Uneven Intervertebral Motion Sharing is Related

to Disc Degeneration and is Greater in Patients

with Chronic, Non-Specific Low Back Pain: An

in Vivo, Cross-Sectional Cohort Comparison

of Intervertebral Dynamics Using

Quantitative Fluoroscopy

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PURPOSE: Evidence of intervertebral mechanical markers in chronic, non-specific low back pain (CNSLBP) is lacking. This research used dynamic fluoroscopic studies to compare intervertebral angular motion sharing inequality and variability (MSI and MSV) during continuous lumbar motion in CNSLBP patients and controls. Passive recumbent and active standing protocols were used and the relationships of these variables to age and disc degeneration were assessed.

METHODS: Twenty patients with CNSLBP and 20 matched controls received quantitative fluoroscopic lumbar spine examinations using a standardised protocol for data collection and image analysis. Composite disc degeneration (CDD) scores comprising the sum of Kellgren and Lawrence grades from L2–S1 were obtained. Indices of intervertebral motion sharing inequality (MSI) and variability (MSV) were derived and expressed in units of proportion of lumbar range of motion from outward and return motion sequences during lying (passive) and standing (active) lumbar bending and compared between patients and controls. Relationships between MSI, MSV, age and CDD were assessed by linear correlation.

RESULTS: MSI was significantly greater in the patients throughout the intervertebral motion sequences of recumbent flexion (0.29 vs. 0.22, p = 0.02) and when flexion, extension, left and right motion were combined to give a composite measure (1.40 vs. 0.92, p = 0.04). MSI correlated substantially with age (R = 0.85, p = 0.004) and CDD (R = 0.70, p = 0.03) in lying passive investigations in patients and not in controls. There were also substantial correlations between MSV and age (R = 0.77, p = 0.01) and CDD (R = 0.85, p = 0.004) in standing flexion in patients and not in controls.

CONCLUSION: Greater inequality and variability of motion sharing was found in patients with non-specific low back pain (CNSLBP) than in controls, confirming previous studies and suggesting a biomechanical marker for the disorder at intervertebral level. The relationship between disc degeneration and MSI was augmented in patients, but not in controls during passive motion and similarly for MSV during active motion, suggesting links between in vivo disc mechanics and pain generation.

KEYWORDS: Back pain; Diagnosis; Fluoroscopy; Kinematics; Spinal injuries

From the FULL TEXT Article:

Background

Concepts of lumbar spine stability cover a range of complexity. Intervertebral angle change (IV-RoM) is not now thought to be very useful, due to wide population variations, although range of translation is generally preferred by spinal surgeons who assess for instability. [1] These measures, although of questionable validity, are nevertheless accessible from plain radiographs. To probe more deeply and investigate more subtle forms of instability, continuous intervertebral motion measures are needed. [2–5]

For the assessment of mechanical destabilisation of the spine caused by injury, the intervertebral neutral zone is thought to be the most sensitive measure. [6] Although its measurement has been generally confined to cadaveric studies, the advancing sophistication of quantitative fluoroscopic systems (QF) is beginning to provide a surrogate in vivo measure in the form of slope of the intervertebral rotation-time curve (also known as the attainment rate). [7–10]

Chronic, non-specific low back pain (CNSLBP) is widely considered to be at least partially of mechanical origin, due to its susceptibility to movement and position. It is also considered to be related to intervertebral disc degeneration. [11] However, no reliable diagnostic tool that could help a clinician to determine if a disc is the source of the pain in patients with chronic LBP is currently available. [12] Instead, relationships between trunk myoelectric activity, co-ordination and directional preference are more prevalent in the clinical biomechanics literature. [13–15] However, without an assessment of the relationship between segmental mechanics and pain, identification of biomechanical markers in CNSLBP will remain elusive.

While the in vivo investigation of intervertebral loading is still problematical, kinematic studies are becoming more common. [16] The lumbar spine is a kinetic chain that requires the sharing of motion between levels during bending. Various aspects of intervertebral motion sharing have been investigated in cadaveric studies [17–19] in plain radiographic studies in vivo [20–24] and in continuous radiographic studies. [3, 5, 8, 20, 25–30] Most of these have studied motion onset and displacement; however, two that studied displacement [4, 24] and one that studied pattern variations [29], found differences between patients with CNSLBP and controls.

Intervertebral motion pattern variation studies are of interest because they provide more information than end of range studies and can be more readily applied to contemporary concepts of spine stability. Reeves and Cholewicki [31] identified impaired restraint and performance in the passive and active intervertebral subsystems as subset measures of subtle instability, where restraint is the ability of a system to resist an imposed perturbation and performance is the ability to return to the original position once the perturbation has been removed. In the lumbar spine, the average range of differences in the sharing of motion by each intervertebral level over the sequence reflects the inequality of restraint across levels (MSI) (Fig. 1). Its variability throughout the motion (MSV), may be considered as an expression of intervertebral motion control.

In assessing the possible role of intervertebral motion sharing in CNSLBP, Mellor et al. [29] investigated the variability of recumbent intervertebral passive motion sharing and found it to be greater in patients with CNSLBP than in controls. Therefore, it was thought as a possible biomechanical marker for CNSLBP. These studies only addressed motion sharing variability (control) and not equality of restraint—and their replication has not been attempted until now. They also did not account for the effects of disc degeneration. We, therefore, attempted to replicate these studies, adding a measure of motion sharing inequality, investigating motion under load and incorporating disc degeneration as a possible source of interaction with these measures.

