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FLUID-STRUCTURE INTERACTION: ADVANCES ON ERECTILE FUNCTIONALITY

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ABSTRACT

In this work the interaction between the blood and the penile structures was treated in order to have an insight into erectile functionality. Coital consummation and, maybe, its concomitant tendency to procreate is only attainable when there exists a fluid-structure synergy. Remarkably, hemodynamic influence is the basis of the behaviour of the penile 'utensils' towards such consummation. The blood flow that induces penile engorgement that precedes vaginal intromission results in both tunical and vascular deformation, as was shown theoretically in this paper. The equations that govern blood flow and those that govern the penile structures' response are derived. One of the results is the equation that furnishes the axial length of a penile shaft under axial displacement by using some tunical characteristics. Of much interest in erectile functionality is the concept of penile buckling. This was given attention here. Contrary to some insinuations occasioned by the treatment of the penile 'shaft' as a column subjected to axially compressive force that buckles under further application of a force, which was reported here, this work sees penile tissue detumescence resulting from the withdrawal of a force as the basis of penile axial buckling.

Keywords: hemodynamics, deformation, elastin, buckling, equations, pressure

INTRODUCTION

The penis plays two vital roles: the primary role is the removal of urine, a liquid by-product of metabolism in humans, and in many other animals, the act of coition which is approved for the divine purpose of procreation, may be seen as a higher-order role. About three physiological fluidic substances flow through the penile structures−urine, semen, and blood. While urine and semen are emissive fluids, blood is not. The flow under consideration here is blood. The flow of blood in arteries and veins is a biological fluid-structure interaction (FSI) problem. In the event of blood flow, the recipient body tissues are innervated. In fine, the integrity of cells and organs is essential for normal development, and therefore blood flow to them is indispensable. Tissue healing is predicated on adequate blood flow and the rate and quality of tissue repair are directly proportional to the blood and oxygen supply (see McMeeken (n.d)). The same, as above, can be said of general tissue functionality. The structures considered in this work are the penile tissues. The mathematical theory of erectile functionality presupposes the knowledge of penile anatomy and physiology. A plethora of literature, which include Robert and Tom (2005), Levin (2016), Ahmed (2004), and Rob (2013) treat hemodynamics of penile erection from the standpoints of anatomy and physiology and therefore furnish some salient facts for

mathematical analysis. Physiological treatment of concern is the hemodynamic influence on penile functionality, while the anatomical treatment concerns the structures of the penile members. Studies on penile structures and functionality deal largely with the behaviour of the arteria-venous vessels, together with the non-vascular structures, albeit under hemodynamic influences. Penile tissues consist of soft tissues. Most biological soft tissues, including blood vessels considered to be thin-walled elastic cylinders, are thought to undergo large deformation (Jian et al.(n.d), Brossollet (1995). The typical substances inside the extracellular matrix of the soft tissue, found in the penile structures, are *collagen* and *elastin*. They play critical roles in the mechanical deformation of the penile shaft during tumescence and erection, as detailed by Hemat (2007). The issue of increased hemodynamic flow, during excitation, that induces tumescence and the response by the penile structures leading to erection may not have received substantial mathematical attention. Penile reaction to applied load was treated in terms of column buckling by Udelson et al. (1998, 1999). Udelson et al. (1998) described penile buckling forces as the magnitude of the axially compressive *force applied to the glans of an erect penis*, resulting in evident curving such that any added small force would prompt collapse (buckling) of the erect shaft. The bulwark of the above works was radial penile deformation. A question arose: Which one of *axial compression* and *radial compression* is instrumental in counteracting penile shaft buckling? This informed a polemic issue that is worth reconciling; while Udelson *et al.* (1998, 1999). saw the penile shaft as an isotropic shaft with axial stresses as the principal stresses governing its column buckling, findings by Timm *et al*.(2001) oppugned the isotropic shaft view with the position that the penis acts as a thin-walled pressure vessel that becomes rigid when its walls reach their elastic limit. They contended that the physical properties of the tissues contained by the penile corpora cause it to behave like a thin -walled pressurized vessel, wherein the stresses, hoop and axial, have a constant relationship. This relationship is not contingent on the length to diameter ratio whilst as an isotropic beam ,this relationship varies. According to them, penile rigidity and ability to withstand buckling may be determined by radial compression measurements rather than by radial penile deformation. Tubes are treated as a pressure vessel when the only load acting is the hydrodynamic pressure from the fluid, and when the load due to viscous and shear stresses is neglected. However, forces that act to engorge the penile tissues may not only be hydrodynamic; the deliberate act of penile massaging in the direction of flow induces titillation. This external contact force adds to the internal fluid pressure to make the structure behave in a manner that is rather more than just a pressure vessel. The concept of axial compression as applied to the arterial vessel led to the modelling of blood vessels as closed-ended vessel segments. In a bid to estimate the critical pressure by use of the Euler buckling equation, Kylstra et al. (1986), Brossollet and Vito (1995) modelled blood vessels as such. This was adjudged unrealistic by Hai-Chao *et al.* (2013) on the contention that *close-ended* vessel structure is incompatible with physiological blood vessels.

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MATHEMATICAL FORMULATIONS

Before proceeding to the mathematical formulations of the problem, it is pertinent to have an insight into the penile anatomy.

The penile structure

The penis is a complex organ comprising ligaments, smooth muscle, skeletal muscle, arteries, veins, and other numerous tissues. The penile structure, under normal non-excitation conditions, is flaccid and pendulous. In these states, it is approximately 8.5–10.5 cm long and 3–5 cm in diameter (Levin (2016)), The constituent parts work together in perfect synchronization when the penis transits from flaccid to erect, and vice versa. Expansion occurs when blood is pumped, and as soon as the penis is full the veins are pressed against the tunica, therefore blood is prevented from leaving the penis (veno-occlusion), thereby sustaining erection. When the limit of the tunica is reached, additional pressure results in stiffness. The outer longitudinal layer encloses the corpus chambers and controls the length of the expanding penis. The main body of the penis consists of three circular chambers. The top two chambers make up the corpora cavernosa. The lowermost chamber makes up the corpus spongiosum. The *tunica albuginea* envelops the corpora cavernosa. It has biomechanical properties, as it is made up of fibrillar collagen entwined with elastin fibres. This composition gives the penis great tissue strength flexibility and rigidity when stretched (Hsu (2013)). The elastic fibres (elastin) contribute more to the ability to tolerate radially compressive loads. The flaccid penis is markedly in a moderate state of contraction, and may further shrink in extremely cold weather conditions. In the flaccid state only a small amount of arterial flow delivers nourishment to the tissue through tunical contraction of the smooth muscles (Hemet (2007)). The cavernous smooth musculature and the smooth muscles of the arteriolar and arterial walls play a crucial role in the erectile process, thanks to the internal pudendal artery− the major vessel that supplies oxygenated blood to the penis. During erection, blood is stored in the lacunar spaces of the cavernosal erectile tissue. The filling of the lacunar spaces with blood causes penile expansion. While collagen and elastic fibres are compliant tissues that permit changes in girth and length during tumescence, they also provide enough resilience for a return to the flaccid state with detumescence (Hemet (2007)). The tunica itself guards against overstretching or compression of the nervous and vascular appurtenances, which are subject to increasing intra-cavernosal pressure during erection.

