

Arterial Pulse Waveform under the watch of Left Ventricular Ejection time: A physiological outlook

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Abstract

The behavior of arterial pulse waves was studied in connection with time interval at different phases of propagation. The essence of the study was to have a clue about the incidence of the time of pulse wave propagation on cardio-vascular parameters. Model analysis shows that arterial waveforms behave like solitons. It was seen, from the soliton solution of the arterial pulse waveform, that time interval between the phases of propagation, which corresponds with left ventricular ejection time (LVET), could supply some information about apparent pathogenesis.

Keywords: pressure; waveform; soliton; incompressible; mathematical; physiology.

1.0 Introduction

Arterial pulse wave is a physiological wave that propagates through arterial compartments. Pressure and flow pulses are created by the dynamics of the left ventricle in the event its intermittent ejection of blood into the aorta, and the subsequent aortic effusion into the arterial network. Waves propagate in arteries as a result of fluid-structure interaction (FSI). There is a balance between the inertial force of the fluid (blood) and the restoring force of the arterial wall. Physiological pulses undergo well defined changes in their waveform when they propagate away from their source [1, 2]. From the foregoing, observed changes in waveform that are not well defined, whether in space or time, should engage physiologic attention. In recent times scientific and medical literatures agree that arterial pulse wave analysis is paradigmatic in patient's clinical details. To this end, studies on pulse wave (PW) propagation were conducted in earlier times [3, 4, 5] and much later [6] in frequency domain. In such cases studies on wave propagation were mainly done through impedance analysis by means of Fourier transform. Interesting work was done by Wang et al. [7] in using frequency harmonics in the analysis of pulse wave. The Fourier modes suppose linearity and periodicity. Analysis of pulse wave in time domain has also been carried out [8, 9, 10, 11].

Parker et al. [12] used the method of characteristics to describe a one-dimensional (1D) arterial wave in time domain. This method does not assume linearity and/or periodicity. In general, time domain analysis is not frequency-based. In this work, pulse waveform was studied in relation to LVET in order to determine its effect on cardiovascular physiology. The spatial domain is the arterial length occupied by the pulse wave, which is apparently dependent on the stature of an individual subject. Recently, Nzerem and Alozie [13] studied arterial pulse wave morphology in spatial domain. The study motivated the present interest on the need to deal on the incidence of LVET on the arterial waveforms.

The aspect of this study is hemodynamics. Hemodynamics is the physical study of flowing blood (fluid) and the entire solid structures (arteries) through which it flows [2]. Literatures [14, 15, 16, 17] agree that Korteweg de-Vries (KdV) equation holds well for arterial pulse waves. In Crepeau and Sorine [14] the goal was to interpret pulse wave characteristics and to provide basis for estimations from non-invasive measurements. Laleg et al. [15] decomposed pressure into a travelling wave which is evident during systolic phase and a windkessel flow during the diastolic phase. Many important physiological interests are explained by PW. O'Rourke and Pauca [18] showed that pulse wave analysis (PWA) is a choice descriptor of physiological states. Wei et al. [19] used it in explaining the resonance of the organs with the heart. The lacuna is this: neither the literatures we have presented here nor has any other work, as much as we know at present, dealt on mathematical theory of pulse wave morphology by means of LVET. This work will, as much as possible, supply the missing link.

In section 2.0 vital elements of FSI were treated. The equation relating pressure to shear modulus was derived. Section 3.0 discussed the emergence of arterial pulse waves in the event of left ventricular ejection. The so called Korteweg de-Vries (KdV) equation was derived. Consequently, soliton-solution, which is characteristic of the KdV equation, was obtained. Two soliton are enough to describe the 'peaking' and 'steepening' wave

phenomena. In section 4.0, a physiological analysis of the resulting pressure waveforms was made. In section 5.0, conclusion was drawn based on the analysis.

