used a shape defined by the birth canal.

Taking advantage of the prominence of the sacrum has produced a bipedal body with quite amazing lifting ability. All vital to our progress through hunting and gathering to building a civilisation.

The "Over-Centre Mechanism" Problem.

However, the spine is not rigid like the jib of a crane and the origin of the ESA on the prominence of the sacrum (and the posterior iliac spines) is below the flexible part of the spine. This makes the whole structure an "over centre mechanism" like the clips often used to close a suitcase or a mole wrench (a sort of spanner). This is a useful mechanism for a multitude of mechanical devices but exactly the sort of mechanism that you don't want in the jib of crane!

If we think about the detailed construction of the disc it is obvious what might happen. As flexion around L5 increases during a lift, the offset of the tension member, which connects to the sacrum, reduces. The compression on the disc increases. The flexion in the disc eventually applies tension to the posterior disc wall and the disc bursts extruding the nucleus straight onto the spinal cord or sciatic nerve roots. This was demonstrated, in vitro, by Adams and Hutton in 1984. (17) On the plains of Africa at any time in the last 5 million years this is an injury fatal to the lifter and dependent offspring. That is a strong evolutionary pressure to find a solution!

There isn't a mechanical solution without giving up the advantages of this design but pain can be used to limit behaviour. And it so happens, probably by chance that a solution presents itself and evolution has taken advantage of it. To "get the sacrum out of the way", the L4-5 and L5-S discs have become very wedge shaped. By about 20° on average.

Note; Actually, the angle from horizontal of the sacral endplate varies very widely in both sexes and all populations. This causes great confusion among researchers and often leads to the conclusion that shape doesn't matter. One detail does matter regardless of the other variations. To make this clear I base the following on average figures.

This average wedge shape of the lowest two disks of 20° is shown in (a) in Figure 7. The 20° at the two joints and 10° wedge shape of the L 5 vertebra make up the 50° sacral angle so that the superior and inferior endplates of L4 are approximately horizontal when standing.

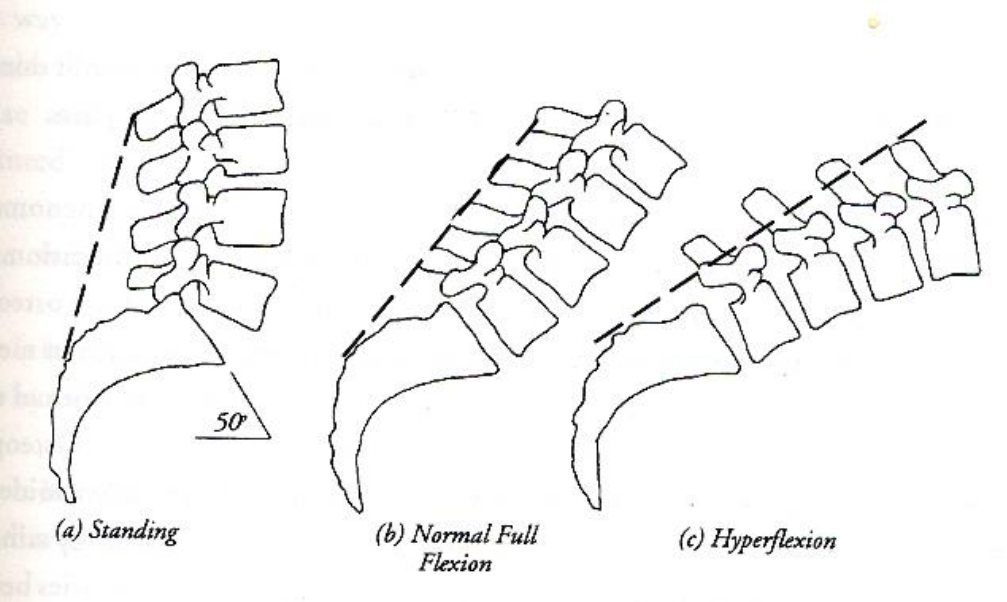


Figure 7

Diagram (b) would mean a flexion of 20° at each joint. This would still be strong. The line of action of the erector spinae apponeurosis (ESA shown as - - - - - -) is still just level with the spinous processes. As flexion progresses beyond the parallel-sided disc shape of diagram (b) towards shape (c), the offset of the ESA obviously becomes inadequate. The tension in this structure and the compression in the spine rise very sharply with flexion and the structure will collapse. This collapse will burst, or prolapse, the disk." (The last 9 paragraphs are from (27)

This is the mechanical problem that requires a solution.

Summary of Section 3

In normal lifting the maximum strength and the maximum offset is provided by the muscles of the back.

The erector spinae and multifidus muscles connect to the pelvis over all of the back of the sacrum and the posterior iliac spines with maximum attachment strength and direct connection to sacrotuberous and sacrospinous ligaments and to tendons of origin of hip extensors such as the gluteus maximus. (And via the sacrotuberous ligament to the hamstrings.)

The areas of attachment on the sacrum and iliac spines also give the greatest offset from the L5/S disc which can be achieved with this configuration of pelvis.

The configuration of the pelvis however is defined by the needs of childbirth. Thus, evolution has taken advantage of the prominence of the human sacrum to achieve a maximum offset for muscle attachment and therefore maximum lifting strength.

However, due again to the configuration of the pelvis and particularly the location, shape and angle of the sacrum, the large offset is seriously reduced by large angles of flexion in the spine. Flexion of L4/5 and L5/S to a shape which is perfectly acceptable for L1/2 or L2/3 (10° wedge with wide end dorsally) will reduce the offset by well over 60% and therefore the lifting strength also:

The whole structure is an "over-centre mechanism".

If strenuous lifts were performed with this flexion, serious damage would be very likely to occur at the lowest two spinal joints and in particular the conditions of flexion and compression would be applied to those discs which would produce a prolapsed disc in "in vitro" experiments. *(17)*

SECTION 4

The Shape Warning Pain

The loss of offset with flexion explains why the spine can suffer serious injury. Damage such as the prolapsed disc will occur if heavy lifting is performed with an excessively flexed lower spine.

However, prolapsed intervertebral discs are rare although severe back pain is common. There must be some protective mechanism which ensures that such serious damage does not occur.

Such protection does not seem to be provided by adequate ligaments. It is probably not possible to envisage such a system as it would have to duplicate all muscle connections to thorax, pelvis and elsewhere.

The alternative is a warning pain of sufficient intensity to force the person, or proto-person, to stop lifting or otherwise overstraining the spine.