The inner penis is encircled by and made up of the pelvic floor muscles, mainly the bulbocavernosus muscle and the ischiocavernosus muscle. Together, these muscles pump blood into the penis, inducing a chain of reactions that leads to an erection. The penis has numerous penile arteries and veins, including the outstanding ones — the dorsal vein and dorsal artery, located on the top part of the penis. The arteries and veins provide the plumbing for the blood being pumped by the pelvic floor muscles to get in and out.

Tissue deformation

Blood vessels are one of the biological soft tissues that undergo large deformation (Jian et al. (n.d), Brossollet (1995)). In general,

arteries and veins are subject to complex mechanical loads. They have to contend with lumen and outer pressures, axial tension due to tissue tethering, twisting and, bending caused by body movement. Soft tissues have some properties which include hyperelasticity, viscoelasticity, incompressibility, anisotropy, and their stress-strain curve is nonlinear. Essentially they are considered a thin-walled elastic cylinder. In what follows, the basic theory of deformation is discussed. In the configurations below, Fig. 1(a) shows a hollow cylinder of length L, with the inner, outer radii R_1 and R_2, respectively. The coordinates of initial and deformed shapes are (R, Θ, Z) and (r, θ, z) respectively; the base vectors of the Cartesian coordinates of initial and deformed configuration are ($\boldsymbol{e}_R$, $\boldsymbol{e}_\Theta$, $\boldsymbol{e}_Z$) and ($\boldsymbol{e}_r$, $\boldsymbol{e}_\theta$, $\boldsymbol{e}_z$) respectively.

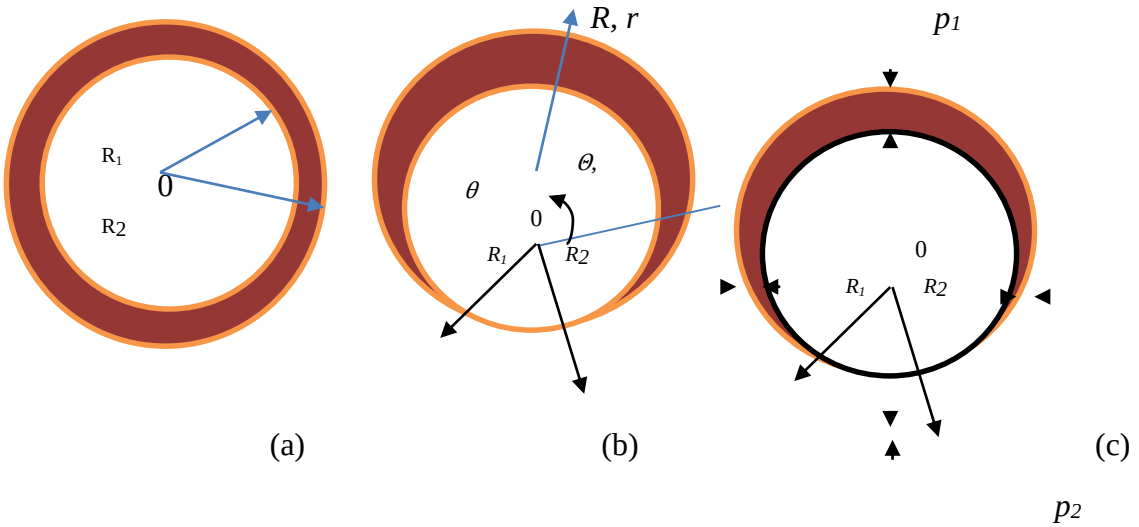

Fig.1 Schematic of (a) the reference configuration, (b) the coordinate system and (c) uniform pressure, of a soft hollow cylinder

The axisymmetric deformation under pressure (as in seen Fig. 1(c)) may be expressed as

$$r = r(R), \theta = \Theta, z = \lambda_1 Z \qquad (1)$$

where λ is the stretch/elongation the along axial direction of the cylinder. The inner and outer radii, of cylinder are such that

$r_1 = r_1(R_1)$, $r_2 = r_2(R_2)$, with $r_1 < r_2$.

The associated left Cauchy-Green deformation tensor (see Jerrold E. M. and Thomas (1983)) is

$$\boldsymbol{B} = \left(\frac{dr}{dR}\right)^2 \boldsymbol{e}_r\boldsymbol{e}_r + \left(\frac{r}{R}\right)^2 \boldsymbol{e}_\theta\boldsymbol{e}_\theta + \lambda_z^2 \boldsymbol{e}_z\boldsymbol{e}_z, \quad (2)$$

with the three invariants given by

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$$\mathrm{I} = \left(\frac{dr}{dR}\right)^2 + \left(\frac{r}{R}\right)^2 + \lambda_z^2,$$
$$\mathrm{II} = \left(\frac{r}{R}\right)^2\left(\frac{dr}{dR}\right)^2 + \lambda_z^2\left(\frac{r}{R}\right)^2 \lambda_z^2\left(\frac{dr}{dR}\right)^2, \quad (3)$$
$$\mathrm{III} = \lambda_z^2\left(\frac{r}{R}\right)^2\left(\frac{dr}{dR}\right)^2.$$

If material incompressibility is assumed, then

$$\mathrm{III} = \lambda_z^2\left(\frac{r}{R}\right)^2\left(\frac{dr}{dR}\right)^2 = 1 \quad (4)$$