2.0 Equations of fluid-structure interaction

Flow inside deformable domains and the resulting motion of the domains is considered. Fig. 2.1 shows the nondeformed arterial configuration. Define the arterial reference domain $\Omega_\varepsilon(t)$ by [20]

$$\Omega_\varepsilon(t) = \{x \in \mathbb{R}^3 : x = (r \cos \theta, r \sin \theta, z), r < R + \eta^\varepsilon(z, t), 0 < z < L\}, \quad (2.1)$$

and define the lateral boundary by

$$\Sigma = \{x = (R(z) \cos \theta, R(z) \sin \theta, z) \in \mathbb{R}^3 : \theta \in (0, 2\pi), z \in (0, L)\}, \quad (2.2)$$

where R and r are the tube's outer and inner radii respectively, L is the tube's length, $\varepsilon = R/L$, η^ε is the radial displacement from the reference state. We assume the flow in $\Omega_\varepsilon(t)$ is fully developed and axisymmetric, with no angular velocity. In Eulerian formulation the equations of momentum are of the form:

$$\rho \left\{ \frac{\partial v_r^\varepsilon}{\partial t} + v_r^\varepsilon \frac{\partial v_r^\varepsilon}{\partial r} + v_z^\varepsilon \frac{\partial v_r^\varepsilon}{\partial z} \right\} - \mu \left(\frac{\partial^2 v_r^\varepsilon}{\partial r^2} + \frac{\partial^2 v_r^\varepsilon}{\partial z^2} + \frac{1}{r} \frac{\partial v_r^\varepsilon}{\partial r} - \frac{v_r^\varepsilon}{r^2} \right) + \frac{\partial p^\varepsilon}{\partial r} = 0, \quad (2.3)$$

$$\rho \left\{ \frac{\partial v_z^\varepsilon}{\partial t} + v_r^\varepsilon \frac{\partial v_z^\varepsilon}{\partial r} + v_z^\varepsilon \frac{\partial v_z^\varepsilon}{\partial z} \right\} - \mu \left(\frac{\partial^2 v_z^\varepsilon}{\partial r^2} + \frac{\partial^2 v_z^\varepsilon}{\partial z^2} + \frac{1}{r} \frac{\partial v_z^\varepsilon}{\partial r} \right) + \frac{\partial p^\varepsilon}{\partial z} = 0. \quad (2.4)$$

The continuity equation representing the incompressibility condition, $\text{div } v = 0$ is

$$\frac{\partial v_r^\varepsilon}{\partial r} + \frac{\partial v_z^\varepsilon}{\partial z} + \frac{v_r^\varepsilon}{r} = 0, \quad (2.5)$$

where v_r, v_z are the radial and longitudinal components of the fluid velocity respectively, μ is the viscosity of the fluid, p is the pressure and ρ is the density.

The lateral boundary responds to fluid flow. The artery responds to stress in the tangential plane that are assumed uniform across the wall thickness (Humphrey, [21]). At this point, we conceive of the arterial wall as a membrane, having a negligible mass and, thus, no bending stress [22]. Arteries undergo large deformation. The forces acting on the membrane make it to deform and reach an equilibrium state (see Fig.2.2).

It is noteworthy that Navier-Stokes equations that govern fluid motion are usually studied in the Eulerian (or spatial) domain, whereas the elastic body is studied in the Lagrangian (or material) frame. The Lagrangian framework has the benefit of keeping the hyperbolic problem linear, as the case may require. When the description of the boundary in the fluid problem does not coincide with the description of the same boundary in the solid mechanics problem, Pedrizetti [23] provides the panacea for the fluid-solid coupling: Rewrite the membrane problem in Eulerian terms by introducing the function $H(t, z)$. Then the following Lagrangian-Eulerian transformation is used

$$R[t, z(t, s)] = r(t, s), \quad H[t, z(t, s)] = s \quad (2.6)$$

where $R(z,t)$ and $H(z,t)$ are the Eulerian counterparts of the Lagrangian co-ordinates of a particle of the membrane, s is the material co-ordinate. The governing equations of fluid-membrane equilibrium in tangential and normal directions are of the form [23, 24]

$$\begin{aligned} R'(T_1 - T_2) + RT_1' &= \tau R(1 + R'^2)^{1/2} \\ \frac{-R''}{(1 + R'^2)^{3/2}} T_1 + \frac{1}{R(1 + R'^2)^{1/2}} T_2 &= p \end{aligned} \quad (2.7)$$

where the prime indicates a z -derivative, T_1 and T_2 are non-dimensional membrane stresses in meridional and circumferential directions respectively, and τ is the fluid viscous shear stress exerted on the wall. The principal deformation ratios in the tangential plane are

$$\lambda_1 = \sqrt{\frac{1 + R'^2}{H'^2}}, \quad \lambda_2 = \frac{R}{R_u} \quad (2.8)$$

where R_u is the undeformed radius.