There will always be warning signals from muscles and ligaments and the spinal muscles appear to be well endowed with such warnings. However, this does not cover the case of reduced offset due to flexion.

When the lowest disks are flexed beyond parallel sided, the reduced offset will not only make a prolapsed disc possible as explained earlier. It will also mean that the extra flexion can occur without sufficient muscular stretching to provide a warning from the normal tissue,stretch or damage pains.

A warning pain is required which is specifically associated with shape, with angle of flexion.

Pain Source

The actual source of the pain could be almost anywhere in the spine from any nerves which give the required geometrical information. There does not need to be any damage to the nerves. "Signals which sometimes announce an innocent event will at other times evoke pain*" (28).*

At the time of writing there was very little agreement about the source of the pain. This is clear in Bogduk's first edition (25) published just after this was written. However, the majority view that I detected at that time was to blame the facet joints. Dr Paul Sherwood's book "The Back and Beyond" published in 1992(29) certainly takes this view with multiple references. "My work over the past forty years has shown that by promoting the recovery of bruised facet joints, it is possible for a troublesome back to be permanently restored to good health."

Allowing the facet joints to recover from trauma was the motivation behind the surgical interventions of Dr Markwalder in Switzerland in that same year,1992. This very important work will be covered in Section 5

Despite this majority view, I was always of the opinion that the nerves in the posterior of the disk were the source of the pain and supported this with the following quote from Alf Nachemson from Spine in 1975:

"the intervertebral disc most likely causes the pain … pain resembling that which the patient had experienced previously could be registered only from the outer part of the annulus or nerve root" (21). There certainly are nerves in the annulus of the disc which could be the source of the pain (30). Ways could be suggested in which such nerves could "detect" the transition through the parallel-sided shape.

By 2007 however there was sufficient agreement and evidence for the following to be the opening paragraph in Spine of research at Aberdeen in Scotland based on upright MRI scans;

"Intervertebral disc problems, principally excessive migration of the nucleus pulposus, ---------- are generally accepted to be one of the main causes of nonspecific back pain". (31)

The MRI scans in this research show how the nucleus remains towards the anterior of the disk on extension and migrates posteriorly on flexion beyond the parallel sided shape. With the criterion for these two lowest disks, that they should not flex beyond parallel sided, the nucleus would never approach the nerves in the posterior annulus even if there were radial fissures.

As explained earlier, these two joints are very wedge shaped and flexion only "half way" to parallel sided still gives a perfectly adequate mobility.

The presence of a warning pain triggered only by the shape of the lowest lumbar joints would fit many of the known facts of back pain such as:

1. Much persistent and acute back pain is not associated with any tissue damage. "in 60% - 78% of cases no cause of the pain can be found" *(28)* and "the … patient is convinced that some tissue is damaged and … physicians and surgeons are convinced on the opposite" *(28).*
2. The severity of back pain even when there is no tissue damage. The pain has to be severe in order to stop the human being from lifting when the back is weak due to excessive flexion. This must have been even more true throughout our evolution when survival required energetic activity despite pain and discomfort.
3. The persistence of back pain long after any tissue damage should have healed. As many people know, back pain can be a lifelong problem though the intensity of pain usually abates eventually. There remains an ache and intermittent bouts of pain can be triggered by lifting or other activities.
4. The very severity of back pain is difficult to explain in evolutionary terms unless its purpose is to protect the person against worse damage such as a prolapsed disc.

Mechanical offset and Warning Pain

The spinal shape defined by the above criterion, that the lowest two joints should not flex beyond parallel sided, seems to correspond to a reasonable minimum offset for the erector spinae apponeurosis and other muscle groups. Figure 7a, b and c show clearly the loss of offset that occurs beyond the defined limit.

Figure 7b represents the flexion limit with L4/5 and L5/S parallel sided. The line is still clear of the L4 and L5 spinous processes. Further flexion to the shape of Figure 7c brings the line very close to the L5/S disc. This represents a serious reduction in offset and looks unacceptable.

This correspondence between the parallel-sided shape of L4/5 and L5/S and the minimum offset for safety is a coincidental effect of the shape of the sacrum. The sacrum was shaped primarily in order to maximise the pelvic inlet and outlet diameters for childbirth, but this shape seems to have been particularly suitable for the generation of a warning pain in the disc.

It is suggested that much persistent and acute back pain is a warning pain the L4/5 and L5/S discs when these dishcs flex or are able to flex beyond parallel sided. This has evolved to prevent actual disk prolapse.

Section Conclusion.

It was shown in Section 2 that the lowest two disks did not normally flex beyond parallel sided in groups or populations free of back pain.

Research at Aberdeen, that was mentioned in this section, Section 4, showed the migration of the nucleus anterior on extension and posterior on flexion beyond parallel sided.

The mechanical analysis of Section 3 showed how lifting with flexion beyond approximately parallel sided could cause prolapse.

Thus, the need for a warning pain and the mechanism of a warning pain, generated only by shape, have provided the conditions for the evolution of a pain severe enough to stop our ancestors, and ourselves, from continuing with any activity which is likely to cause an actual disk prolapse.

This is the evolution of low back pain.

SECTION 5

Civilised Sitting as a Cause

The previous sections have suggested that the L4/5 and L5/S discs should not flex beyond parallel sided. This is equivalent to saying that a minimum level of lordosis should be maintained at these joints in all circumstances (shape b in figure 7).

The limit to the flexion will be provided mainly by the ligaments of the spine. These will only come tight to limit the flexion occasionally in most activities but will be tight continuously in some sitting postures. Ligaments are collagen and "a collagen fibre creeps when it is stretched … for a prolonged period" (*32*). The ligaments can therefore be stretched by some sitting postures and the limit in flexion of some spinal joints can be increased. Sitting can therefore eliminate the lumbar lordosis completely and allow the L4/5 and L5/S joints to flex beyond the defined limits.

Many authors have concentrated on the extent and the importance of the effect of sitting on the lordosis (*33*, *34*, *35*, *5*). "The mechanics and statics of sitting have been carefully studied by Schoberth. He showed roentgenographically that, on a change of posture from standing to sitting, the pelvis is rotated on the average 40° and that this rotation is accompanied by a simultaneous compensatory kyphotic movement of the lumbar spine" (*35*).