The Cauchy stress for incompressible materials is

$$\boldsymbol{\sigma} = -p\boldsymbol{I} + 2\left(\frac{\partial W}{\partial \boldsymbol{I}}\boldsymbol{B} - \frac{\partial W}{\partial \boldsymbol{II}}\boldsymbol{B}^{-1}\right), \quad (5)$$

where p is the unknown pressure, W is the strain energy density of the elasticity, $\mathbf{B} = \mathbf{F}.\mathbf{F}^{\mathrm{T}}$ is the left Cauchy-Green deformation tensor, F is the deformation gradient. The radii of the deformed and initial cylinders are related by

$$\lambda_z r dr = R dR$$
$$\lambda_z r^2 = R^2 + A \quad (6)$$

where A is a constant. Using the relation (5) the nonzero components of the Cauchy (normal) stress in Cartesian coordinates of cylinder read (see Jian *et al*. (n.d)),

$$\boldsymbol{\sigma}_{rr} = -p + \left[\nu_1\left(\frac{R}{\lambda_2 r}\right)^2 - \nu_2\left(\frac{\lambda_2 r}{R}\right)^2\right],$$
$$\boldsymbol{\sigma}_{\theta\theta} = -p + \left[\nu_1\left(\frac{r}{R}\right)^2 - \nu_2\left(\frac{R}{r}\right)^2\right], \quad (7)$$
$$\boldsymbol{\sigma}_{zz} = -p + \left(\nu_1\lambda_z^2 - \nu_2\frac{1}{\lambda_z^2}\right).$$

where υ_i are the material parameters. The equilibrium equation satisfied by Cauchy stress is

$$\nabla.\boldsymbol{\sigma} = 0 \quad (8)$$

Substitute (7) into (8) to get the component of Cauchy stress in the form

$$\frac{d\sigma_{rr}}{dr} = \frac{A}{\lambda_z^2 r^3}\frac{2\lambda_z r^2 - A}{\lambda_z r^2 - A}\left(\nu_1 + \nu_2\lambda_z^2\right). \quad (9)$$

Any axisymmetric deformation of a cylinder, which as well satisfies (6), has the surface deformation gradient given by

$$\boldsymbol{F}_s = \frac{r}{R}\boldsymbol{e}_\theta\boldsymbol{e}_\Theta + \lambda_z\boldsymbol{e}_z\boldsymbol{e}_Z \quad (10)$$

and the surface left Cauchy-Green deformation tensor reads

$$\boldsymbol{B}_s = \boldsymbol{F}_s.\boldsymbol{F}_s^T = \left(\frac{r}{R}\right)^2 \boldsymbol{e}_\theta \boldsymbol{e}_\Theta + \lambda_z \boldsymbol{e}_z \boldsymbol{e}_Z \ . \quad (11)$$

Informed by surface effect in elasticity theory the surface stress, $\boldsymbol{\sigma}_s$, on elastic body satisfies the equation

$$\nabla_s.\boldsymbol{\sigma}_s + \boldsymbol{\varsigma}_s - \boldsymbol{\sigma}.\boldsymbol{n} = 0, \quad (12)$$

on the surface, where ∇_s is the gradient operator on the deformed surface, $\boldsymbol{\varsigma}_s$ is the external traction per unit area on the deformed surface, $\boldsymbol{n}$ is the unit vector along the normal direction of the deformed surface. With the invariants of the surface left Cauchy-Green deformation tensor in (3) which reads

$$\mathrm{I}_s = \left(\frac{r}{R}\right)^2 + \lambda_z^2 = \ \mathrm{II}_s = J_z^2, \quad (13)$$

where J_s is the ratio of the deformed surface area to the initial surface area, substituting (13) into (12) gives the surface stress in the form

$$\boldsymbol{\sigma}_s = \left[\sigma_0 + k_s\left(\frac{\lambda_s r}{R} - 1\right)\right]\mathbf{I}_s, \quad (14)$$

where σ_0 is the residual surface stress, and k_s is the surface stiffness. Let p_1 and p_2 be pressures applied on the inner and outer surfaces respectively, as shown in Fig. 1(c), and assume the condition of equation (12) is satisfied. Then the component of the stress, σ_{rr}, at the inner and outer surfaces has the boundary conditions given by

$$\begin{aligned}\sigma_{rr}\big|_{R=R_1} &= -p_1 + \frac{\sigma_0}{r_1} + k_s\left(\frac{\lambda_z}{R_1} - \frac{1}{r_1}\right),\\ \sigma_{rr}\big|_{R=R_2} &= -p_2 + \frac{\sigma_0}{r_2} + k_s\left(\frac{\lambda_z}{R_2} - \frac{1}{r_2}\right).\end{aligned} \quad (15\ \text{a, b})$$

Integrate (9) to get the stress, σ_{rr}, which satisfies the boundary condition (15a) as

$$\sigma_{rr} = -p_1 + \frac{\sigma_0}{r_1} + k_s\left(\frac{\lambda_z}{R_1} - \frac{1}{r_1}\right) + \left(\nu_1 + \nu_2\lambda_z^2\right)\frac{1}{2\lambda_z^2}\left[\frac{R^2}{r^2} - \frac{R_1^2}{r_1^2} + \lambda_z In\left(\frac{R^2}{R_1^2}\frac{r_1^2}{r^2}\right)\right]. \quad (16)$$

The stress, σ_{rr}, which satisfies the boundary condition (15b) is

$$p_2 - p_1 = \left(\nu_1 + \nu_2\right)\left[\begin{array}{l} -\lambda_k - \dfrac{\lambda_\sigma - \lambda_k}{1+\lambda_R}\lambda_{\hat{R}1}^{-1}\left\{1 + \left[1 + \left(\lambda_R^{-2} - 1\right)\lambda_{\hat{R}1}^{-2}\right]^{-\frac{1}{2}}\right\} \\ \qquad + \dfrac{1}{2}\lambda_{\hat{R}1}^{-2}\left\{1 - \lambda_R^{-2}\left[1 + \left(\lambda_R^{-2} - 1\right)\lambda_{\hat{R}1}^{-2}\right]^{-1}\right\} + \dfrac{1}{2}In\left\{\lambda_R^2\left[1 + \left(\lambda_R^{-2} - 1\right)\lambda_{\hat{R}1}^{-2}\right]\right\} \end{array}\right] \quad (17)$$

where $\lambda_R = \dfrac{R_1}{R_2}$, $\lambda_{\hat{R}1} = \dfrac{r_1}{R_1}$, $\lambda_\sigma = \dfrac{\sigma_0}{\nu_1 + \nu_2}\left(\dfrac{1}{R_1} + \dfrac{1}{R_2}\right)$, $\lambda_k = \dfrac{k_s}{\nu_1 + \nu_2}\left(\dfrac{1}{R_1} + \dfrac{1}{R_2}\right)$. Here λ_σ is the residual surface stress.

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This deformation-pressure relation is under non-elongation along the axial direction (in which $\lambda_z = 1$). A positive surface stiffness is essential for the stiffness of the whole cylinder to increase. In effect, a larger pressure or tensile load is necessary to attain the same deformation of the cylinder without any surface effect.

Penile response to blood flow

The response discussed here is that which is induced by increased flow due to excitation. Blood supply to the penis is through the internal pudendal arteries. In this section, penile deformation and buckling are considered.

