SPONDYLOLYTIC SPONDYLOLISTHESIS IN IMMATURE LUMBAR SPINES: IN VITRO AND FINITE ELEMENT ASSESSMENTS

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Introduction: The progression of spondylolyis (spondylolisthesis) is more prevalent among children and occurs most often in the adolescent growth spurt. End-plate lesions, lumbar lordosis, and disc degeneration have been implicated as being possible risk factors. How these factors influence the pathomechanism of the disorder is not clearly understood and the most critical risk factor has not been identified. Literature relating to the biomechanics of lyis and olisthesis is sparse. Thus, the role mechanical factors play is not clear. Additionally, studies that have been done have used the adult spine as the model. This study uses the immature Chama Baboon (Papio ursinus) lumbar spine as the biomechanical model to study the disorder and be nearer to being able to clearly identify the pathomechanism of lumbar olisthesis in the pediatric population. Furthermore, finite element modeling (FEM) is used to evaluate the role of a weakened pars and a weakened growth-plate as etiological factors in the occurrence of pars defects.

Materials & Methods: Twelve immature baboon functional spinal units (FSUs) were used: six 6 and six L6-S1. Bilateral pars defects to the rostral vertebral endplate of the FSU were created. Each specimen was assigned to one of two groups: Disc Intact (DINT) or Disc Dissected (DDIS). In the DINT group, the disc was left intact. In the DDIS group, 75% of the anterior and anterolateral ligamentum were anteriorly incised. Rostral and caudal vertebral bodies of a FSU were then fixed using a specially designed ring-screw device. The specimens were placed within the uniaxial MTS machine, (Fig. 1). The specimens were tested in A-P shear mode until failure at a rate of 25 mm/min. Load-displacement curves were obtained for each specimen and failure load and displacement at failure were calculated. A non-linear 3-D FEM of an immature baboon L4-L5 FSU was developed (Fig. 2) by scaling an adult baboon L4-L5 FEM, which was created from CT scan.[3,5]. Growth-plate architecture for the immature baboon FEM was derived from the literature on the human and from histology of the immature baboon lumbar spine. As material property data literature for the baboon were limited, material properties from human studies were taken from the literature.[2,3,5] Additional models were then created from this intact model, including a thin pars model, a fractured pars model, as well as the incorporation of a weakened growth-plate into all models. An immature human FEM was developed in a manner similar to the immature baboon FEM. Loading conditions for all models consisted of application of 100 N A-P shear force.

Results: All specimens failed through the growth-plate at either the upper surface of the caudal vertebra or the lower surface of rostral vertebra. The twelve FSUs in the DINT group failed at 220.3 ± 9.9 N following 10.3 ± 5.4 mm displacement. In the DDIS group, the failure of the specimens was also through the growth-plate, and not through the intervertebral disc. Specimens in the DDIS group failed at 120.6 ± 33.2 N following 12.0 ± 4.6 mm displacement. Failure locations were assessed radiographically as well as histologically. (Fig. 3). All five upper plate failures involved FSUs in the DDIS group, compared to only 14% lower plate failures involving FSUs in the DDIS group. Failures through the growth-plates suggest that the growth-plate is the weakest link for A-P shearing force leading to forward slip. In the intact immature baboon FEM 29% of the load was carried by the lower growth-plate of L4, while 34% of the load was carried by the upper growth-plate of L5. When the lower growth-plate of L4 was weakened, loads through the growth-plate remained approximately the same as for the intact case. However, there was a slight increase in von Mises stresses through the pars region, as well as a slight increase in A-P translation of L4 with respect to L5 for the weakened growth-plate model compared to the intact model. The effect of a thin pars was a substantial increase in shear and von Mises stresses through the pars, while loads through the growth-plates and A-P translations remained approximately constant. For the fractured pars, with and without a weakened growth-plate, 90% of the load was carried by the growth-plates. For the fractured pars with a weakened growth-plate, the A-P translation of L4 with respect to L5 increased from 6.03 mm for the fractured pars to 6.10 mm for the fractured pars with a weakened growth-plate. The immature human FEM predicted that 74% of the load would be carried by the lower growth-plate of L4 and 89% by the upper growth-plate of L5.

Discussion & Conclusions: It has been hypothesized that olisthesis in children with lyis is due to slippage through the epiphyseal plate.[1] Clinically, the slip may occur between the osseous and cartilaginous end-plates.[4] The weakest link in A-P shear mode was determined to be the growth-plate, not the disc. These results confirm the findings of our calf investigation [6], and further support the hypothesis of Farfan. However, growth-plate failure in this study was observed in both the lower growth-plate of the superior vertebra and in the upper growth-plate of the inferior vertebra. In the calf study, failure site was consistently in the upper growth-plate of the inferior vertebra. Additionally, a greater percentage of the load was experienced by the human growth-plates than by the baboon's. An explanation could be the location of the posterior elements in relation to the growth-plate. In the immature baboon FEM, the posterior elements lie next to the upper growth-plate of L5, while in the immature human FEM, the posterior elements are located just below this growth-plate, allowing more load to be carried by the human growth-plates. The location of the posterior elements may vary between species, possibly accounting for upper and lower growth-plate failures. The results of this study show that failure occurs through the growth-plate in A-P shear mode. This suggests that in the pediatric lumbar spine with pars defects, slippage may occur between the growth-plate and osseous end-plate.[4] Surgeons may assess growth-plate lesions using MRI technology, possibly predicting and preventing the development and progression of olisthesis. Further investigations will assess the role of repetitive loading in progressive slippage. FEM results predict that a thin pars and/or a weakened growth-plate increase stresses through the pars region, eventually leading to a defect. The fractured pars model results suggests that lyis is a prerequisite for slippage to occur.

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Figure 1: MTS set-up. Figure 3: Histology: intact L5-L6-S1 (left), tested L5-L6-S1 (right).

Figure 2: FEM of the immature baboon L4-L5 FSU. a) axial, b) sagittal. A similar model was developed for the immature human FEM.