In civilised sitting we frequently wish to have the spine upright and fairly straight despite the pelvis being tipped backwards by 40°or so. This will tend to concentrate the 40° into the lowest few joints of the spine, typically the critical ones, L4/5 and L5/S. This is particularly true in the working posture where the upper body may even be beyond the vertical and face down to the work, and in the vehicle driving posture where the upper body needs to be upright and active to control the vehicle. The truck driver needs the upper body to be even more vertical than the car driver.

Epidemiological studies confirm that these postures are associated with back pain. "Truck drivers are about five times more likely to develop an acute herniated lumbar disc than males who are not truck drivers" (11), and (referring to the same research) "it is suggested that prolonged sitting, regardless of whether it is in a motor vehicle, is detrimental, since length of time spent sitting on the job was in fact found to be associated with acute herniated lumbar disc. The relative risk for sitting while driving, however, was nearly twice as high as that for sitting in a chair regardless of the type of chair" (*35*).

These results were published in 1975 but recent work *(recent in 1987, that is*) "confirmed previous findings that motor vehicle transportation was an indicator for first time experience of LBT" (12). (LBT or Low Back Trouble includes all pain instead of the single factor of herniated lumbar disc.) It is suggested therefore that civilised forms of sitting, particularly the desk working posture, and the vehicle driving posture, are likely to increase the flexion of the L4/5 and L5/S joints beyond the defined limit and thus cause back pain associated with the shape warning pain.

It will undoubtedly be argued by the car industry that car seats have improved since that was written in 1987. Certainly, the construction, adjustments, upholstery and crash safety have improved and the comfort but the mechanical effect on the spine is worse.

There are two reasons for this;

1) Lumbar support, and adjustable lumbar supports, were introduced through the 1980's. It was claimed that this would stop the pelvis rolling back when sitting and maintain the lumbar lordosis.

As explained below in Section 8, lumbar support does not maintain the standing shape of the spine. It distorts it, flexing the lowest joints of the spine and extending or hyperextending upper lumbar joints. This applies even when the lumbar support succeeds in forcing the pelvis to the angle in the body that corresponds to standing.

The critical importance of this flexion of the lowest lumbar joints by lumbar support was not recognised at the time. An analysis of the seminal paper advocating lumbar support is at (36)

2) Around this time, in the 1980's, it was recognised that the effectiveness of seat belts was compromised by "submarining". This was the tendency for the lower body to slip under the lap part of the seat belt in a crash. It became important to ensure that the pelvis couldn't slip forwards and the solution was to tip the sitting surface backwards by about 15°. This tipped the thigh back by the same angle and also, to some extent, the pelvis. This further increased the flexion of the lowest joints of the spine.

The effect of car driving was noted as early as 1972 by Kelsey and Hardy (11), was confirmed in 1986 in Spine (12) and was strongly emphasised by the work of Professor Mark Porter, Professor of Ergonomics at Loughborough University UK who observed "-- -- a sixfold increase in -- -- low back trouble by car drivers who spend more than half the working day behind the wheel"(13)

Many researchers, particularly Dr Mark Porter, confirmed that the association of low back pain with driving has continued despite developments in seat construction in the 1990s and more recently.

Sacral Angle Variation

In the examination of X-rays as a part of this study one variation was observed which may help to explain the variation in the incidence of back pain among those with identical occupations (e.g. truck drivers). The variation in sacral angle was seen to be very large, both among the sample of non back sufferers used by Pearcy et al. (6) and among back pain sufferers in patient records.

Among the sample used by Pearcy et al (6) the extremes were 22° and 70°. (This is the angle between the sacral endplate and the horizontal with the pelvic position adjusted such that the anterior superior iliac spines are in the same vertical plane as the pubic tubercles.) These figures are necessarily only approximate because the whole pelvis was not included in the X-ray film.

A sacral angle of 70° will tend to make the lower lumbar joints very wedge shaped when standing. Much more flexion will then be required for these joints to reach the parallel-sided shape than if the sacral angle is only 22°.

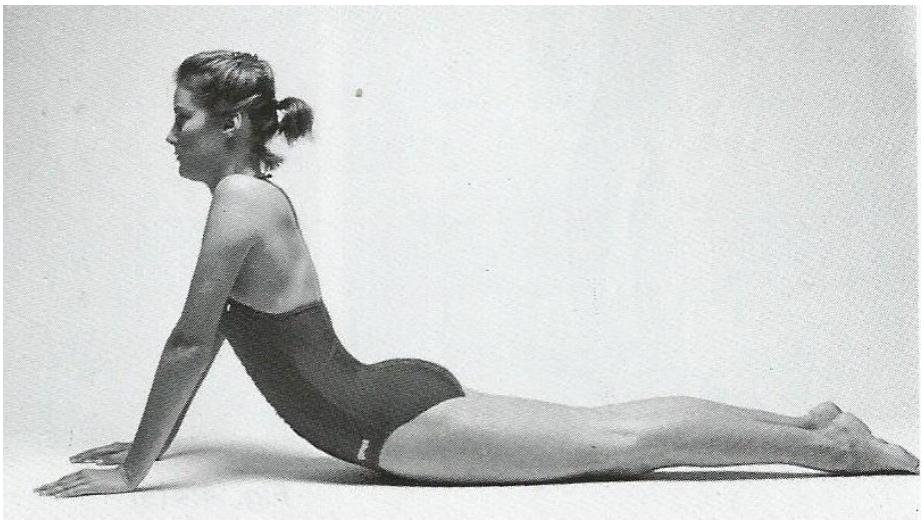
It would seem likely therefore that a larger sacral angle will make a person less susceptible to this form of serious back pain.

If, however, the large sacral angle is advantageous, the large variation is then surprising. It would be interesting to know whether a similar variation was found among the sample from a population chosen because of "the rarity of the disc syndrome" (1). This information was not included in that report.

As mentioned above, Prof Van Niekerk obtained X-rays for me from the same population. I can confirm that the variation is similar in that, more naturally living population in South Africa, to the English group.

SECTION 6 ***Figure 8***

Solution Number 1.
McKenzie Therapy



McKenzie will be known to any physiotherapist. This is the typical exercise of the McKenzie therapy which I refer to as the "Floppy press-up" Fig 8 is on page 45 of Ref (37).

It should be obvious that this exercise will extend the joints near the lumbo-sacral junction, and if done forcibly, the range of extension will be increased towards the natural limit in extension. This will squeeze the nucleus of the disk to the anterior of the disk and away from the nerves in the posterior annulus.

I trained in McKenzie with the McKenzie institute (UK) in 1989, the same year that I qualified as a chiropractor and have used McKenzie throughout my twenty-five years in practice. In most cases of low back pain of recent onset, it simply switches off the pain with ten or fifteen minutes of work and repetition. This applies to most reasonably active patients aged less than sixty or so.