Deformation

The following assumptions regarding the fluid shall apply:

(i)The fluid is Newtonian, viscous, incompressible, and homogeneous.

(ii)The flow is axisymmetric.

(iii)The flow in the angular direction is negligible.

(iv)The domain under consideration contains a fully developed flow.

It is assumed, from the standpoint of penile structure, that:

(i) The penile shaft consists of a single artery and a single vein. (All the arteries and veins are normalized as the dorsal artery and the dorsal vein — the outstanding artery and vein respectively).
(ii) The artery is thin-walled, elastic and with circular cross-section.
(iii) The artery is a homogeneous, isotropic membrane shell (i.e with negligible bending stress).

In a flaccid and pendulous state, let the aspect ratio (the ratio between the radius R and the length L of the artery) be $\varepsilon := R/L$ and for each fixed $\varepsilon > 0$, this state is the reference domain (see Figure 2) define by

$$\Omega_\varepsilon(t) = \{x \in \mathbb{R}^3 : x = (r\cos\theta, r\sin\theta, z),\ r < R + \eta^\varepsilon(z, t),\ 0 < z < L\} \qquad (18)$$

where η^ε is the radial displacement from the reference state. Define the arterial lateral boundary by

$$\Sigma = \{x = (R(z)\cos\theta, R(z)\sin\theta, z) \in \mathbb{R}^3 : \theta \in (0, 2\pi),\ z \in (0, L)\} \qquad (19)$$

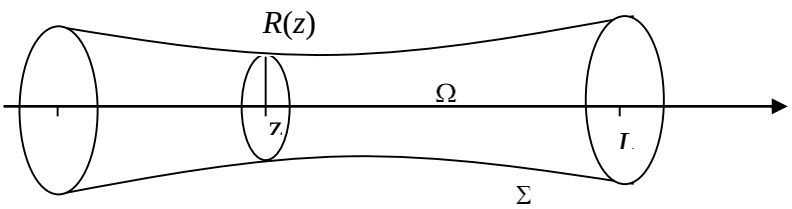

Fig. 2 Reference domain schematic

The reference configuration is assumed to be prestressed (it is of note that the penile shaft shrinks in response to variations in weather; the actual size under normal temperature is taken for a prestressed state).When penile stimulation occurs there is a chain of events: arterial dilatation results; increased blood flow into the trabeculae follows; the expansion of the sinusoids and accumulation of blood in the penis ensues. This results in the engorgement of the corpora cavernosa, the glans, and, to some extent, the corpora spongiosum. The consummation of all these is the penile erection.

The equations of flow, in the Eulerian formulation of $\Omega_\varepsilon(t) \times R_+$, in cylindrical co-ordinate, (R,θ, Z), which also governs flow during tumescence, read (Pedrizzetti and Dominichini (2003))

$$\rho\left\{\frac{\partial v_r^\varepsilon}{\partial t}+v_r^\varepsilon\frac{\partial v_r^\varepsilon}{\partial r}+v_z^\varepsilon\frac{\partial v_r^\varepsilon}{\partial z}\right\}-\mu\left(\frac{\partial^2 v_r^\varepsilon}{\partial r^2}+\frac{\partial^2 v_r^\varepsilon}{\partial z^2}+\frac{1}{r}\frac{\partial v_r^\varepsilon}{\partial r}-\frac{v_r^\varepsilon}{r^2}\right)+\frac{\partial p^\varepsilon}{\partial r}=0 \tag{20}$$

$$\rho\left\{\frac{\partial v_r^\varepsilon}{\partial t}+v_r^\varepsilon\frac{\partial v_z^\varepsilon}{\partial r}+v_z^\varepsilon\frac{\partial v_z^\varepsilon}{\partial z}\right\}-\mu\left(\frac{\partial^2 v_z^\varepsilon}{\partial r^2}+\frac{\partial^2 v_z^\varepsilon}{\partial z^2}+\frac{1}{r}\frac{\partial v_z^\varepsilon}{\partial r}\right)+\frac{\partial p^\varepsilon}{\partial z}=0 \tag{21}$$

with the continuity equation,

$$\frac{\partial v_r^\varepsilon}{\partial r}+\frac{\partial v_z^\varepsilon}{\partial z}+\frac{v_r^\varepsilon}{r}=0 \quad \text{in } \Omega_\varepsilon\times R_+ \tag{22}$$

In the equation above v_r, v_z are the radial and longitudinal components of the fluid respectively, μ is the viscosity of the fluid, as it will be all through, p is the pressure and ρ is the density. Equation (22) represents the incompressibility condition, *div* $v = 0$. It is important to note that the equation of flow above holds well for increased flow that brings about the increase in intracavernosal pressure above the flaccid state. The increased pressure may represent average blood pressure values within the corpora in the course of tumescence.

The first stage of interaction is the interface of blood with the arterial wall. The interaction has dual effects: the blood cells and the vessel wall deform. The action of the fluid on the wall is described by Milne-Thomson (1974),

$$\mathbf{F}=\mathbf{n}\left(p-\frac{4}{3\mu}\nabla\mathbf{q}\right)+\mu\mathbf{n}\wedge\Im \tag{23}$$

where $\boldsymbol{q}$ is the fluid velocity at X and $\Im$ is the vorticity (for non-vortex flow, $\Im = 0$).

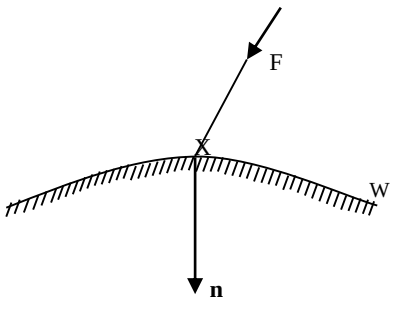

Fig. 3 Fluid action on the wall

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In the cylindrical polar coordinates (R, θ, Z) the position vector of a point on the artery reads

$$\boldsymbol{B} = R_0\boldsymbol{e}_r + Z\boldsymbol{e}_z \tag{24}$$

where R_0 is the reference radius, $\boldsymbol{e_r}$, $\boldsymbol{e_\theta}$ and $\boldsymbol{e_z}$ are the unit base vectors in the cylindrical polar coordinates and Z is the axial coordinates of a material point in the natural state. Assuming the artery is subjected to an axial stretch ratio λ_z and a static pressure $P_0(Z)$ then the deformation is described by Fung (1993),

$$\boldsymbol{r}_0 = \left[r_0 - f(z)\right]\boldsymbol{e}_r + z\boldsymbol{e}_z, \quad z = \lambda_z Z \tag{25}$$

where z is the axial coordinate at the intermediate configuration, r_0 is the deformed end radius at the origin, and $f(z)$ is the post-deformation stenosis function. The deformation stimulates an arc in the meridional and circumferential directions in the form (Demiray (1976)),

$$ds_z^0 = \left[1 + (-f')^2\right]^{1/2} dz, \quad ds_\theta = [r_0 - f(z)]\, d\theta, \tag{26}$$