Using Lagrangian description of the motion of the elastic wall referred to a material domain Ω (0), corresponding to the rest state, where $v_r = v_z = 0$, the evolution of the response of the lateral boundary to the fluid flow is described by [25, 26]

$$\rho_w h \frac{\partial^2 v_z}{\partial t^2} = \frac{Eh}{1 - \sigma^2} \left(\frac{\sigma}{R} \frac{\partial v_r}{\partial z} + \frac{\partial^2 v_z}{\partial z^2} \right) + \chi_z \text{ in } \Sigma \times (0, L) \quad (2.9)$$

where, h is the arterial wall thickness, E is the Young modulus of elasticity, R is the arterial reference radius at rest, ρ_w is the arterial volumetric mass, σ is the Poisson ratio, χ is the term due to the external forces, including the stress from the fluid.

Suppose only radial displacement of the lateral wall is admissible: $\Sigma_\varepsilon(t) = \{r = R + \eta^\varepsilon(z, t)\} \times (0, L)$. The radial contact force is given in the form [20]

$$F_r = \frac{-h(\varepsilon)E(\varepsilon)}{1 - \sigma^2} \frac{\eta^\varepsilon}{R^2} + h(\varepsilon)G(\varepsilon)\kappa(\varepsilon) \frac{\partial^2 \eta^\varepsilon}{\partial z^2} - \rho_w h(\varepsilon) \frac{\partial^2 \eta^\varepsilon}{\partial t^2}, \quad (2.10)$$

where κ is the Timoshenko shear correction factor (see, Reisman [27]), G is the shear modulus. In the right-hand of equation (2.10), the first term is the elastic response function, the second term is related to the radial pre-stress state of the vessel and the third term is the inertia term that is proportional to the radial acceleration of the vessel wall. The fluid velocity at the deformed interface $(R + \eta^\varepsilon, z, t)$ must be equal to the Lagrangian velocity of the membrane. With only the radial displacements as non-zero we have

$$v_r^\varepsilon(R + \eta^\varepsilon, z, t) = \frac{\partial \eta^\varepsilon}{\partial t}(z, t) \text{ on } (0, L) \times \mathbb{R}_+, \quad (2.11)$$

$$v_z^\varepsilon(R + \eta^\varepsilon, z, t) = \frac{\partial S^\varepsilon}{\partial t} = 0 \text{ on } (0, L) \times \mathbb{R}_+. \quad (2.12)$$

We assume that continuity of velocity along the longitudinal displacement from the reference state S^ε is zero, and thus $\frac{\partial S^\varepsilon}{\partial t} = 0$. The radial contact force is equal to the radial displacement component of the stress exerted by the fluid on the membrane. Thus,

$$-F_r = [(p^\varepsilon - p_{ref}) \mathbf{I} - 2\mu D(v^\varepsilon)] \mathbf{n} \cdot \mathbf{e}_r = (p^\varepsilon - p_{ref}) \mathbf{I} - 2\mu \frac{\partial v_r^\varepsilon}{\partial r}, \quad (2.13)$$

where p_{ref} is the pressure at the reference domain, \mathbf{n} is the unit normal at the deformed structure, $D(v^\varepsilon)$ is the deviatoric part of an axially symmetric vector/tensor-valued function $v^\varepsilon = v_r^\varepsilon \mathbf{e}_r + v_z^\varepsilon \mathbf{e}_z$ given by

$$D(v^\varepsilon) = \begin{pmatrix} \frac{\partial v_r^\varepsilon}{\partial r} & 0 & \frac{1}{2} \left(\frac{\partial v_r^\varepsilon}{\partial z} + \frac{\partial v_z^\varepsilon}{\partial r} \right) \\ 0 & \frac{v_r^\varepsilon}{r} & 0 \\ \frac{1}{2} \left(\frac{\partial v_r^\varepsilon}{\partial z} + \frac{\partial v_z^\varepsilon}{\partial r} \right) & 0 & \frac{\partial v_z^\varepsilon}{\partial z} \end{pmatrix}$$