The back does initially remain sensitive and painful on flexion. Time and persistence are necessary to become totally pain free. Very strict habits must be adhered to in sitting. For many it is just too much trouble in a normal civilized lifestyle to keep to a regime that will allow the back to return to really natural limits of joint mobility. In my experience very few people return to being as pain free as they were before the first problem.

On the other hand, one of the great advantages of this therapy is that the patient does all the work and, once learned, has the experience to deal with future episodes.

I look upon the McKenzie system as very strong evidence for the hypothesis of this paper.

SECTION 7

Solution Number 2

In 1987, I wrote "I suspect that surgical building of carbon fibre ligaments could achieve in half an hour, what has taken five years in my natural ligaments" (38) Over the subsequent thirty years I tried to interest many orthopaedic surgeons in this, but failed.

Recently I found that something very similar had been done in Switzerland between 1990 and 1995 with close to 100% success post op and still 75% success at five year follow up. A success rate not normally seen in surgery for low back pain. (And the failures over time are probably due to the well-known problem of the adjacent joint taking over the lost mobility! Adjacent joint syndrome.) Having identified which joint was responsible for the pain, Dr Markwalder had used Graf ligaments to immobilise the joint in its extended shape. (39, 40)

The motivation for this surgical intervention was the common belief at that time that trauma of the facet joints was the source of low back pain. I remarked on this belief in Section 4. Dr Markwalder believed that immobilization allowed recovery. (This is stated in his papers and was my understanding on a private visit to Switzerland.)

Subsequently it has become generally accepted that the source of low back pain lies in the disk as referred to in Section 4. By 2007 this was sufficiently general for the previously quoted introduction to a paper in Spine "--- excessive migration of the nucleus pulposus, ----- causes of nonspecific back pain". (31) and for the conclusions on page 204 in the 4th edition of Nikolai Bogduk's very comprehensive and authoritative book. (26)

This general agreement on the source of low back pain gives a complete explanation for the success of Dr Markwalder's intervention. It also removes the need for complete immobilization which was the reason for the Graf ligament. A far simpler ligament at the most mechanically advantageous and far less invasive position would achieve the necessary limitation of flexion to the natural evolved shape. I wrote fairly detailed suggestion for this implementation sometime in the 1990's. (41)

Figure 9

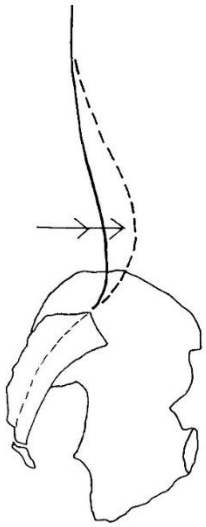
Figure 9 is one figure from that document. The prosthetic ligament would have to be strongly "integrated" with the supraspinous ligament at the tip of L4 and to the sacrum. This document is just preliminary suggestions in a field where I do not claim any experience or knowledge.

I believe from my years as chiropractor, my experience of McKenzie, my mechanical understanding of the problem and my own experience, that this simple surgical intervention could become a very widely implemented solution in the early stages of low back pain. Even where the pain level is acceptable, non-specific low back pain so often progresses to disk degeneration and further problems, so, as well as being a cure, this might prevent further degeneration.

SECTION 8
Solution Number 3

In Section 5 it was argued that civilized sitting distorted the spine and caused back pain. There is no clearer demonstration of this than Nachemson's seminal paper of 1976 in Spine advocating lumbar support. (5)

The tabulations in that paper, based on x-rays of people sitting with variable lumbar support, clearly showed that increased lumbar support flexed the lowest joints of the spine while hyperextending those near the lumbar-thoracic junction. (This point is explained in some detail on pages 10 and 11 of The Evolution of Low Back Pain (42). Again, if you still don't understand this point, ask an engineer, not a medic. But you will have to explain the structure first! They won't know what is inside their body!) This effect was not recognized by the authors of that paper.



This is exactly what is shown in this diagram and would be expected by anyone looking at the mechanics of sitting with direct support for the lumbar spine.

Obviously this will distort (flex) the lowest lumbar joints towards or beyond the parallel sided shape This effect was confirmed by the tabulations in that paper. An analysis of these tabulations appears at (36)

From the previous sections it should be clear that this will take these joints nearer to, or into, the range of flexion which will cause low back pain.
This is just one form of civilized sitting but is particularly relevant to the car seat which I have analysed in more detail elsewhere. (34,36,42) Many other sitting postures are also covered in detail in these references.The solution in this case must be to support the pelvis directly instead of supporting the lumbar spine.

Pelvic Support Not Lumbar Support.

Having understood this point, the solution is obvious. Any backrest must be designed to support the pelvis and not the lumbar spine. This principle was incorporated into my patent for Pelvic Support applied for in 1982 which expired in 2007 (43)

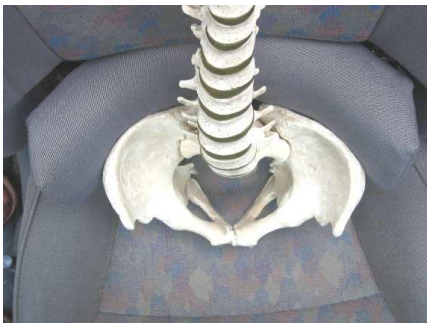


Figure 11
Prototype electrically adjustable pelvic supports in car seat. Note that the pelvic supports have to come right around the sides of the pelvis to support the highest point of the pelvis, the iliac crest.

Figure 12
Pelvic Support Office Chair in production from1995 till 2005 approx.
This or similar pelvic support office chairs may still be in production by Pledge Office Chairs of Leighton Buzzard, UK.

Figure 13
"Backseat." pelvic support car seat in production from 2000 till 2002 by Gorman Design Ltd.

There is no current production of this or other pelvic support products and I claim no design or patent rights. John Gorman

SECTION 9

The Sacro-Iliac Joint

There was no mention of sacro-iliac joint pain in my 1987 paper. This was probably because I didn't understand at the time, how the sacro-iliac joint caused pain including sciatica, in an episode of low back pain. I think it is fair to say that medical specialists and researchers still don't understand. Nikolai Bogduk's very comprehensive book, mentioned earlier, devotes twenty-four pages of the 4th edition to "Low Back Pain" and where it comes from, but the short "Summary" on page 205 effectively says "we haven't a clue!"

