Technical note

A biomechanical model of Peyronie’s disease

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Abstract

Peyronie’s disease is a pathological condition of the penis which is characterized by localized ossification of the tunica albuginea. A common symptom of the chronic stage is penile deformity during erection, which is frequently associated with pain and erectile dysfunction. A two-dimensional biomechanical model of the penis was applied to study the development of Peyronie’s disease by simulating the mechanical stress distribution which would result from the interaction of the ossified tunical tissue with other penile soft tissues. The model was solved by using commercial finite element software for a characteristic erectile pressure. The results demonstrate that Peyronie’s plaques may induce intensified stresses around the penile nerves and blood vessels, up to double those in the normal penis. These elevated stresses may cause a painful sensation of neural origin or ischemia in regions of compressed vascular tissue. Severe penile deformities have been shown to develop if Peyronie’s plaques develop only around one of the corpora cavernosa due to the non-homogeneous resistance of the tunica to expansion during erection. The present model can be clinically applied as an aid in the planning process of reconstructive surgery or insertion of a prosthesis.

1. Introduction

Peyronie’s disease is a pathological condition of the penis characterized by the alteration in the appearance and cellularity of collagen within the tunica albuginea. These changes may occur in single or multiple sites of plaque formation. As the disease progresses, the dorsal and middle parts of the tunica albuginea gradually become fibrotic or ossified (Vande Berg et al., 1982; Hamm et al., 1986; Davis, 1997). In its early acute stage, the disease involves only the tunica albuginea, while secondary nerve damage and ischemia may induce fibrosis of the cavernosal erectile tissue in the chronic stage (Devine, 1997). Painful erections during the acute stage and penile deformities during the chronic stage are common symptoms of the disease, which is also accompanied by erectile dysfunction in about 20% of the cases (El Sakka and Lue, 1998). The prevalence of Peyronie’s disease is 0.4–1% (Akkus et al., 1997; Chevallier et al., 1997).

The nerves and blood vessels located at the dorsal aspect of the penis play a major role in achieving and maintaining a normal erection (Anderson and Wagner, 1995; Yang and Bradley, 1999). It follows that interference of neural activity or obstruction of blood vessels due to structural or functional damage in the dorsal/middle parts of the tunica albuginea, such as that which occurs in Peyronie’s disease, decreases the capability to achieve and/or maintain a normal erection (Gefen et al., 1999). In support of this contention, blood flow abnormalities have been associated with impotence in Peyronie’s disease (Montorsi et al., 1994; Culha et al., 1998).

The mechanisms by which Peyronie’s disease progresses are not well known, and this naturally affects patient management (Marzi et al., 1997). In order to understand the development of Peyronie’s disease better, we employed a computational model of the penis for the analysis of the stresses within its tissues during erection (Gefen et al., 1999). The findings are presented together with information on the etiology and evolution of the disease, all essential data for clinical decision making, e.g., when considering surgical plaque removal or insertion of a prosthesis.

Keywords: Erectile dysfunction; Numerical model; Finite element method; Tissue ossification; Plaque
2. Methods

The methodology used to build the two-dimensional (2D) computational model for the analysis of the structural stresses in the penile tissues is described in detail in Gefen et al. (1999); its essential components relevant to the present report are given below.

2.1. The penis model

The 2D geometry of the model was extracted from an anatomical schematic section through the middle of the penis (Dalton, 1983). The penile components incorporated within the model include the tunica albuginea, skin, dorsal blood vessels and the urethral channel (Fig. 1a).
An equivalent erectile pressure $P_s = 50 \text{ mmHg}$ was applied to the boundaries of the cavernosal spaces, reflecting the difference between the inflation pressure induced by arterial blood flow into the penile cavities and the resistant stress of the stretched spongy cavernosal tissue to inflation (Gefen et al., 1999). The penile tissues were assumed to be made of homogeneous, isotropic, linear elastic materials, whose properties are detailed in Table 1. The stress distribution within the penile tissues was determined by finite element analysis (ANSYS) in which the general equilibrium equations were solved for plane stress. Automatic meshing was used to generate optimally converging meshes of 1500–2000 quadrilateral and triangular elements that described the cross-sectional geometry. The model was constrained to allow the expansion of the penis during erection without rotation (Fig. 1a).

### 2.2. Simulation of Peyronie’s disease

The stable Peyronie’s plaques are rigid structures due to the lack of elastin and the nature of reorganization of the collagen fibers (Akkus et al., 1997; Ehrlich, 1997). In the absence of the experimental data for Young’s modulus and Poisson’s ratio of the ossified tunica albuginea, we employed experimental results of ossified bovine cartilage (Mente and Lewis, 1994), as shown in Table 1.