When the cylinder is filled with fluid, the initial condition under reference pressure p_{ref} , is such that

$$\eta^\varepsilon = S^\varepsilon = \frac{\partial \eta^\varepsilon}{\partial t} = \frac{\partial S^\varepsilon}{\partial t} = 0, \text{ and } v^\varepsilon = 0 \text{ on } \Sigma_\varepsilon \times \{0\}. \quad (2.14)$$

The temporal dynamic pressure prescribed at both ends of the cylinder drives flow. The following boundary (inlet/outlet) conditions apply [20]:

$$v_r^\varepsilon = 0, \quad p^\varepsilon + \frac{\rho(v_z^\varepsilon)^2}{2} = p_0(t) + p_{ref} \quad \text{on } (\partial\Omega_\varepsilon \cap \{z=0\}) \times \mathbb{R}_+, \quad (2.15)$$

$$v_r^\varepsilon = 0, \quad p^\varepsilon + \frac{\rho(v_z^\varepsilon)^2}{2} = p_L(t) + p_{ref} \quad \text{on } (\partial\Omega_\varepsilon \cap \{z=L\}) \times \mathbb{R}_+, \quad (2.16)$$

$$\frac{\partial S^\varepsilon}{\partial z} = \eta^\varepsilon = 0 \text{ for } z=0, \quad S^\varepsilon = \eta^\varepsilon = 0 \text{ for } z=L \text{ and } \forall t \in \mathbb{R}_+, \quad (2.17)$$

where pressure drop is assumed to be $A(t) = p_L(t) - p_0(t) \in C_0^\infty(0, +\infty)$.

The fluid-structure interaction problem is encapsulated in the following:

$$\left. \begin{array}{l} \text{Equations (2.3), (2.4), (2.5)} \quad \text{in } \Omega_\varepsilon, \\ \text{Equations (2.11), (2.12)} \quad \text{on } \Sigma_\varepsilon \times \mathbb{R}_+, \\ \text{Equation (2.13)} \quad \text{on } \Sigma_\varepsilon \times \mathbb{R}_+, \\ \text{Equations (2.15), (2.16)} \quad \text{on } (\partial\Omega_\varepsilon \cap \{z=0, L\}) \times \mathbb{R}_+, \\ \text{Equation (2.17)} \quad \text{for } z=0, L \forall t \in \mathbb{R}_+. \end{array} \right\} \quad (2.18)$$

The method of solution of equation (2.18) was overcome by asymptotic expansion about the parameter ε embedded in a careful solution procedure [28]. The pressure content of the problem, which relates to the shear modulus, was rigorously derived as

$$p(z, t) = \frac{E_0}{R^2(1-\sigma^2)} \eta - G_0 \frac{\partial^2 \eta}{\partial z^2} + O(\varepsilon^2). \quad (2.19)$$

Shear modulus is crucial in the determining wave speed; it also describes the response to the shear wave. The assumption, here, of negligible shear modulus and material incompressibility implies that $G_0 = 0$ and $\sigma = 0.5$ respectively. Hence,

$$p = \frac{4E_0}{3R^2} \eta = \frac{4E_0}{3R} \left(1 - \sqrt{\frac{A(0)}{A}} + O\left(\frac{\eta}{R}\right) \right), \quad (2.20)$$

where $A(0)$ is the cross-sectional area at z corresponding to zero pressure.

3.0 Equation of pressure wave

Equation (2.20) is the expression of pressure that is required to drive waves in an arterial segment. Such waves are presented in this section. As usual, we assume an elastic artery and incompressible fluid flow. The governing Navier-Stokes equations are [29]

$$A_T + Q_Z = 0, \quad (3.1)$$

$$Q_T + \left(\frac{Q^2}{A} \right) + \frac{A}{\rho} P_Z + \nu \frac{Q}{A} = 0, \quad (3.2)$$

where $A(T, Z) = \pi R^2(T, Z)$ is the cross-sectional area of the vessel, $Q(T, Z)$ is the blood flow volume, $P(T, Z)$ is the blood pressure, with T and Z being temporal and spatial dimensions respectively, ρ is the blood density, ν is a coefficient of the blood viscosity and the quantities A_T , P_Z and Q_Z are gradients associated with their respective subscripts. Yomosa [30] described the motion of the arterial wall by