One of his comments in this section is particularly indicative of medical attitudes; "There are no data on underlying pathology that justify the belief that muscles can be a source of chronic low back pain." (26) (page 205) As I wrote earlier, medics think that pain must be caused by pathology and have no understanding of the problems caused by tight muscles. I also repeat another of my comments "the whole of alternative medicine is different ways of loosening tight muscles." In total contradiction to Nicolai Bogduk's statement, tight muscles are the source of most chronic back pain including sacro-iliac pain (as well as being the source of most of the income of most chiropractors!)

In explaining how this subject relates to low back pain, I can't do better than the start of chapter 2.2 of "The Evolution of Low Back Pain" (42) written in 1992 after five years in chiropractic, (two after qualification.) My experience after twenty-five years as a chiropractor confirms this interpretation but there is no medical research that I know of other than that mentioned in this chapter.

"Chapter 2.2 The Sublesation

Although the idea of a warning pain explains some aspects of back pain it does not explain the muscular spasms that are so often associated with back pain.

It seems very likely that these muscular effects are associated with a phenomenon that occurs in the spine of any animal that I shall refer to as a sublesation. The chiropractor refers to this effect as a "chiropractic subluxation" and the osteopath refers to it as an "osteopathic lesion". I shall avoid both words because in medical terms it is neither a subluxation nor a lesion because it is within the normal range of joint mobility and because there is no associated tissue damage. I have also avoided the word "fixation" because this includes cases where the joint is "fixated" by adhesions following trauma. The word sublesation should avoid all these ambiguities because it doesn't exist. It is a combination of the chiropractic term subluxation and the osteopathic term lesion. I hope that sublesation will eventually be accepted by both professions and also by the medical profession so that this important phenomenon can receive the recognition and research effort that it deserves. (Note; this was written in 1992. Progress so far? Less than zero. Most chiropractors don't even use the word subluxation any more because of medical opposition.)

In a sublesation local muscles around a joint are permanently energised by nerves that are in some way excited by the clamping effect of the muscles. Thus the effect is self-perpetuating. The mobility of the joint is greatly reduced and it is distorted asymmetrically because the muscle in spasm is only on one side. The osteopath and chiropractor both recognise the characteristics of the sublesation as asymmetry, loss of mobility and local muscle abnormality. The effect can occur in any spinal joint of any animal and has probably evolved in order to allow an injured joint to recover. (This phenomenon is the central point of chiropractic and is also central to osteopathy.)

When the warning pain has been triggered and there is disc damage or potential disc damage, the sublesation is ideally suited to provide muscular immobilisation of the vulnerable part of the spine. It seems likely that the phenomenon of the sublesation has evolved to be a very much more powerful effect in the human lumbo-sacral area. It also seems likely that evolution has used sublesations in the pelvis and sacro-iliac joints to cause muscle contraction in many of the muscles of the lumbar region." (42)

This muscle spasm is what so many back pain sufferers will recognise. It has to be powerful and painful because it has evolved to stop any very determined proto-human in the last five million years from continuing the activity that is likely to result in a prolapse from the disk straight onto the spinal cord or nerve roots.

The phenomenon of the sublesation applies at all spinal joints, including the four or five sacral joints that are no longer mobile joints. Any nerves exiting the spine can also be affected, so the nerve signal that initially comes from the disk can switch on sublesations in local lumbar or sacral segments. These can cause pain and muscle spasm in any tissues that get their innervation from the relevant segment. Hence sciatica without any physical nerve impingement. Evolution has incorporated all of this into a pain to protect the actual disk; a pain that even a determined proto-person can't ignore. And all of this occurs without any pathology!

Just one more anecdotal point; frequently the sacro-iliac pain persists long after the initial low back pain. If the initial pain signal from the disk has died away over time, then the sacro iliac locking and pain can persist. If so, it is easy for the osteopath or chiropractor to release the sublesation and "cure" the pain. I always say that most osteopaths and chiropractors make most of their money, and reputation, by releasing the sacro-iliac joints. It is therefore not surprising that many osteopaths and chiropractors believe that the real cause of low back pain lies in the pelvis.

Section 10. Conclusion.

There is one new thing that stands out from this interpretation of the shape, structure and warning pains in the spine.

This is not the effectiveness of McKenzie which I have experienced for thirty-five years personally and for twenty-five years as chiropractor. McKenzie was always controversial and seems, under pressure, to have "backed off" from the original forceful use of McKenzie extensions regardless of pain. In my experience, it will rarely be effective without forceful application and persistence.

It is also not the light that this understanding shines on civilised sitting with a backrest as the cause. Nor the exposing of the "schoolboy" error that has persisted for half a century, or much more, of failing to recognise that lumbar support makes the situation worse by flexing the lumbo-sacral junction. Correct pelvic support will still only be one useful tool in a complete solution.

The one thing that stands out is the explanation of why a simple surgical intervention twenty-five years ago had almost 100% success in curing back pain. It simply "switched off" the pain because there never was any pathology to cure! (at least initially.) History suggests that surgeons will be as successful in developing a simple, effective and permanent prosthetic supra-spinous ligament as they have been in the case of anterior cruciate ligaments in the knee.

Appendix 1. This was the submission 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. Changes and additions are in italics.

After submission in 1987 I remained in this field for the rest of my career. I am now 72 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.

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. (This, of course, in 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 posterio-lateral sectors of 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.)

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 twenty-nine 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!

I suggested twenty-nine, 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.

John D Gorman.
MA (Cantab.)
Chartered Engineer(UK). Member of the Institution of Mechanical Engineers(UK), Member of the Institution of Engineering and Technology(UK)
Registered Chiropractor 1989-2014

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

Appendix 2

This section was Section 3 in the original 1987 paper. The new Section 3, above, is simpler.

Strength when Lifting (1987 Version)

In order to estimate the offset, or moment arm length of muscles used in lifting, some categorisation of muscle groups and strengths must be made. Here again, there is considerable variation in the literature. Gracovetsky et al write "in the best case, the maximum moment contribution can be calculated to be about 250 Nm" (19) but McGill & Norman calculate a best value of 449 Nm (44). This is a difference of 80% between the values calculated in two detailed analyses by computer model. It is difficult for the reader to work through and check such analyses because the quantity of data and detail is not included in the published papers.

The analysis in this paper will concentrate on the offset between the compression and tension components at their connection to the pelvis. As with the crane jib this is critical to the lifting strength.