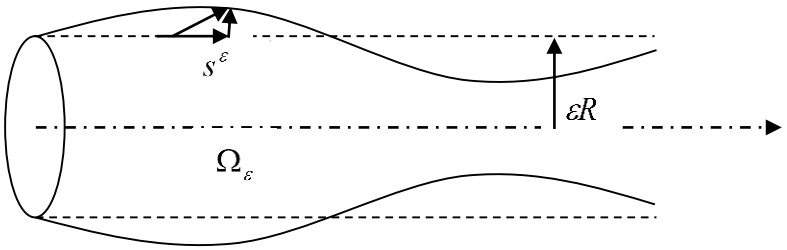

Fig.4 Wall displacement

where a prime denotes the differentiation of the corresponding field variable with respect to z. Suppose that there is negligible axial displacement and let $v(z, t)$ be a finite dynamical displacement superimposed on the initial static deformation. The position vector $\boldsymbol{r}$ of a generic point on the tube is

$$\boldsymbol{r} = [r_0 - f(z) + v(z, t)]\boldsymbol{e}_r + z\boldsymbol{e}_z \tag{27}$$

Now the dynamic deformation stimulates the resulting meridional and circumferential arc lengths respectively:

$$ds_z = \left[1 + \left(-f' + \frac{\partial v}{\partial z}\right)^2\right]^{1/2} dz, \quad ds_\theta = [r_0 - f(z) + v(z, t)]d\theta \tag{28}$$

The connected effect of meridional and circumferential changes due to tumescence is the mechanical compression of the emissary vein (note that there are several of such veins; here one vein was assumed), which drain trabeculae against the tunica albuginea. This is the fundament of veno-occlusion. The flow through the occluded vein may be modelled as flow through a critically tapered duct. The vein is considered to play a passive role in the erection process **(**Robert and Tom (2005)), and therefore the model is not considered here.

The meridional and circumferential curves in the deformed configuration have the equivalent stretch ratios:

$$\lambda_1 = \lambda_2\left[1+\left(-f+\frac{\partial v}{\partial z}\right)^2\right]^{1/2}, \qquad \lambda_2 = \frac{1}{R_0}[r_0 - f(z)+v(z,t)] \tag{29}$$

The unit tangent ***T*** along the deformed meridional curve and the unit outward normal ***n*** to the deformed membrane are given by

$$\mathbf{T} = \frac{\left(-f'+\frac{\partial v}{\partial z}\right)\mathbf{e}_r + \mathbf{e}_z}{\beta}, \quad \mathbf{n} = \frac{\mathbf{e}_r - \left(-f'+\frac{\partial v}{\partial z}\right)\mathbf{e}_z}{\beta}, \tag{30}$$

where the function β is defined as $\beta = \left[1+\left(-f'+\frac{\partial v}{\partial z}\right)^2\right]^{1/2}$.

By assuming material incompressibility, the thickness *H* and *h*, before and after final deformation is such that $h = \frac{H}{\lambda_1\lambda_2}$

Let M_1 and M_2 be membrane forces along the meridional and circumferential curves respectively. Consider a small tube segment located between the planes *z* = constant and *z* + *dz* = constant, θ = constant and $\theta + d\theta$ = constant. The radial motion of such a segment may be described by

$$\frac{\partial}{\partial z}\left[\frac{M_1}{\beta}(r_0 - f + v)\left(-f'+\frac{\partial v}{\partial t}\right)\right] - M_2\beta + \beta(r_0 - f + v)F_r = \rho_w \frac{H}{\lambda_z} R_0 \frac{\partial^2 v}{\partial t^2} \tag{31}$$

where ρ_w is the volumetric mass of the membrane material, and F_r is the radial fluid reaction force on inner surface of the tube given by

$$F_r = \frac{-h(\varepsilon)E(\varepsilon)}{1-\gamma^2}\frac{\eta^\varepsilon}{R^2} + h(\varepsilon)G(\varepsilon)\kappa(\varepsilon)\frac{\partial^2\eta^\varepsilon}{\partial z^2} - \rho_w h(\varepsilon)\frac{\partial^2\eta^\varepsilon}{\partial t^2} \tag{32}$$

where $E(\varepsilon)$ is Young's modulus of elasticity, $h(\varepsilon)$ is the wall thickness, κ is the Timoshenko shear correction factor (see Iemma and Pontrelli (2004)), *G* is the shear modulus, γ (= 0.5 for an incompressible material) is the Poisson ratio.

In equation (32) above the right-hand terms are described as follows: the first term is the *elastic response function*, the second term is related to the *radial pre-stress state* of the tube and the third term is the *inertia* term that is proportional to the radial acceleration of the tube wall.

If $\tau\Sigma^*$ be the strain energy density function of the tube material, where τ is the shear modulus, then the membrane forces M_1 and M_2 are expressed in terms of the stretch ratios as

$$M_1 = \frac{\tau H}{\lambda_2}\frac{\partial\Sigma^*}{\partial\lambda_1}, \; M_2 = \frac{\tau H}{\lambda_1}\frac{\partial\Sigma^*}{\partial\lambda_2} \tag{33}$$

By using equation (33) in equation (32) the equation of the tube in the radial direction is given by

$$\tau R_0\frac{\partial}{\partial z}\left\{\frac{\left(-f'+\partial v/\partial z\right)}{\beta}\frac{\partial\Sigma}{\partial\lambda_1}\right\} - \frac{\tau}{\lambda_z}\frac{\partial\Sigma}{\partial\lambda_2} + \frac{\beta\mathbf{F_r}}{\mathrm{H}}(r_0 - f + v) - \rho_w\frac{R_0}{\lambda_z}\frac{\partial^2 v}{\partial t^2} = 0 \tag{34}$$