$$\frac{\rho_w h_o R_o}{A_o} A_{TT} = (P - P_e) - \frac{h_o}{R_o} \sigma_s, \quad (3.3)$$

where ρ_w is the wall density, P_e is the pressure outside the tube, h_o is the mean thickness of the wall, R_o is the mean radius and σ_s is the extending stress in the tangential direction. Since the wall is elastic, the local compliance of the vessel is such that

$$\sigma_s = \frac{E(A - A_o)}{2A_o}, \quad (3.4)$$

where A_o is the arterial cross-sectional area at rest, and E is the coefficient of elasticity. By the theory of thin walled cylinder the arterial local compliance is given by [9]

$$C_o = \frac{2\pi(1 - \sigma^2)r_o^3}{hE} \quad (3.5)$$

where the subscript o indicates reference states at initial pressure $p = p_o$.

The non-dimensional representations of equations (3.1)-(3.4) are

$$a_t + q_z = 0, \quad (3.6)$$

$$q_t + \left(\frac{q^2}{1+a} \right)_z + (1+a)p_z = -\wp \frac{q}{1+a}, \quad (3.7)$$

$$\alpha \varepsilon^5 a_{tt} + a = p, \quad (3.8)$$

where $(\wp = \frac{v\lambda}{A_o c_o})$, λ is the typical wavelength of the wave propagated in the tube and c_o the Moens-Korteweg sound wave velocity in the fluid-filled tube.

When equations (3.6), (3.7) and (3.8) are expressed as an asymptotic expansion at the second order of ε , and noting that the coefficients of series must vanish, we have the following KdV equation:

$$P'_Z + d_o P'_T + d_1 P' P'_T + d_2 P'_{TTT} = 0, \quad (3.9)$$

$$\text{with } d_o = \frac{1}{c_o}, d_1 = \frac{-3}{2} \frac{1}{A_o c_o^2}, d_2 = \frac{-\rho_w h_o R_o}{2 \rho c_o^3}.$$

Suppose $P' = P_s$ is a soliton solution, then equation (3.8) can be in the form

$$\frac{\partial P_s}{\partial z} + (d_o + d_1 P_s) \frac{\partial P_s}{\partial t} + d_2 \frac{\partial^3 P_s}{\partial t^3} = 0. \quad (3.10)$$

With the following transformation: $\Gamma = t - d_o z$, $\vartheta = d_2 z$ and $u = \frac{d_1}{6d_2} P_s$, we get

$$u_{\vartheta} + 6uu_{\Gamma} + u_{\Gamma\Gamma} = 0 \quad (3.11)$$

The two soliton solution (2SS) of equation (3.11) is [31]

$$u(z,t) = \left(\frac{k_1^2 - k_2^2}{2} \right) \left(\frac{k_1^2 \cos^2 \text{ech}^2 \frac{\check{\gamma}_1}{2} + k_2^2 \sec^2 h^2 \frac{\check{\gamma}_2}{2}}{\left(k_1 \coth \frac{\check{\gamma}_1}{2} - k_2 \tanh \frac{\check{\gamma}_2}{2} \right)^2} \right), \quad (3.12)$$

where $\check{\gamma}_i = \alpha_i z - \alpha_i^3 t + \beta_i$; β_i being an arbitrary constant ($i = 1, 2$), k is the wave number. From the works of Womersely [32], the wave number k for thin walled vessels with the compliance as defined in equation (3.5) is

$$k = \frac{\omega}{c_o} \frac{1}{\sqrt{1 + if_v}} \quad (3.13)$$

where ω is the angular frequency, $f_v = E_i/E_r$ is the viscous fraction, where E_i and E_r are the imaginary and the real part of the complex modulus $E^*(\omega)$, respectively. For small value of f_v the quantity k can be approximated by

$$k \approx \frac{\omega}{c_o} \left(1 + \frac{1}{2} if_v \right). \quad (3.14)$$

The solution of the wave problem given by equation (3.12) describes the ‘peaking’ and ‘steepening’ phenomena. Such phenomena are typical of solitary waves.