Method of Evaluating Offsets (moment arm length)

Offsets are taken from a scale drawing of the pelvis and spine in the sagittal plane. **Figure 6** is a full-scale reproduction of the central part of the drawing. The pelvis is shown in the normally defined standing position with the anterior superior iliac spines in the same vertical plane as the pubic tubercles. The spine is shown erect (shape 1) and with three different flexions. Shape 2 shows a flexion of about 5° per joint. Shape 3 corresponds to full flexion in the two papers discussed in Section 1 (1, 6). (The important characteristics of shape 3 are described in Section 3.) Shape 4 shows each spinal joint flexed to an extent that looks reasonable for the joint (10° - 15° per joint wedged with wider end dorsally).

In trying to select a "typical" pelvic shape from X-rays great variation was found, particularly in the size, shape, position and angle of the sacrum. A larger than average male subject was therefore chosen (182 cms – 80kg) who was known to be strong in lifting and free of LBP till the age 43 (but recently suffered an attack with no visible cause in X-rays). The X-rays were scaled down in ratio 5:4 approximately (film focal distance 100 cm), this ratio being confirmed by in vivo measurement of pelvic height. This subject is near the top of the height/weight range used by McGill & Norman (44) but lighter than competitive weight lifters (19). The sacral angle is 50° which is the angle shown by Jonck & Van Niekerk (1) in their Figure 1. The mean of the sample used by Pearcy et al (6) was 48° (estimated by this author from the X-rays of the subjects mentioned in Section 1. "The sacral angle" as used here is the angle of the sacral endplate from the horizontal with the pelvis aligned as defined above).

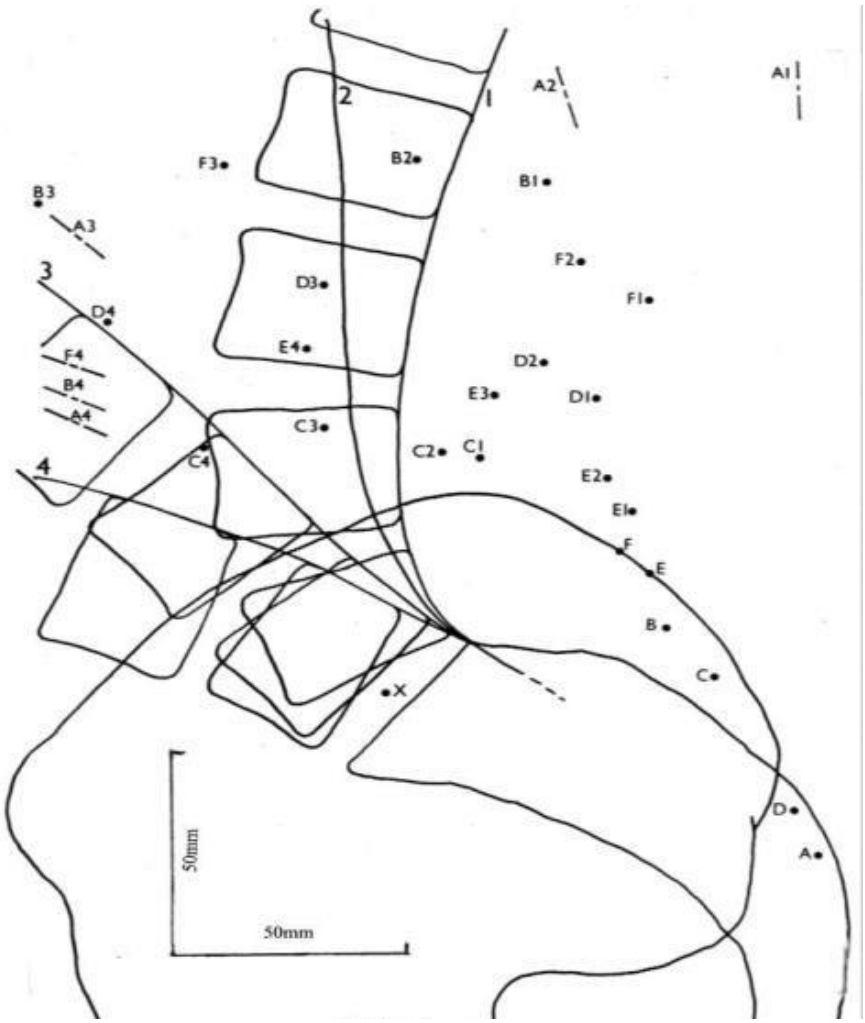


Figure 14 shows the lower right part of the drawing. The full sized (A0) drawing was used to evaluate offsets of muscle and ligament lines of action from the centre of the L5-S disc (X). This picture is reduced so that the whole drawing can be seen on the screen at the same time.

Shapes 1,2,3and 4 are of erect (1), slight flexion (2), full flexion (3) and hyperflexion (4).

Points A,B,C,D,E and F define origins on the pelvis of muscle or ligament groups defined in the text with the same letter

Point A1 defines the point of insertion of the muscle group A in the spine in shape 1 and similarly for muscle groups B,C etc and for shapes 2,3 and 4.

The lines of action are not shown for clarity and where a point (e.g. A1) is outside the range of Figure 14, the line of action is shown near the edge of the drawing.

Table1, after muscle group definitions, lists the offsets (moment arm lengths)

Lines of Action of Muscles and Ligaments

In separating the spinal muscles into groups the anatomical definitions of Bogdug's "reappraisal of the anatomy of the human lumbar erector spinae" (24) will be used. However it is useful mechanically to group these according to their connection points. The following groupings will be used.

Muscle Group A

Included in Group A are the muscles which connect the thoracic area to the sacrum. Muscular connection to the thorax is widely spread between all the ribs and the thoracic vertebrae from T12 to T6. Connection to the sacrum is via the erector spinae aponeurosis which is "ultimately attached to the lumbar and sacral spinous processes " (24).

The muscles are "the thoracic fibres of the medial division of the lumbar erector spinae" which "arise from the deep surface of the erector spinae aponeurosis" (24) together with "the thoracic fibres of the lateral division of the erector spinae" which "arise from the erector spinae aponeurosis (esa)" (23).

The esa connects onto the dorsal surface of the sacrum and the sacral spinous processes and also onto the posterior iliac spines. However, "those (muscles) to successively more rostral (thoracic) levels arise progressively more medially across the erector spinae aponeurosis" (23). The point of origin of the esa in Figure 6 has therefore been placed on a line from the most prominent sacral spinal process to the posterior iliac spine but nearer the sacral process in a ratio 2:1.