Discussion

In recent years, the usefulness of dynamic analysis of spinal disorders has become more apparent. For example, Shiba et al. [34] found that by examining dynamic, as opposed to static global sagittal alignment at the beginning and end of a gait sequence in patients with degenerative lumbar kyphoscoliosis, loss of global sagittal alignment at the end of the gait sequence was more readily detected.

The present studies bring to three the number of cohorts in which uneven continuous motion sharing has been found to be greater in CNSLBP patients than in controls. In the present study, uneven passive restraint across the lumbar spine (MSI) was greater in patients with CNSLBP than in pain free controls. There would seem to be at least two possible explanations for this. The first is that unequal restraint (MSI) could add to increased muscle metabolic demands during activities of daily living causing recurrent muscle pain. [35] The second may relate to the rapid accelerations associated with inadequate restraint at an injured level, which has been suggested to be a nociceptive pain generator producing a single point of pain. This could also cause motion sharing inequality. [36, 37]

It is notable that age and disc degeneration were substantially correlated with uneven passive motion sharing (MSI) in the back pain patients and hardly at all in the controls in Cohort 1. This suggests that CNSLBP is linked to disc degeneration when there is uneven restraint in the passive subsystem. Barz et al. put forward a new conceptual model of CNSLBP that links such structural degeneration with mechanical compensation and stability. [38] Thus, more evenly shared restraint (MSI), despite the presence of degenerative change, may be seen as the result of structural compensation that allows the individual relative freedom from symptoms.

In contrast, in weight bearing active flexion in patients, the correlations found between age, disc degeneration and increased variability of motion sharing (MSV) suggests relationships with control in the active subsystem. However, the finding that motion sharing variability (MSV) was not greater in patients during active weight bearing motion may have been due to the stabilising influence of the trunk muscles during active bending. Thus, control of MSV may be an important factor in the avoidance ofCNSLBP.This possibility could be explored by future research using the above techniques in combinationwith electromyography. [39] However, Von Forell et al. found, using finite element modelling, stresses on the spinal holding elements would generally be lower when not all lumbar discs are degenerated. [40]

It is uncertain whether these relationships are causative or consequent to back pain, or both. The above suggested relationships to rapid accelerations and/or increased muscle metabolic demands are possible explanations for a causative effect. For example, in the recumbent studies, it is difficult to conceive how pain alone could have selectively affected passive segmental restraint when muscle electrical activity was minimal. [7]

Recent research by Lao et al. [41] supported the hypothesis of Kirkaldy-Willis [42] that disc degeneration has different effects on intervertebral motion at different stages. Contemporaneous discographic and profilometry studies have supported the hypothesis that painful discs are also usually disrupted. [43] The strong associations found here between disc degeneration and uneven intervertebral motion in patients, but not in controls, seem consistent with this. It is also consistent with other recent research, which found that disc degeneration was associated with low back pain, especially when associated with end-plate signal change. [11, 44, 45] However, unlike the present work, these studies used MRI disc degeneration grading, while radiographic grading based on structural aspects rather than biochemical changes may be better correlated with pain when considered alongside intervertebral motion patterns.

The other two main biological generators of CNSLBP are chemical and neuroplastic. Circulating inflammatory markers have been found in such patients [46] and it may be hypothesised that the greater the unevenness of motion sharing, the greater the likely prevalence of rapid displacements during physical tasks causing the release of cytokines from failing holding elements.

Central sensitisation seems to be a consequence of many factors that are linked to the experience of having chronic pain; however, the role of nociception, once it has become embedded, is less clear. [47] Exercise in the treatment of chronic pain has neurological as well as mechanical effects, making its monitoring by brain mapping a variable of interest for comparison with changes in the above mechanical factors with treatment. [48] Future work could, therefore, usefully compare MSI and MSV in passive recumbent and active weight bearing motion in patients and controls to monitor their responses to physical therapies.

Limitations

The main limitation of these studies is their small numbers. However, the strength and significance of the correlations and replication of previous work suggests that the relationships found should be robust. Many clinicians would prefer weight bearing motion studies to be conducted using free bending rather than with the pelvis constrained to capture ‘natural’ motion patterns. However, this makes comparison between participant groups difficult, as it introduces sources of extraneous uncontrolled variation, including that from large hip joint motions. [26] It would also have been useful to have obtained both recumbent and weight bearing sequences for both patients and controls in Cohort 2, but this was not possible owing to missing data and will need to wait for the future studies.

Lastly, imaging studies that use image intensifiers are associated with a radiation dose; however, image acquisition times for such studies are considerably less protracted than in clinical procedures, resulting in smaller doses. Mellor, reported a mean effective radiation dose of 0.561 mSv for the four sequences which were used with Cohort 1 in the present studies. This is approximately half the dose of a conventional plain radiographic examination of the lumbar spine. [49]

Conclusion

This research confirms and extends the results of previous studies [8, 24, 29] that found abnormalities of shared intervertebral motion to be consistent with having CNSLBP and suggests possible mechanisms for this. It also suggests routes for improved understanding of the role of disc degeneration in common back pain in which degeneration may be considered a pain source when it is associated with uneven motion sharing and end-plate signal change. The results open a route to the study of motion sharing as a moderator of outcomes and of prognosis in clinical studies and its role among other known biological factors, such as muscle metabolic demands and chemical markers. However, further confirmatory work is still needed.

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