4.0 Physiological Analysis

Pulse waves propagated through the arterial bed are an estimator of the systolic phase of blood pressure. Waves are reflected at various arterial sites which may include points of bifurcation, areas of changes in arterial geometry, peripheral and terminal regions. Multiple reflected waves may integrate as a single wave. The sum of the reflected waves and the forward (ejected) wave forms the final profile of the pressure pulse wave.

The understanding of the waveform at various time intervals occupied by the wave would provide a clue to physiological conditions. To this effect, the systolic time interval (STI) and the left ventricular ejection time (LVET) are of interest. The graphs of Figure 4.3 represent two soliton solution at various times, at a constant distance ($z = 40$ units). Each LV ejection has an accompanying wave. In each of the graphs A-F of Fig 4.1, the aortic pre-ejection period is the interval of time before the appearance of the taller (tandem) wave. Graph A shows the emergence of a tandem wave (during LV afterload). In Graph B the tandem wave approaches the shorter (precursor) wave. In the real sense, another wave in tandem with the tandem wave had evolved, which moves behind the tandem wave and, expectedly, would overtake it at some time. We had said that two solitons are enough to describe the wave phenomena. Graph D shows the enveloping of the precursor wave by the tandem wave. LVET is the interval from the commencement to the termination of aortic flow. It is associated

with LV preload and afterload [33]. In the wave analysis, it is the time it takes the LV to create a tandem wave that would eventually interact with the precursor wave. This time interval may be obtained by detecting the position of the precursor wave just before the onset of the tandem wave. Within LV preload interval, the tandem wave would have been on the verge of interaction with its precursor. This is seen in Graph B. Physiological LVET must be maintained. Shortened ejection duration is a cardiac liability [33]. In event of shortened LVET, there is the tendency of early wave reflection to have a dominant effect on flow than on pressure [34, 35]. Graph F shows that a physiological tandem wave must displace a precursor wave unscathed before its eventual evanescence at infinity.

Shortened LVET would generate rather more waves with stunted amplitudes in a relatively short time. On account of this condition, the time taken by the tandem wave to approach and interact with the precursor wave will be too short, thus, resulting in profusion of tandem waves. There must be a balance between pre-ejection period and LVET. Shortened LVET, together with prolonged pre-ejection period, amounts to LV dysfunction. In such circumstances a dicrotic pulse wave that marks an incipient pathology may be observed. The implication is that a subject is likely to present with issues related to systolic heart failure (SHF) [36, 37].

On the other hand, the prolonging of LVET, due to slowed calcium re-uptake [38] will cause the early reflected waves to be dominant on pressure than on flow. When this happens, the precursor-tandem wave interaction would be such that the precursor wave is enveloped for a relatively long time. This may account for the perception that there is no second wave in subjects living with diabetes or atherosclerosis [39]. The dominance of early reflected waves on pressure than on flow may predispose a subject to LV hypertrophy [37],

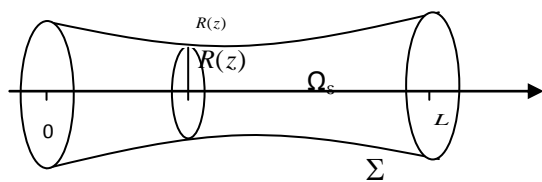


Fig. 2.1. Reference domain

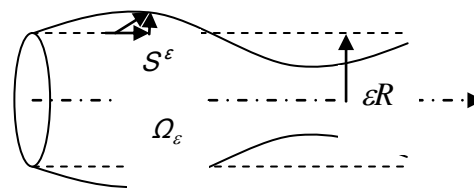


Fig. 2.2. Wall displacement

Type equation here.