The point of origin on the thorax has been chosen to be on T9 (central between T6 and T12) and half way between the transverse processes and the most dorsal point on the rib. This point is A1 for spinal shape 1 and A2, A3 and A4 for spinal shapes 2, 3 and 4 respectively. Because these points are outside the size of Figure 6 they are shown on the line of action near the edge of the drawing.

Muscle Group B

This muscle group includes those muscles that arise from the ilium via the lumbar intermuscular aponeurosis (lia) near the posterior iliac spine. They connect to the accessory and transverse processes of L1, L2 and L3.

A common line of action to L2 is shown in Figure 6 as B1, B2, B3 or B4 for spinal shapes 1, 2, 3 and 4 respectively. B2, B3 and B4 are outside the range of Figure 6 and are shown on the line of action near the edge of the figure.

The muscles are: The lumbar fibres from L1, L2 and L3 of the medial division of the lumbar erector spinae together with the lumbar fibres from L1, L2 and L3 of the lateral division.

Muscle Group C

These muscles arise directly from the ilium near the posterior iliac spine and connect onto the accessory or transverse processes of L4 and L5.

The origin is shown as point C on the medial surface of the iliac crest near the posterior iliac spine and the insertion is shown on L4 as point C1, C2, C3 and C4 for spinal shapes 1, 2, 3 and 4 respectively.

The muscles are those lumbar fibres of the medial and lateral divisions of the lumbar erector spinae which connect to L4 and L5.

Muscle Group D

This is the multifidus. "In the sacral region, the fasciculi arise from the back of the sacrum as low as the fourth sacral foramen from the aponeurosis of origin of the sacrospinalis, from the medial surface of the posterior superior iliac spine and from the posterior sacro iliac ligaments" (45). This would suggest an origin similar to point A but with more connection to the posterior iliac spine. Point D is shown above point A.

It is assumed that L2, L3, L4 and L5 will have direct connection and D1 is shown half way between the midpoints of the spinous processes of L3 and L4. Connection points are D1, D2, D3 and D4 for spinal shapes 1, 2, 3 and 4 respectively.

Ligament Group E

This is the L4 to iliac connection of the deep lamina of the posterior layer of the thoracolumbar fascia as described by Bogdug & Macintosh (23). The point E has been estimated roughly by transferring the ratio of vertical heights in the photograph Figure 4 in their report to the Figure 6 here.

This is the only direct connection from the supraspinous ligament to the pelvis. The supraspinous ligament is continuous and strong from the cranium (as the nuchal ligament) down to L4. However, surprisingly, "the ligament end(s) between L4 and L5" (23). This is confirmed by Risannan (46).

The supraspinous ligament does not reach L5 or the sacrum and for this reason the corresponding lamina running from L5 to the iliac crest will not be considered in this analysis.

Ligament group E connects to the tip of the L4 spinous process at E1, E2, E3 and E4 for spinal shapes 1, 2, 3 and 4 respectively.

Ligament Group F

These are the individual connections via the lateral raphe from the spinous processes of L3 and L2 to the iliac crest (23).

The position of the connection point F on the iliac crest was estimated as described for E above.

The vertebral connection points F1, F2, F3 and F4 for spinal shapes 1, 2, 3 and 4 respectively are placed at a point half way between the tips of the spinous processes of L2 and L3.

It should be noted that this ligament connection can only be effective at all if there is a tension in the abdominal muscles to hold the lateral raphe in place. This may significantly affect the line of action but this has not been considered.

Figure 6 shows these points for all muscle and ligament groups. For clarity lines of action are not drawn.

The compression component, the column of vertebral bodies and discs, connects to the pelvis at L5/S" and point X marks the centre of this disc. The offset from X of the line of action of each group is listed in Table 1. (Lines of action are taken as straight except where these will pass through bone.)

Table 1 **Offsets in mm**	**Shape 1** **(erect)**	**Shape 2**	**Shape 3** **(normal full flexion)**	**Shape 4** **(hyper-flexion)**
Muscle Group A	92	78	39	30
B	62	61	50	39
C	54	52	45	35
D	67	66	51	33
Ligament Group E	62	63	63	57
F	46	53	61	51
Mean of Muscle Groups **(A, B, C and D)**	69	64	46	34

Results

1. The largest offset is achieved by the muscle group A. This group makes direct connection from the prominent sacrum to the thorax.
2. The offset for all muscle groups is reduced by flexion and very seriously reduced in flexion beyond shape 3. This result is very clear and is not dependent on the detailed shape of the pelvis or the accuracy of the drawing.
3. The multifidus muscle, group D, has the second largest offset.
4. The offset reduction from shape 1 to shape 2 is small compared with that beyond shape 2 or 3.
5. The offset of the ligament groups E and F is greatest at shape 3 and is only reduced slightly between shapes 3 and 4.
6. The mean muscular offset in shape 2 (64 mm) is close to the figure of 61 mm from McGill & Norman (44).

Discussion of Results

Result 1

It is clear that the maximum offset can be achieved by muscle group A. This is the muscle group that arises from the back of the sacrum via the esa and connects over the whole of the back of the thorax to ribs and vertebrae.

From the "jib of a crane" analogy one would expect this group to provide a substantial part of the whole strength of the spine in lifting. In the crane jib there will always be a tension member which runs from near the support (with maximum offset from the compression member) to meet the compression member near the point of attachment of the load. (Note that connection on the vertebrae is not to the spinous processes but to the transverse processes. This is what one would expect in order to bring the line of tension close to the line of compression, the vertebral body, near the support point of the load.)

The probable importance of this muscle group is also suggested by the very good spread of muscle connections to the thorax. Eleven ribs have muscle connections and seven vertebrae (23).

In the structure of the pelvis there are also reasons for expecting muscle group A to be a major contributor to lifting strength. These are as follows:

1. Group A connects to the sacrum via the esa which "blends with the sacrotuberous and sacro iliac ligaments. Some of its fibres are continuous with the fibres of origin of the gluteus maximus" (45). The arrangement must substantially reduce the bone strength required in the pelvis.
2. All the compressive force in the vertebral column will be transferred directly to the sacrum. Any part of the tension force which is applied to the sacrum will not result in a shear force in the sacro iliac joint. Any part of the tension force which is applied to the iliac crest or posterior iliac spine will appear also as a shear force in the sacro iliac joint. Thus any evolutionary transfer of muscle tension to the sacrum rather than the ilium will reduce the strength required in the sacro iliac joint to withstand shear forces. This might possibly have additional importance because of the need for the sacro iliac joint to separate slightly during childbirth as noted in Section 1.