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By neglecting the elastic deformations in the azimuthal direction, the evolution of the response of the lateral boundary to flow is of the form (Iemma and Pontrelli (2004)), Jerrold and Thomas (1983))

$$\left.\begin{aligned}\rho_w h\frac{\partial^2 v_z}{\partial t^2} &= \frac{Eh}{1-\gamma^2}\left(\frac{\gamma}{R}\frac{\partial v_r}{\partial z}+\frac{\partial^2 v_z}{\partial z^2}\right)+\beta_z \\ \rho_w h\frac{\partial^2 v_r}{\partial t^2} &= \kappa G_s h\frac{\partial^2 v_r}{\partial t^2}-\frac{Eh}{1-\gamma^2}\left(\frac{\gamma}{R}\frac{\partial v_z}{\partial z}+\frac{v_r}{R^2}\right)+\beta_r\end{aligned}\right\} \qquad (35\ a, b)$$

where R is the arterial reference radius at rest, ρ_w is the arterial volumetric mass, β is the forcing term due to the external forces, including the stress from the fluid. The inviscid fluid model entails $\beta_z = 0$. If a negligible longitudinal deformation is assumed, eqn. (35 a) is inevitably satisfied. Therefore,

$$\rho_w h\frac{\partial^2 v_z}{\partial t^2} = \kappa G_s h\frac{\partial^2 v_r}{\partial z^2}-\frac{Eh}{1-\gamma^2}\frac{v_z}{R^2}+\beta_r \quad \text{in } \Sigma\times(0,L)\,. \qquad (36)$$

The transverse elastic effect is produced by radial displacement (second term on the right-hand side of eqn. (36)), and represents the contribution of the restoring force due to the deformation of the annular sections of the vessel. It is noteworthy that this model is based on a Lagrangian description of the motion of the elastic wall, referred to a material domain $\Omega(0)$, corresponding to the rest state, where $v_r = v_z = 0$.

The effectuation of tumescence

As earlier stated, collagen and elastin fibres permit an increase in penile girth and length during tumescence. The collagenous tissue is considered here as a continuum composite material consisting of one or numerous groups of oriented collagen fibres embedded in an extremely compliant isotropic solid matrix. The collagen fibre alignment is defined by the so-called *structural* or *structure tensor* which gives the impression as an argument of the strain energy function (Spencer (1992)) The kinematic model is represented by the deformation gradient **F** defined as:

$$\mathrm{F}(\mathbf{X}) := \frac{\partial\varphi(\mathbf{X})}{\partial\mathbf{X}} = \sum_{i,I=1}^{n}\frac{\partial\varphi_i}{\partial \mathrm{X}_\mathrm{I}}\mathbf{e}_i\otimes\boldsymbol{E}_I\,, \qquad (37)$$

where X is the position of a material point on the collagen fibre in the *Lagrangian* (reference) configuration, $\mathbf{x} = \phi(\mathbf{X})$ is its material location in the *Eulerian* (*current*) configuration. $\mathbf{E}_I$ and $\mathbf{e}_i$ are fixed orthonormal bases in the Lagrangian and Eulerian configurations, respectively. **F** maps infinitesimal line vectors from the material to spatial configuration while cofactor (**F**) = $J\cdot\mathbf{F}^{-\mathrm{T}}$ and J = det (**F**), respectively, maps oriented infinitesimal surface and volume. The *principle of material frame indifference*, (Limbert (2017)), is defined by the right and left Cauchy–Green deformation tensors, $\mathbf{C}=\mathbf{F}^{\mathrm{T}}\cdot\mathbf{F}$ and $\mathbf{b}=\mathbf{F}\cdot\mathbf{F}^{\mathrm{T}}$ respectively. These tensors only contain information about a change in fibre length, with no local material rotations.

Let $\boldsymbol{g}(z, t, \boldsymbol{n})$ be the force per unit area at point z at time t across the tunica surface element with unit normal $\boldsymbol{n}$; that is to say, $\boldsymbol{g}$ is the force per unit deformed area produced by the material outside (the $+\boldsymbol{n}$ direction) acting on the material inside (the $-\boldsymbol{n}$ direction). By balance of linear momentum the response of any tunical sub-body $\omega \subset$ B (the tunica body) to venous compression obeys the continuum analog of Newton's second law (Jerrold and Thomas (1983)) reads:

$$\frac{d}{dt}\int_{\omega_t} \rho_t \boldsymbol{v} dz = \int_{\partial\omega_t} \boldsymbol{g}(z,t,\boldsymbol{n}) da + \int_{\omega_t} \rho_t \boldsymbol{b} dz \quad , \tag{38}$$

where $\boldsymbol{b}(z, t)$ is the applied body forces per unit mass on the tunica, which may be negligible. The force $\boldsymbol{g}$ depends linearly on $\boldsymbol{n}$ if the balance of momentum holds, by Cauchy's theorem. Thus, there is a two-tensor $\boldsymbol{\tau}_t(z, t)$ such that $\boldsymbol{g}(z, t, \boldsymbol{n}) = \boldsymbol{\tau}_t(z, t)\boldsymbol{n}$. Substituting $\boldsymbol{g} = \boldsymbol{\tau}_t \cdot \boldsymbol{n}$ into the balance of linear momentum and applying the divergence theorem gives

$$\frac{d}{dt}\int_{\omega_t} \rho_t \boldsymbol{v} dz = \int_{\partial\omega_t} div\boldsymbol{\tau}_t dz + \int_{\omega_t} \rho_t \boldsymbol{b} dz \, . \tag{39}$$

The arbitrariness of ω_t and conservation of mass permits Cauchy's equation of motion of the tunica in the form

$$\rho_t \dot{\boldsymbol{v}} = div\boldsymbol{\tau}_t + \rho_t \boldsymbol{b} \, . \tag{40}$$

where the subscript, t, relates the parameters to the tunica. Now consider the equation

$$(h_t - \Delta h_t H(\zeta_z))\left[\rho_t \frac{\partial^2 v_z}{\partial t^2} = \kappa_t G_t \frac{\partial^2 v_z}{\partial z^2} - \frac{E_t}{1-\gamma_t^2}\frac{v_z}{Z^2} \right] \quad \text{in } \Sigma^* \times (0, L + \Delta L[H(\zeta_z)]) \, , \tag{41}$$

where the Heaviside function $H(\zeta_z)$ reads

$$H(\zeta_z) := \begin{cases} 1, & \zeta_z > 0 \\ 0 & \zeta_z \le 0 \end{cases} \quad , \tag{42}$$

and all t-subscripts represent tunica; Δh_t is the change in thickness of elastic fibre within the tunica, ΔL is the associated change in of the penile shaft; ξ_z is an ensemble of forcing term due to the external forces, including the stress from the fluid partly due to venous occlusion. Other quantities in (41) retain their assigned codes. Now, while the length of the flaccid penis is in the set $(0, L)$, the set $(0, L+\Delta L)$ represents the length (stretch) of the stiff, saturated, and erect penis as it angles out from the body. Equation (41) is insightful; when the forcing term applies, tumescence induces a reduction in the tunical thickness, h_t, due to tunical elongation. Thus, $h_t - \Delta h_h$ indicates a reduction in the tunical thickness. In this case, $H(\zeta_z) = 1$. When the forcing term is remove (i.e. $H(\zeta_z) = 0$), as is the case of seminal ejaculation with the concomitant opening of the venous segment, the equation (41) reduces to

$$\rho_t h_t \frac{\partial^2 v_z}{\partial t^2} = \kappa_t G_t h_t \frac{\partial^2 v_z}{\partial z^2} - \frac{E_t h_t}{1-\gamma_t^2}\frac{v_z}{Z^2} \qquad \text{in } \Sigma \times (0, L) \quad , \tag{43}$$

which is a return to the penal flaccid state (detumescense).

