• Effect of new dynamic stabilization system on the segmental motion and intradiscal pressure. An in vitro biomechanical study.

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ABSTRACT INTRODUCTION

Non fusion techniques for the lumbar spine have become more and more popular. Dynamic posterior stabilization is an alternative to fusion in the treatment of lumbar degenerative diseases. The aims of such techniques are to preserve motion at the joint and restore normal segmental kinetics to the spine. Preclinical biomechanical evaluation of such implants is still an issue.

The purpose of this in vitro study was to analyze the influence of new lumbar posterior dynamic stabilisation device (PDSD) on lumbar intersegmental range of motion (ROM) in all the three principal motion planes, and intradiscal pressure (IDP) in flexion-extension.

We hypothesized that lumbar stabilization with such a system would cause a decrease in ROM and IDP at the instrumented level.

METHODS.

Seven fresh frozen lumbar cadaveric spines (L3-S1) were extracted with intact discoligamentous complexes. They were obtained from the University Anatomy Board after approval of the local ethics committee. The specimens were loaded in a spine tester with pure moments of ± 10Nm (steps of 1Nm) in all the three principal motion planes. Four situations were studied: Intact spine, Intervertebral disc injury with implantation of a segmental stabilization system (with and without decompression), Injured spine plus instrumentation, Injured spine. Injury performed included a section of supra and interspinous ligaments, ligamentum flavum, resection of the lower portion of the overlying lamina and upper portion of the underlying lamina. These ligament and bone resection are needed in the surgery of lumbar spinal stenosis treated by fenestration PDSD was installed using the manufacturer’s recommended operative procedure. The implant has three rigid titanium alloy elements (TA6Veli): a movable piston rod and a fixed attached to the spine by a pedicle screw, a hollow cylindrical body containing two viscoelastic silicone implants. The screws were inserted into L4 and L5 pedicles. The dynamic rod was secondary fixed to the pedicle screw in the neutral position. Lateral pedicle insertion technique was systematically used.

L4-L5 functional unit ROM was recorded using a three-dimensional opto-electronic based motion analysis system (Polaris®), EOS® low dose stereoradiography was systematically performed before testing for three dimensional reconstruction.

Three miniature pressure transducers (Entran EPL B02 100P, Measurement Specialties Les Clayes-sous-Bois, France ) were placed and secured in the intervertebral disc space of L4-L5 (anterior annulus: AA, nucleus pulposus: NP, posterior annulus: PA). Pressure transducers were configured with signal conditioner (National Instrument SCXI 1000). Amplified discal pressures were recorded synchronously on a computer via an analog to digital converter.

Statistical analyses were performed by the SPSS version 17.0 software (SPSS, Inc., Chicago, IL). Wilcoxon matched-pairs signed rank tests were run to assess significant differences between the four tested conditions. Statistical results at p<0.05 were considered significant.

RESULTS.

ROM. (Figure1/2/3). The mean ROM was 9.9±2.8 (min : 5.9°, max :14°) in flexion-extension, 8.3±1.7° (min : 5.5°, max :10.4°) in lateral bending and 6.3±1.2° (min :4.4°, max :7.8°) in axial rotation. Additional implantation of the PDSD system led to a significant reduction in ROM compared to the intact spine: 17% in flexion-extension (p=0.009*), 23.4% in lateral bending (p=0.009*) and 13.6% in axial rotation (p=0.02*). Injury led to a significant increase in ROM compared to the intact spine: 39.4% in flexion-extension (p=0.014*), 12.6% in lateral bending (9.2±1.7°, min:6.9° max :11.6° , p=0.009*) and 16.4% in axial rotation (p=0.009*). Injury and dynamic stabilization led to a decrease in ROM compared to the intact spine: 9.7% in flexion-extension (p=0.054), 17.1% in lateral bending (p=0.009*) and 5.2% in axial rotation (p=0.155).

IDP. In neutral position and for the intact configuration, the mean IDP was 0.18MPa±0.04 in the AA, 0.14MPa±0.10 in the NP and 0.13MPa±0.08 in the PA. In flexion, the mean IDP was 0.49 MPa± 0.16 in the AA, 0.26 MPa±0.10 in the NP and 0.20 MPa±0.14 in the PA. In extension, the mean IDP was 0.18MPa±0.04 in the AA, 0.16MPa±0.07 in the NP and 0.19MPa±0.1 in the PA.

In extension, IDP significantly decreased in the AA (-24.8% p=0.045*) and in the NP (-20.9% p=0.032*) for instrumented spine. IDP significantly decreased in the AA (-24.8% p=0.045*) and in the NP (-20.0% p=0.022*) for injured spine plus instrumentation.

In neutral position, IDP significantly decreased in NP (-19.39% p=0.014*) for instrumented spine . IDP significantly decreased in the AA (-12.8% p=0.045*) and in the NP (-21.43% p=0.014) for injured spine plus instrumentation. No significant change was observed in flexion.

DISCUSSION.

The first objective of our in vitro study was to evaluate the influence of dynamic stabilization system (with and without decompression) on intervertebral ranges of motion. Intervertebral motions determined in the current study are in the value ranges reported in the literature; this indicates that our testing method and setup were reliable.

The investigated PDSD allowed segment stabilization in all three principal motion planes. The influence of dynamic stabilization system seems to depend on the biomechanical characteristics of the implant itself.

The second goal of this study was to investigate the effects of a dynamic stabilization in the load transfer of the treated segments, through the measurement of intradiscal pressure. Pressure measurements were here performed in three different disc locations, which provided complementary information compared to most in vitro studies where pressure changes are evaluated in the nucleus only. IDP is indeed a non hydrostatic pressure and may vary depending on the sensor position. Load sharing with the intervertebral disc after implantation was found primarily in the neutral position and in extension.

Figure n°1: Evolution of ROM in flexion-extension

Figure n°2: Evolution of ROM in lateral bending

Figure n°3: Evolution of ROM in axial rotation