BIOMECHANICAL EFFECTS OF LUMBAR MULTIFIDUS AND PSOAS MAJOR MUSCLE DYSFUNCTION ON THE LUMBOSACRAL SPINE

Robin Remus (1), Andreas Lipphaus (2), Sascha Selkmann (1), Beate Bender (1)

1. Chair of Product Development, Dept. of Mechanical Engineering, Ruhr-University Bochum, Germany
2. Biomechanics Research Group, Dept. of Mechanical Engineering, Ruhr-University Bochum, Germany

Introduction

Low back pain (LBP) is a major health issue whose causes are manifold. Numerous problems are associated with a lack of core stability, altered segmental intervertebral motions, and a reduced range of motion [1,2]. Correlations with morphological and structural changes as well as weakness or contractures of the lumbar multifidus (MF) [2] or psoas major (PM) [1,3] muscles have been observed in vivo. However, methods for detecting muscle recruitment patterns and biomechanically relevant changes in the musculature in patients with LBP are limited. To improve understanding and treatment, this study aims to investigate whether changes in MF or PM in an active hybrid simulation model lead to pathological responses that are consistent with clinical observations of their role in the ethology of LBP.

Methods

We utilize a validated hybrid model of the healthy ligamentous lumbosacral spine [4] built in ArtiSynth [5]. Vertebrae L1-S1 are interconnected with hyper-elastic fibre-reinforced finite element discs, ligaments, and facet joints. The intra-abdominal pressure is considered as a force on the thorax via muscle forces acting on the abdomen. For the active model component, twelve sagittal symmetric muscle groups are implemented using 258 muscle fascicles with a Hill-type muscle model and a resting muscle tone of 0.1%. The muscle redundancy problem is solved using an inverse-dynamic tracking controller (TC) which provides a solution of the forward dynamic simulation. Values of the cost function are muscle activities squared as well as target poses of the thorax and the lumbar vertebrae. Thus, all bones cranial to the stationary sacrum are free to move. From an upright posture, the movements into different postures (-10° extension to +30° flexion) with loads of up to 20 kg held in both hands are simulated. Based on results of the healthy muscles in the respective postures, four sagittal symmetrical muscle variations are examined: Limitation of the force of PM (1) or MF (2) and increase of the resultant force of PM (3) or MF (4). For this, the upper or lower excitation limits of the TC are adjusted. All other muscles and settings are unchanged.

Results

Dysfunctions of MF and PM affect the biomechanical model results. With exception of (2), PM is not relevantly activated to a greater extent for the load cases considered. For (2) in 30° flexion, all other muscle sections of the erector spinae (ES) (Figure 1B), the lateral internal abdominal muscles, and PM are increasingly activated. This barely changes the intradiscal pressure (IDP). However, the stability of the model is reduced by extension of the settling phase when reaching the maximum deflection. The IDP increase is most pronounced in (3) (Figure 1C). The proportional increases in IDP and facet joint contact force is higher caudally. In upright standing lumbar lordosis remains almost constant, rotational compensation occurs inter-vertebrally. The intervertebral angles (IVA) increase for L2/3 to L4/5 and vice versa for L1/2 and L5/S1. Higher PM forces result in ES being more activated as well.

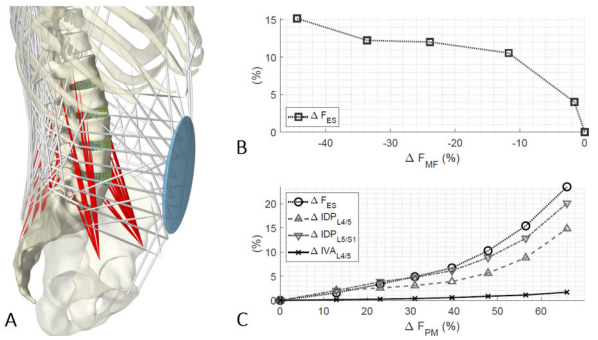

Figure 1: A) Active model with MF and PM highlighted in red. B) ES force change ΔF for (2) in 30° flexion C) Results for (3) in upright position without load in hands.

Discussion

The results of this study show that the simulation model used and solved with an inverse-dynamic TC provides consistent pathological model responses with PM and MF dysfunctions. These include a decrease in core stability, increased loading of lumbosacral structures, alteration of muscle activation patterns, and changes of IVA [6]. Overall, MF and PM are important stabilizers of the lumbar spine and have a low negative correlation. To further improve the validity and provide clinical relevance additional dysfunctions, personalized muscle parameters, and anatomies should be investigated in the future. One problem that remains to be solved is the consideration of the relation between pathophysiologic habits and the perception of LBP.

References

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