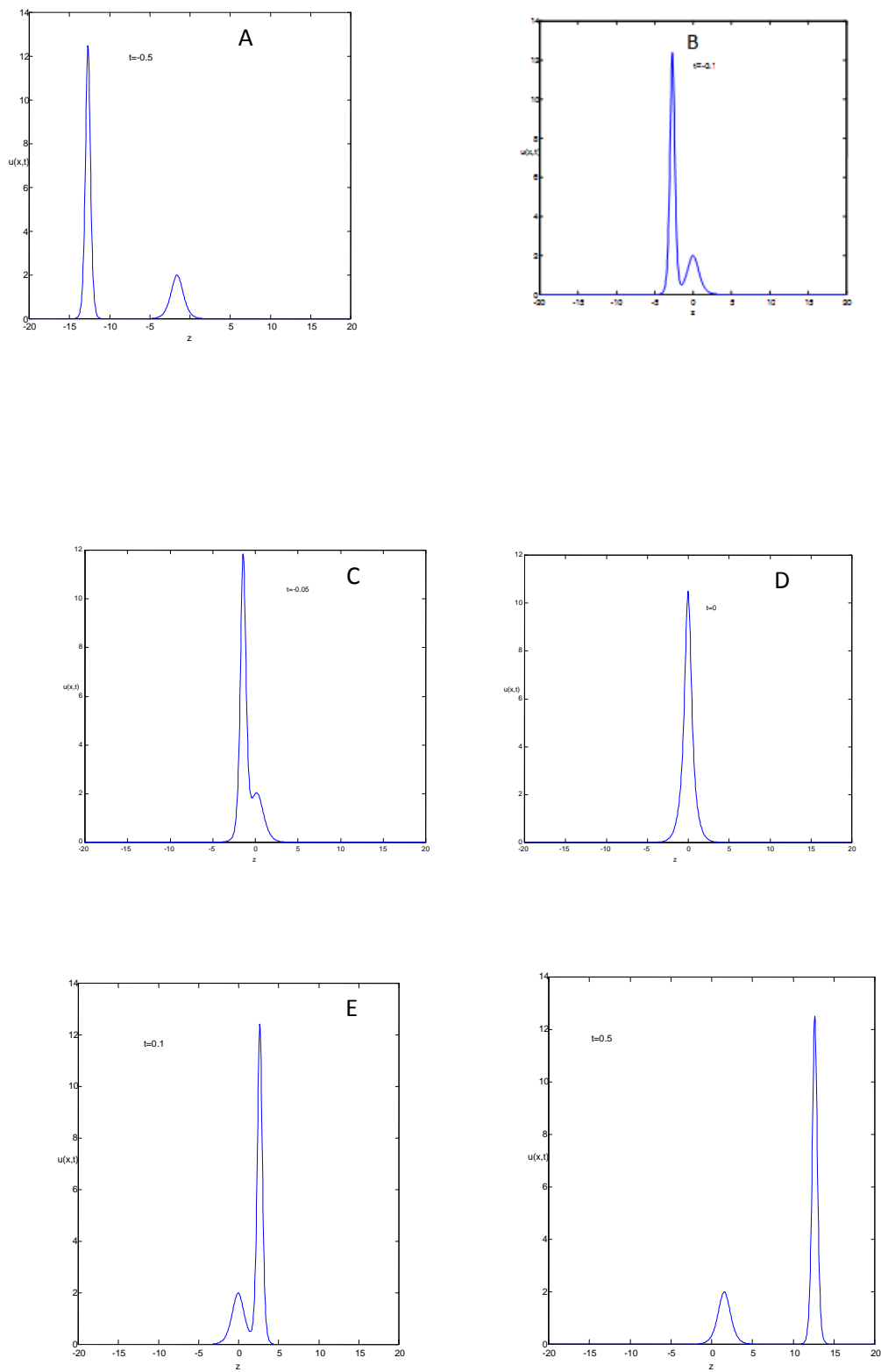


Fig. 4.1. 2-Soliton Solutions for various time intervals, at different phases of its propagation.

5.0 Summary and Conclusion

Arterial pulse wave is a phenomenon of choice in describing physiological states. This work considered pulse waveforms with respect to LVET, in a bid to determine its influence on cardiovascular events. Model equations governing FSI and pulse wave were derived. The analysis of two soliton solution obtained from the resulting wave problem gave an insight into role of LVET in physiological mediation. LVET explains the morphology of pulse wave in time domain. It is definitive in the prognostic assessment of cardio-vascular patho-physiology. In subjects living with diabetes or atherosclerosis, it was conceived that the second wave was non-existent. On the contrary, the present analysis indicates that in event of LVET prolongation the said second wave is 'unduly' encapsulated on interaction. We compare shortened LVET to prolonged LVET for clinical purposes: the former has the propensity to induce SHF, and the later is closely associated with LV hypertrophy. Therefore, both shortened and prolonged LVET are independent markers of cardio-vascular events.

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