Muscle group A is thus of considerable importance both in the strength of the spine and of the pelvis. It is not however the only muscle of importance. Muscle groups A, B, C and D can all have offsets in excess of 54 mm and will together provide the tension components of the spine required for lifting.

Together these muscles connect to the whole of the area of the back of the sacrum and to the medial surface of the posterior iliac spine area of the ilium. The possible surface for muscular connection is also greatly increased by the presence of the aponeuroses, esa and lia, the former having direct ligamentous connection to hip joint extensor tendons.

Thus, the muscle groups considered here make maximum use of the whole of the back of the pelvis, both sacrum and iliac spines, to achieve the most substantial muscular attachment with maximum offset.

Result 2

There is, however, a problem with this arrangement. The offset is seriously reduced by flexion of the spine. If the mean of the geometrical offsets of muscle groups A, B, C and D is taken (giving equal important to each muscle group) then the strength in shape 4 is reduced to only 49% of that in shape 1. This must make serious damage to the spine possible.

If, in attempting to lift a heavy weight, the offset becomes seriously reduced, the muscles may not be able to limit the flexion of the lowest lumbar joints. The supraspinous ligament will limit the flexion at all joints above L4 but this does not exist at L4-5 and L5-S (46, 47). A constant weight and tiring muscles may allow further flexion. Alternatively extension of the strong hip joint may flex L4-5 and L5-S instead of raising the weight. Whatever the reason, further flexion at these joints will reduce the offset even more and reduce the bending force that they can withstand. The resulting further flexion will again result in a reduced offset and reduced resistance to flexion.

The disc at the relevant joint will be subjected to very high compression while its flexion increases. This combination of flexion and compression is exactly the combination used "in vitro" to produce the "slipped" or prolapsed disc (17). Ligaments such as the capsular ligaments will have too small an offset to stop further flexion and may be damaged.

The interspinous ligament has a slightly greater offset but it runs obliquely posterocranially (46, 47) which reduces its strength considerably and it will also be liable to rupture in such a case.

The reduction in offset with flexion explains many of the serious injuries of the lower spinal joints, particularly the prolapsed disc. This simple mechanical problem seems to confirm the common preconception that lifting with a bent back is dangerous and liable to result in a back injury.

Result 3

The multifidus muscle (group D) has the second largest offset and is probably of great importance in protecting L4/5 and L5/S. This may be significant in view of the statement of Mattila et al (48) that "we are willing to consider that the small size of the type 2 fibres (in the multifidus) indicates atrophy both in the patients and in the controls … A possibility is that sedentary people in modern society do not use their multifidus muscles in such a way that the type 2 fibres would retain their normal size".

Result 4

The total offset in shape 2 is reduced by 9% only and may therefore be perfectly adequate for heavy lifting. It is not suggested that the spine must be kept fully extended for maximum weight lifting, particularly if the compressive strength of vertebrae can be increased by some degree of flexion as suggested by Adams & Hutton (3). The greatest loss of offset occurs as shape 3 is approached.

Result 5

The role of the ligament groups E and F will be discussed under the heading Contra-Arguments below.

Conclusion from Results

It seems therefore that the muscular groups considered provide a very substantial tension component for the structure of the spine as a cantilever. Muscle connections spread over the whole of the back of the thorax and the spine itself and make connection to all parts of the back of the pelvis with additional strength from aponeuroses which are continuous with ligamentous connections to hip extensor tendons. The attachment area on the back of the pelvis also provides the maximum offset for these muscle connections and this is maximised by concentrating some connections on the sacrum which is prominent for obstetric rather than mechanical reasons.

The disadvantage of this arrangement is that the high offset is only achieved if the spine is not flexed fully. An angle of flexion which would be perfectly reasonable for the spinal joints (10° - 15° wedging in flexion per joint in shape 4 will reduce the offset of all these muscle groups by more than half.

If a person lifts a weight with a spine approaching this degree of flexion there is a serious danger of tearing ligaments near the joint (capsular or interspinous) and of prolapse of the intervertebral disc itself.

Evolution has taken advantage of the prominence of the sacrum because this maximises lifting strength but has had to accept the consequent reduction in strength on flexion. No evolutionary reshaping of the pelvis was possible because the shape of the whole pelvis is mainly defined by obstetric considerations.

Contra-Arguments

This consisted mainly of criticism of Gracovetsky's papers in Spine in the early 1980s because they were still current in 1987. These proposed mechanisms of lifting were shown to be non-existent by 1990 so my contrary arguments are not included here.

Appendix 2

Prolapsed intervertebral discs are rare although severe back pain is common. There must be some protective mechanism which ensures that such serious damage does not occur.

Such protection does not seem to be provided by adequate ligaments. To provide full protection the strength of such ligaments would need to equal or exceed the strength of the muscles. They would have to have the same or greater offset and the same or equally substantial connections. This does not seem to be the case for ligamentous connections to the pelvis.

In considering lifting strength in Section 2, little mention was made of ligaments near the joint such as the longitudinal, capsular or interspinous ligaments. An example will show why these are of little use to lifting strength. If, in a fairly heavy lift, the muscle moment at L5 is 500 Nm with an offset of 50 mm, then the compression on the disc is 10,000 N or about 1 tonne. This is within the compressive strength of the vertebrae. If, while still lifting, the spine flexes such that the capsular ligaments come tight with offset of 25 mm and take a tension equal to the muscle tension then muscle and ligament tension will be reduced to two-thirds or a tonne. However, the vertebral compression will be increased to 1.3 tonnes which would probably exceed its capacity. Since the compressive strength of vertebrae is one of the known limitations on lifting strength the capacity has actually been reduced by involving such ligaments in lifting. For maximum lifting strength the offset is the most important factor.

The longitudinal, capsular and interspinous ligaments will of course protect the disc from excessive flexion in most activities but will themselves be liable to damage in flexed weight lifting. In his sample, Rissanen found ruptures in the interspinous ligament "in 21% of persons over 20 years of age". "All were in the lowest 3 lumbar interspinous spaces" (46) (93% of these in the lowest 2 spaces).

It seems that neither the supraspinous ligament with its fascial connection to the pelvis nor the smaller ligaments between adjacent vertebrae can provide an adequate ligament system to protect the lumbar spine in weight lifting. It is probably not possible to envisage such a system as it would have to duplicate all muscle connections to thorax, pelvis and elsewhere.

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