Mobility – Even in the Lumbar Region?

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Aright. I did it this time. This will get me banned off Eric Cressey's Christmas card list, pull Dr. McGill away from his study of belly dancers–seriously, I did not make that part up, check the reference), and send most "personal trainers" running for the hills (cue the Iron Maiden music).

Yep, I am talking about LUMBAR FLEXION and EXTENSION. Holy crap, I just suggested that you should move your low back and for some of you (not the readers of this site--way too bright for that) are thinking that I have lost my friggin’ mind now and confirmed that I really am out on a weekend pass from the funny farm.

Here is why I believe you should move your lumbar spine.

1) I am paraphrasing Geoff Neupert here, "If you were not supposed to move it, there would be a BONE there instead of a joint" The premise is that you were designed with a joint there, so you move it!
You should never move into pain, but pain free movement is good! Maybe this means you need to move slower, cut the range of motion or in extreme cases cut the loading (work in a pool for example). Either way, the body was designed for movement.

Living systems are built up with use, and ATROPHY with disuse.

This is the direct opposite to anything else. If you built a bridge and stuck it in a vacuum, it would last forever. Shoot someone up into space, confine them to bed rest, or sever the nerve supply to muscles and they will atrophy like crazy. Countless studies show that one of the WORST things is space flight (due to zero gravity and the unloading effects) and bed rest!

**What is normal?**

There is a debate about what is considered a "normal" ROM (range of motion) for the lumbar (low back) area. Zigler, J et al. (15) stated, “a normal ROM at the implanted level (for L3-L4 and L4-L5 between 6° and 20°; for L5-S1 between 5° and 20°).

Herp et al (14) compiled a nice table for range of motion in degrees in 20-30 year old from 5 different studies. Click here for the study

Denoziere, G et al. (1) stated “The rotational mobility offered by the device is limited to 12° in flexion, extension, lateral bending and is not limited in axial rotation.” This study was done to investigate the normal ROM for a computer model.

The take away is that none of them said ZERO for a ROM. Strike 1
What else you got?

An extreme case would be zero ROM and lots of ROM. Well, that type of well controlled study is hard to find, but if you look in the biomedical engineering literature you can find some neat stuff.

Zigler, J et al. (15) did just that in a prospective, randomized, multicenter (all good words for studies!) FDA (Food and Drug Administration) investigational device of a disc replacement versus fusion for the treatment of 1-level degenerative disc disease. So we have one case with some movement (artificial disc) and another case with no motion (fusion). Not the best since we have to jam this foreign object into someone’s back, but it is a start.

Keep in mind, that for this study what they define as “success.” “By the FDA definition in this study, ROM success required greater motion at 24 months than at preoperative baseline for investigational patients. Using this analysis, 89.5% of investigational patients were clinically successful." (15) We all know that it just not as simple as a ROM test 2 years later, but in the realm of this study, it is a “success.”

To get a new medical device approved, you need to show that your new widget is better than the FDA approved widget/therapy at that time. In this case that is spinal fusion!

The study (15) showed a trend towards less pain (done by VAS--Visual Analogue Scale pain score) in the disk group, but the patients were still in a fair amount of pain at 24 months with a small (although statistically significant) reduction. Hmm, slightly better ROM and still in pain after 24
month—welcome to state of the art! Realistically, this just shows that pain is a huge, really complicated area.

**Spinal Proprioception?**

There is also a hypothesis that spinal proprioception may play a role in modulating protective muscular reflexes that prevent injury or facilitate healing. That would make sense that the body would want to protect the spine at all costs.

Feipel et al. and others (2, 9-11) has shown a loss of proprioception in patients with chronic low-back pain, although not conclusive (5). I saved you a diatribe on each study, but some very fascinating stuff.

2) Mechano vs noci (what the hell is he talking about now?)

As the spine gets more “locked down” and approaches a more fusion type state, there is evidence to support the idea that there will be an increase in the number of nociceptors and a decrease in the number of mechanoreceptors. I remember Dr. Cobb mentioning this once also. So why do you care?

Mechanoreceptors are little guys (ok, not really but go with me on this) that live in the joints (and muscle) to monitor mechanical forces. Now there are all sorts of flavors of them, but we will keep it general for now and just call them mechanoreceptors.

Nociceptors are little guys that monitor noxious (bad) stimuli. Now, a noxious stimulus may or may NOT be painful; but it’s generally viewed as bad and can lead to pain. Remember, pain lives in the brain and that part get complicated really fast.
So, if the number of mechanoreceptors are going down we get less info about the environment and if the nociceptors are going up, there is an increased chance of noxious stimulation. Sounds cool in theory, but are there any data?

Roberts, S et al. (13) at first glance looks like a killer study, but there were no controls; so the data is not really useful—drat. McLain, RF et al (6-8) has completed some interesting studies looking at concentrations of mechanoreceptors around the spine and shown that there are more in the cervical area than any other area. That makes sense, look how much more you can move your head than your lower spine! As Mc Lain, RF (6) states “The predominance of receptors in the cervical spine is consistent with its greater mobility, the need to accurately position the head in space, and the need for coordinated muscle control for protection and posture.”

**We have a bingo!**

Onodera T et al (12) did a great study looking at the density and distribution of neural endings in rabbit lumbar facet joints after anterior spinal fusion and to evaluate the effects of intervertebral immobilization. The author states, “These results suggest that immobilization of the intervertebral segment causes a reduction in the number of mechanoreceptors in the facet joint capsules because of the reduction in mechanical stimulation. Moreover, in the upper adjacent facet joint there may be neural sprouting caused by nociceptive stimulation.” This is further evidence (in an animal model) that the body will remodel in possibly a negative way to immobility. Is that really that far of a reach?
Now, one study does not “prove” anything and neither does a collection of studies, but it goes give us evidence toward the right direction as long as we are asking the correct questions.

**What if everyone is talking about the same thing?**

Johansson, H et al (3, 4) have found a close relationship between activation of joint mechanoreceptor and stimulation of the gamma efferents (to sensitize the spindles) which results in increases in muscle "stiffness" and joint stability. Now the work of Johansson was done on knees, but the same principals probably apply.

**So maybe our end result is more muscle “stiffness” but we need to TRAIN MOBILITY to get there?**

Mc Lain, RF states (7) “Previous studies have suggested that protection muscular reflexes modulated by these types of mechanoreceptors are important in preventing joint instability and degeneration”

In English, this means you should move your dam lumbar spine! Please discuss.

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References