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As a rule, a post-ejaculation virtual restorative force applies to the elastin. The tunical motion has the similitude of a *harmonic oscillator*; therefore the axial displacement, $z = z(t = tunica)$, may be described by the second-order linear differential equation

$$\ddot{z}(t) + \frac{c}{m} z = 0 \ . \qquad (44)$$

The general solution of equation (44) is

$$z(t) = C_1 \sin\left(t\sqrt{\frac{c}{m}}\right) + C_2 \cos\left(t\sqrt{\frac{c}{m}}\right), \qquad (45)$$

where c is the force constant that depends on the fibre material, m is the elastin fibre mass. The displacement, z, encodes the distance and direction the fibre is deformed from its equilibrium length. The arbitrary constants, C_1 and C_2 may be determined by considering the initial displacement and velocity of the mass.

Assume the following initial conditions on equation (44)

$$z(0) = l; \quad z'(0) = l^* \ . \qquad (46)$$

Then equation (45) becomes

$$z(t) = l^* \gamma^{-1/2} \sin\sqrt{\gamma}t + l\cos\sqrt{\gamma}t \ , \qquad (47)$$

where $\gamma = c/m$. Equation (47) holds well for the axial length of the penile shaft depending on the tunical characteristics.

Empirical details (Veale et al.(2014)) suggest that there is a 42.94 % (approx. 2 p.d.) average extension of the penile shaft from a flaccid state to erect state, while the change in girth from the former to the later state is approximately 25.41%. (This is to say that the erect to flaccid length ratio, (L_E/L_F), is approximately 1.43:1 units, while the erect to flaccid girth ratio, (V_E/V_F), is approximately 1.25:1)

Notion of penile and buckling

A phenomenon of interest is the so-called penile buckling. Fung (1993) (see Hai-Chao et al. (2013)) derived the buckling equation for a generic thin-walled circular cylindrical tube under critical transmural pressure as

$$(p_e - p)_{crit} = \frac{3}{R^3}\frac{Eh^3}{(12(1-\varsigma^2))} \qquad (45)$$

where p_e is external pressure, p is internal lumen pressure. In (45) above $Eh^3/(12(1-\varsigma^2)) = E'I$ encodes the flexural rigidity of the thin-walled tube, where E is Young's modulus of the material, $I = h^3/12$, is the cross-sectional area moment of inertia per unit length, h is wall thickness, ς is Poisson's ratio, and R is tube median radius at zero transmural pressure. The penile shaft is considered as "not long", and so are its vessels. For tubes in such category, the *Von Mises* expression for combined radial and axial critical pressure reads:

$$p_{crit} = \frac{1}{n^2 - 1}\left[\left(\left[n^2 + \left(\frac{\pi D}{2L}\right)^2\right]^2 - l_{1,2}\right) \times \frac{1}{3}\frac{2E}{1-\varsigma^2}\left(\frac{h}{D}\right)^3 + \frac{2E}{D}\rho^{-2}\right] \tag{46}$$

where n is the number of buckling waves in the circumferential direction, $l_{1,2} = 2l_1 n^2 + l_2$,

$l_1 = (1+(1+\varsigma)\rho)(2+(1-\varsigma)\rho)$, $l_2 = (1-\rho\varsigma)\left[1+\rho(1+2\varsigma)-\varsigma^2(1-\varsigma^2)\left(1+\frac{1+\varsigma}{1-\varsigma}\rho\right)\right]$, $\rho = \frac{1}{\left(\frac{2nL}{\pi D}\right)^2 + 1}$.

If the critical buckling pressure and other parameters are known, then the pressure-lumen relationship for arteries developed by Drzewiecki *et al*.(1997) may be determined by

$$(p - p_e) - p_{buc} = A_1(e^{A_2(\frac{A-A_{buc}}{A_{buc}})} - 1) - \frac{EI}{R^3}\left[\left(\frac{A_{buc}}{A}\right)^{m_*} - 1\right] \tag{47}$$

where A_1 and A_2 are material constants. A is the cross-sectional area, A_{buc} is the cross-sectional area at critical buckling pressure, and p_{buc} is the critical buckling pressure, m_* is a constant.

Udelson *et al*. (1998, 1999) treated penile deformation from the standpoint of column buckling. In the event of buckling the original shape of a structure cannot withstand added loads. Therefore the structure changes its shape in a bid to find a new equilibrium configuration. Udelson *et al.* (1998) described penile buckling forces as *the magnitude of the axially compressive force applied to the glans of an erect penis, resulting in evident curving such that any added small force would prompt collapse (buckling) of the erect shaft.* Penile buckling was treated with the following assumptions:

(i) The shaft has a circular cylindrical form.
(ii) The neutral axis lies on the diameter.
(iii) The tissue is isotropic.
(iv) The modulus of elasticity, E, of the pendulous penis is the same as that of the corpora cavernosa.
(v) The same as above holds for the volume to flaccid volume ratio, V/V_F.
(vi)The expansion ratios of length to flaccid length (L/L_F) are the same in the axial and

The radial directions, (D/D_F).

The following penile buckling formula was derived [5, 6]:

$$F_{Buc} = \frac{3}{64}\pi^3(2\zeta - 1)\left(\frac{D}{L}\right)_P^2 \frac{(D_F)_P^2}{X} \times \left[\left(\frac{V_E}{V_F}\right)_P + \chi_P e^{-X(\Delta p)}\right]^{2/3} \times \left[1 - \frac{e^{X(\Delta p)}}{\chi_P}\right], \tag{48}$$

,

where F_{Buc} is the penile buckling force; D is the diameter; L is the length; Δp is the increase in intracavernosal pressure above the flaccid state; ζ is the (cavernosal) Poisson's ratio; V_E/V_F is the distensibility (i.e. erect to flaccid volume ratio); X is the cavernosal expandability and $\chi = \left(1 - \frac{V_E}{V_F}\right)$. In (48) the subscripts represent the following: '*E*' = *erect* penis, *'P'* = *pendulous*, '*F*' = *flaccid*. The only term on the right-hand side with dimensions is D_F^2 / X. While D has a dimension of length, X has a dimension of pressure.

