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主　論　文　の　要　旨

論文題目　Investigation into the developing mechanism of idiopathic scoliosis by means of computational mechanics (計算力学的アプローチによる特発性側弯症進行メカニズムの解明)

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論　文　内　容　の　要　旨

Idiopathic scoliosis comprises spinal irregularity with lateral curvatures together with rotation without any marked abnormality of the vertebrae or associated musculoskeletal condition. Since almost all cases of the disorder appear during adolescence, particularly during growth spurts, growth has been recognized as associated with the etiology of idiopathic scoliosis in some way. The objective of the present thesis is to investigate the etiology and the developing mechanism of idiopathic scoliosis by means of computational mechanics.

The thesis consists of seven chapters. Chapter 1 reviews the relevant background literature including the information of the characteristic, the classification, the harm, and the treatments of idiopathic scoliosis. A large number of hypotheses and physical models have been proposed for the pathogenesis of idiopathic scoliosis. From the point of view of the mechanics, we classify these concepts into the following issues. (1) The growth itself is asymmetrical. (2) The buckling by symmetrical growth of the vertebral bodies induces the deformation of scoliosis (the buckling hypothesis). For the buckling hypothesis, we review the literature and points out that Dickson presented an important observation of a flattening of the thoracic spine in the specimens of the idiopathic scoliosis. They identified the trigger of the rotational instability as a median plane asymmetry, that is, the flattening or decreasing of normal thoracic kyphosis at the apex of the curvature, and they declared this instability to be a buckling phenomenon.

In Chapter 2, in order to demonstrate the buckling hypothesis, we introduced the theory for buckling phenomenon caused by the growth of vertebral bodies, and analyzed the buckling phenomena using three types of plate models simplified

the spine. We analyzed linear buckling modes caused by the growth deformation using the finite element method, and we confirmed the existence of buckling phenomena and clarified the range of the geometrical parameters in which this buckling occurs. By the comparison of the ranges between the three models, we obtained the following results. (1) The growth of the frontal part of spine in depth around 10 mm from the frontal plain causes the buckling phenomena most easily. (2) The model with physiological curvature enlarges the buckling area than the strait model. (3) The structure having holes in the rear part also enlarges the buckling area. These results support the buckling hypothesis as a cause of initiation of the idiopathic scoliosis.

However, the results in Chapter 2 are only valid for infinitesimal deformation and not applicable to estimate the stability for post-buckling behavior. Thus, in Chapter 3, we conducted the theory for post-buckling deformation caused by the growth of vertebral bodies considering the geometrical nonlinearity, and demonstrated the nonlinear post-buckling simulation using the simple plate models. In these analyses, the buckling modes obtained from the linear buckling analyses of the simple plate models in Chapter 2 were chosen as the initial perfections of the plate models. To solve unstable nonlinear post-buckling deformation, the incremental Arc-length method was employed. From the results, the stable post-buckling deformations were obtained in almost simple plate models, while an unstable post-buckling deformation was confirmed after introducing the initial imperfection to the 3rd buckling mode.

In Chapter 4, based on the results from Chapter 2, the similar linear buckling analysis was performed using the spine finite element model without rib cage instead of the simple plate models. The spine finite element model consisted of 69,658 nodes and 59,356 elements. For the boundary conditions, we assumed that the sacrum was fixed. From the results, the 4th buckling mode which is similar to the clinical scoliosis deformity was obtained. By a comparison of different growth regions, we investigated the influence of the region of the buckling phenomena on the physiological curvature of the spine. We found when the growth of the frontal parts of spine in depth around 10 mm, the spine model was in the easiest state to get buckled. This result accords with the result in Chapter 2.

Chapter 5 explores post-buckling deformations caused by the growth of vertebral bodies using the spine models by the Arc-length Method. The 4th mode obtained from the linear buckling analysis of the spine model in Chapter 4 was chosen as the initial perfection of the spine model for post-buckling analysis.

However, we did not obtain any deformation similar to the clinical modes. Thus, we shrank the width of the spine model, and fixed the node at the center of front boundary of C7 in horizontal plane considering the controllability of posture. From the results, the existence of the non-linear buckling phenomena was confirmed. However, the magnitude of the deformations are too small to explain the pathogenesis of the severe deformity observed in patients of idiopathic scoliosis. Thus, although the buckling hypothesis can explain the pathogenesis of the onset in the idiopathic scoliosis, it cannot explain the developing mechanism.

In Chapter 6, in order to investigate the mechanism of progression of the idiopathic scoliosis, the influence of bone remodeling after the buckling was analyzed. The bone remodeling is a phenomenon of absorption and formation of the bone which occurs according to the change in the mechanical and physiological circumstance. In this study, the bone formation was simulated by increasing of the volume of the bone in proportion to the strain distribution which was obtained from the nonlinear post-buckling deformation analysis in Chapter 5. The bone resorption was simulated by decreasing of the volume of the bone in proportion to the strain distribution. From the results, it is confirmed that the bone formation corrects the original curve, while the bone resorption worsens the original curve. These results suggested that, the bone resorption with respect to strain at post-buckling can be a candidate of the developing mechanism of the idiopathic scoliosis.

Chapter 7 presents the conclusions conduced from this study and suggestions for the future work on the topic. From the investigation in Chapters 2 and 4, since the linear buckling modes are similar to the scoliotic modes, the buckling hypothesis is effective as the pathogenesis of the onset in the idiopathic scoliosis. However, from the results in Chapters 3 and 4, since any severe scoliotic curve is not obtained by the post-buckling deformation analysis, it is difficult to explain the pathogenesis of the progression by the buckling hypothesis. On the other hand, the investigation of bone remodeling in Chapter 6 declares that the bone resorption in proportion to the strain at the post-buckling deformation progress the scoliotic curves. Thus, based on the results obtained in the present study, it is concluded that the buckling phenomena with respect to the growth deformation of the vertebral bodies can be a pathogenesis of the onset of the idiopathic scoliosis, and the bone resorption in proportion to the strain at the post-buckling deformation can be a mechanism developing the idiopathic scoliosis.