The formation of symmetric Peyronie’s plaques at the dorsal aspect of the tunica albuginea and along the tunical septum was simulated through the quasi-steady analysis in which the stress distribution was solved at the progressing stages of the disease, i.e., when the plaque occupied approximately 5, 10, 20 and 40% of the total tunical cross-sectional area (Fig. 1b–e). Similarly, development of non-symmetric plaques at the dorsal aspect of the right cavernosum were analyzed for simulated cases in which the ossified tunica albuginea occupied about 10, 15 and 20% of the total tunical area (Fig. 2). In order to quantitatively characterize the effects of progressive fibrosis and ossification of the tunica albuginea, we examined the averaged values of stresses across a linear course $S$, which originates at the center of the dorsal face of the penile cross-section, crosses the dorsal and tunical nerve roots, and terminates at the cavernosal apex (Fig. 1a).

### 3. Results

The computed von Mises equivalent stresses generated during simulations of full erection are shown in Fig. 1 for a healthy penis (Fig. 1a) and for progressing stages of development of symmetric Peyronie’s plaques (Fig. 1b–e). Stresses were seen to be transferred mainly through the stiffer tunica albuginea, while the penile skin appeared to bear negligible loads. In the healthy penis, stresses were shown to concentrate on the dorsal and lateral aspects of the tunica albuginea (Fig. 1a). In Peyronie’s disease, stresses were significantly larger at the dorsal aspect of the tunica albuginea and tended to expand to its middle part (or septum) as the ossified area of the tunica albuginea increased. Contrarily, in the lateral aspects, stresses were shown to decrease with the increase in plaque size.

Inflation of the elliptic cross-sectional shape of the cavernosum during erection in a healthy penis yielded a more circular corporal profile (Fig. 1a). Formation of symmetric dorsal plaques constrained the expansion of the corpora during erection, yielding corporal profiles which were closer to elliptic as the plaque size increased. The overall cross-sectional shape of the penis, however, remained symmetric insofar as the plaque equally affected the deformation of both corpora (Fig. 1b–e). When asymmetric plaques were generated, expansion of only one cavernosum was constrained, resulting in asymmetric deformations of the overall penile cross-section, with little relation to the plaque size (Fig. 2).

The average dorsal stresses (along $S$ in Fig. 1a) that had been generated as a result of progressive ossification of the tunica albuginea (Fig. 3) are seen to increase exponentially with the plaque size. When the plaque occupied more than 30% of the tunical cross-sectional area and was completely ossified (i.e., its stiffness being maximal), the values of these intensified stresses could rise to more than double of those in the healthy penis.
4. Discussion

Peyronie’s disease has been characterized by ultrastructural changes in the tunica albuginea leading to loss of its elasticity (Akkus et al., 1997) and resulting in significantly magnified and unevenly distributed penile stresses. The present numerical simulations show that the pathologic tunica albuginea may bear stresses of more than twice the normal value, depending on the plaque size and location. Although they are too small to cause mechanical failure of the tunica albuginea itself (Bitsch et al., 1990), these elevated stresses are transferred to the tunical nerves as well as to the adjacent dorsal nerves and blood vessels (Devine et al., 1997). The intensified stresses may irritate nerves and/or impose an abnormally large pressure on the vascular bed, leading to pain from the resultant ischemia. These events are likely to cause discomfort or painful erections in the early stages of the disease, and may lead to penile deformation, erectile insufficiency and/or inability
It is most likely that this mechanism is responsible for penile deformities during erection in Peyronie's disease. The local distortion caused by an irregularly shaped plaque may also cause torsion of the erect penis and further increase in stresses around the adjacent nerves and blood vessels.

In order to deal with the structural complexity of the penis and contend with the lack of experimental data on material properties of its tissues, assumptions needed to be made for the purposes of simplification, and this should be kept in mind while interpreting the results. For instance, penile tissues were assumed to be isotropic and linearly elastic. This assumption is generally adequate for the analysis of the ossified Peyronie's plaque, but a quasi-linear viscoelastic assumption should be considered for all other tissues (Fung, 1994). Replacing the tunical elastic modulus with a non-linear constitutive law of a ligamentous tissue (Fung, 1994), which is of similar biological structure, yielded averaged dorsal stresses which were greater by 30–50%, depending on the plaque size.

In conclusion, our model demonstrated that ossification of the dorsal and middle parts of the tunica albuginea induces intensified stresses that may irritate nerves and/or cause vasopathology, leading to the loss of capability to achieve and/or maintain functional erection. By integrating newly developed powerful algorithms for 3D geometry reconstruction and finite-element analysis, the present approach can be extended to introduce realistic 3D models that will be useful for surgical planning.

**References**