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The study by Udelson et al. (1998) seems to indicate that the penile shaft was treated as a solid cylinder with constrained *closed ends* (see Udelson et al. (1998), Fig.1, p.26). It also depicted that for buckling to occur an axially compressive force must be applied to the glans of an erect penis. There is an inherent feeling that the axial compressive force prevailing on the buckling pipe seems to conflict with the fact that the ends are pinned or fixed. The mechanics of *coition* requires repetitive penile thrust in the vagina, as long as the penis is excited until orgasm and ejaculation occur. Internal pressure gradient drives the hydrodynamic flow that creates erection in a normal subject. It is also true that an erect (and stiff) penis angles out of the pendulous state; buckling analysis may well take cognizance of the angle. A good way to consider the pressure effects in pipe buckling is to evaluate an equivalent force in the axial direction related to pressure loads, as detailed by Craveiro and Neto (2016). However, this must be done with restraint since pipelines with no end caps subject to internal pressure have the tendency of contraction in the axial direction**.** In the absence of restriction in the pipe the existing tensile hoop strain generates, as a consequence of Poisson effect, a compressive strain in the axial direction. It was argued that the force that governs the buckling of pipe is not the real force specified by the integration of the stresses on the cross-section (Craveiro and Neto (2016), Fyrileiv O. and Leif (2005)), but by the so-called *effective axial force*, which becomes compressive owing to internal pressure. Critical internal pressure is an important factor in buckling. The longitudinal stress set up in a straight thin-walled axially constrained pipe by internal pressure, p, is $\varrho pD/2h$. The resultant force over a complete cross-section perpendicular to the tube axis is $(\pi/4)D^2p(1 - 2\varrho)$, which is compressive, and the difference between the compressive force $(\pi/4)D^2p$ carried by the fluid within the pipe and the tensile force $(\varrho pD/2h)\ (\pi\ Dh)$ carried by the wall of the pipe, $\upsilon < 1/2$. The pipe may be seen as a column carrying this resultant compressive force, and buckling may occur when the force reaches the Euler buckling load (see Palmer (n.d)). This occurs when

$$\left(\frac{\pi}{4}\right)D^2p(1-2\zeta) = 4\pi^2\left(\frac{(\pi ED^3h)/8}{L^2}\right) \qquad (49)$$

where $(\pi ED^3h)/8$ is the flexural rigidity. Thus,

$$p = \frac{2\pi^2 EDh}{(1-2\zeta)L^2} \qquad (50)$$

Let the resultant force applied by the enclosed fluid pressure on the pipe wall be $\boldsymbol{P}ds$. As the fluid element must be in equilibrium, resolving forces perpendicular to the pipe axis reads:

$$\boldsymbol{P}ds = \left(\frac{\pi}{4}\right)D^2\,pd\varphi\,,$$

or

$$\boldsymbol{P} = \left(\frac{\pi}{4}\right)D^2\,pd\varphi\Big/ds\,, \qquad (51)$$

where ds, *is* an element of the pipe wall and φ represents the inclination of the deflected pipe to its original line. Thus, the enclosed fluid exerts a lateral force on a deflected pipe. The magnitude of the force per unit length is the product of pressure and the cross-section and the curvature, and it acts in the direction of the outside of the curve. Analysis of buckling by investigating the pipe deflection from its initial position must be cognizant of the existence of this force.

SUMMARY AND DISCUSSIONS

Penile blood flow and the reaction of the penile structures to flow were given a theoretic treatment. The flow volume was assumed to be on physiological increase owing to excitation and the erection that precede vaginal intromission. The neuronal activities that aid the chain of events that culminate in erection are not within the purview of this work. Blood flow was seen to cause elastic deformation of the vascular and non-vascular structures of the penis. For ease of analysis, the myriads of the penile arteries and veins were normalize in each of the two vascular structures. This step is adroit since the other arteries and veins are tributaries to the main artery and vein named earlier. In treating vascular behaviour during tumescence and erection, the vein is considered a passive member. This is so because the vein did not elect to constrict during the so-called veno-occlusion when it is pressed against the tunica. The tunica albuginea, which houses the collagen and elastin fibres is "touted" to be the bastion of the mechanical properties of the penile shaft. It is of note that the rheology of the entrapped blood as per deformation is not treated here as the vascular inlet, outlet, and wall boundary conditions are only necessary when flow characteristics and quantity are of the essence.

The question of what determines penile rigidity engaged attention. Which one of *axial compression* and *radial compression* could be advanced? It is expected that penile (mal-) functionality be treated from the standpoint of the causes rather than the effects. A compressive strain in the axial direction develops due to tensile hoop strain in the absence of restriction in a pipe (i.e. in the absence of end caps). An effective (virtual) axial force that becomes compressive, due to internal pressure, prevails in fluid-carrying ducts. Udelson *et al.* (1998) described penile buckling forces as the magnitude of the axially compressive force applied to the glans of an erect penis, resulting in evident curving such that *any added small force* would prompt collapse (buckling) of the erect shaft. For a virile subject, penile shaft buckling occurs just after seminal emission. After emission comes *detumescence*. The main cause of penile collapse is detumescence- a situation in which the penile artery narrows and the vein enlarges, thereby draining blood from the penis. In effect, the shaft returns its pendulous state. Therefore, detumescence implies withdrawal of force rather than a further application of force *on the glans.* This seems contrary to the position held by Udelson *et al.* (1998).

The response of the tonica albuginea's members−collagen and elastin to detumescence, which is in a sense a *retrograde flow*, is essential in determining their overall penile system compliance. When blood rheology is physiological, both the arterial and venous blood cannot be a culprit in the inability to accomplish tumescence and detumescence. Therefore the tunica albuginea and its members may be investigated. As a rule, the vein must be free from *occlusion* for outflow to occur. In the event of persisting veno-occlusion after seminal emission, one is left to surmise that the tunical restorative capability may have been compromised. Therefore, non-ischaemic *priapism* may be blamed on the failure of tunical restorative capability to unclog the vein. It is hereby suggested, from the standpoint of rheology, that deliberate localized blood- thinning under a clinical

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watch be conducted as a way to decongest the clogged vein. This would in turn improve the mechanics of the tunica.

As for impotence, any or both of two main factors come into play: (i) inadequate blood inflow (which may be due to poor innervation, vascular insufficiencies, among a group of pathologies as well as psychological issues), (ii) no/poor veno-occlusion. A case-by-case study is required to address each offending circumstance. Measures that improve blood flow, especially to the organ, are the panacea to inflow insufficiency. It is of note that frequent tunical stretch could enhance the elasticity of the elastin fibres. Subjects of (very) weak penile erection, who could afford the barest intromission are therefore most likely to attain virility through increased coital frequency. This is due to the increased stimulation of the elastin fibres and the associated increase in blood inflow.

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