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COMPUTATIONAL STUDY OF LEFT VENTRICULAR DYSFUNCTION

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Abstract— ***Left ventricular dysfunction (LVD) is a prevalent cardiovascular condition that significantly impacts heart function. This computational study employs a comprehensive cardiovascular model to investigate how various physiological factors influence LV performance. The model incorporates realistic representations of LV mechanics, including detailed descriptions of muscular properties and contractile behavior. Additionally, the model integrates a comprehensive representation of systemic circulation, encompassing blood vessel elasticity and resistance. Following validation against clinical data, simulations are conducted to explore the influence of different physiological parameters on LV function. These parameters could include preload, afterload and contractility. The simulations generate detailed pressure and volume waveforms within the left ventricle for further analysis. By analyzing these waveforms, the study aims to identify key relationships between the chosen physiological parameters and specific features of LV mechanics, such as stroke volume and ejection fraction. This computational approach not only deepens our understanding of the mechanisms underlying LVD but also paves the way for developing patient-specific models in the future. Such models could be instrumental in guiding personalized treatment strategies for cardiovascular patients with LVD.***

Keywords: cardiovascular model, parameters, waveforms.

I. INTRODUCTION

Cardiovascular diseases remain a significant global health concern. A crucial area of investigation lies in understanding the intricate interplay between respiratory patterns and cardiac function. This study focuses on the complex relationship between left ventricular (LV) dysfunction, a hallmark of heart failure, and various respiratory patterns.

Left ventricular dysfunction compromises the heart's ability to pump blood efficiently, leading to hampered systemic circulation. In recent years, computational modeling has emerged as a powerful tool for investigating the complex dynamics of cardiac function. This study leverages computational techniques to delve into the intricate connection between LV dysfunction and different respiratory patterns.

By simulating and analyzing the interplay between the respiratory and cardiovascular systems, we aim to gain insights into how various respiratory patterns, such as variations in breathing rate, tidal volume, and inspiratory/expiratory ratio, influence LV function and hemodynamics. We will explore how changes in these respiratory parameters affect cardiac performance, including metrics like stroke volume and cardiac output.

Beyond waveform generation, our study incorporates rigorous statistical analysis to extract meaningful insights from the simulated data. By applying statistical methodologies, we intend to quantify and interpret the observed changes in LV function under the influence of different respiratory patterns. The overarching goal of this computational journey is to enhance our understanding of the intricate relationship between respiration and LV dysfunction within the broader context of cardiovascular function.

The outcomes of this study hold the promise of not only contributing to the scientific understanding of heart failure mechanisms but also providing a foundation for future developments in personalized therapeutic interventions targeting both respiratory and cardiac aspects. By bridging computational modeling, MATLAB simulations, and statistical analyses, we aspire to pave the way for novel insights into the complex dynamics of heart failure in the context of various respiratory patterns.

II. LITERATURE REVIEW

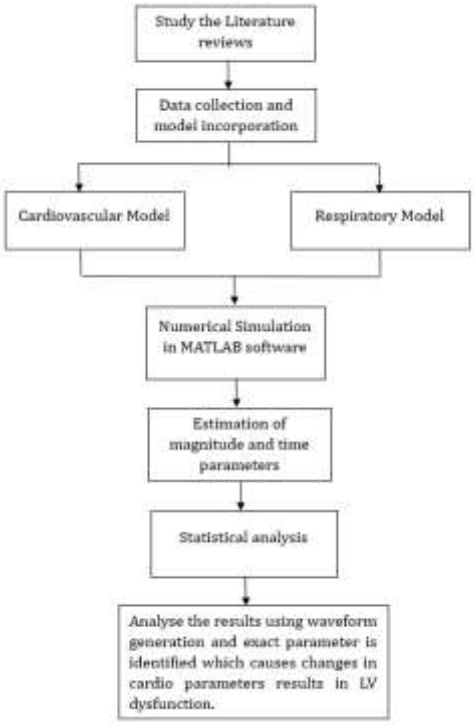
In 2011, Hemalatha K. and Manivannan M. carried out a study named "Investigation of Detailed Lumped Parameter Model for Cardiopulmonary Hemodynamic Interaction." The anatomical portrayal of the baroreflex, cardiovascular (CV) system, and respiratory pump system (RPS) was thoroughly delineated and finely detailed in this work. Breathing-induced changes in the cardiovascular system (CVS) were successfully modelled by simulating the influence of intrapleural pressure, or Ppl. The impact of this respiratory pump system (RPS) on aortic pressure was evaluated using both the Valsalva manoeuvre and relaxed breathing scenarios.. Its usefulness for studying hemodynamic interactions in both CVS and RPS under normal and diseased conditions as well as their reciprocal influences in the aforementioned scenarios enhanced when the results closely matched the data from the literature. It is important to note that the model has a two-step parameter identification approach, which is helpful for studying CP illnesses such as cheyne stoke respiration (CSR) and sleep apnea. The model does, however, have many drawbacks, notable among them being the omission of any discussion of gas transfer at the tissue level.

In 2011 Hemalatha K and Manivannan M conducted research on the "cardiopulmonary model for investigating hemodynamic interaction in the Muller manoeuvre." The objective of this research was to combine the benefits of several models for the three systems under consideration that have been independently reported in the literature. Nevertheless, a thorough comprehension of these systems and how they interact with one another was still lacking. In order to address this, the dynamics of the cardiopulmonary (CP) system were qualitatively evaluated using a controlled breathing simulation. The resulting model, in agreement with previously published data, showed the ability to forecast important physiological events within individual system dynamics as well as their interaction when breathing calmly. Interestingly, silent breathing was associated with a significant drop in pulse pressure, which in turn activated the Muller manoeuvre (MM) baroreflex. This implies that the arterial baroreflex is more responsive to pulsatile pressure fluctuations than mean pressure variations. Despite certain drawbacks, such as the absence of the arterial pulse wave propagation phenomena, the model is an invaluable resource for investigating complex and abrupt physiological interactions in the CP system, especially under a range of normal and aberrant settings. As a result, the model shows a significant drop in pulse pressure, which activates the baroreflex in the MM, even though the mean arterial pressure barely changes.

The paper titled "Model studies of the effects of the thoracic pressure on the circulation" by R. Beyar and Y. Goldstein, published in the Annals of Biomedical Engineering in 1987, focuses on investigating the impact of thoracic pressure on the circulatory system through model studies. The authors delve into the complex interactions between thoracic pressure changes and the cardiovascular system, aiming to enhance the understanding of the mechanisms involved. The work likely involves the development and utilization of mathematical models to simulate and analyze how variations in thoracic pressure influence cardiac function, blood flow dynamics, and related physiological parameters. Through their studies, the authors contribute valuable insights into the intricate relationship between thoracic pressure and cardiovascular responses, providing a foundation for comprehending the physiological implications and potential clinical relevance of such interactions.

III. METHODOLOGY

The methodology proposed for a computational model of left ventricular (LV) dysfunction with respect to Cheyne-Stokes respiration (CSR) using pleural pressure, incorporating the electrical equivalence of the cardiovascular system (CVS) model and pleural pressure, and performing numerical simulations in MATLAB software, followed by statistical analysis, may involve several steps:



3.1 Flow chart of the proposed work

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The computational model of hybrid cardiopulmonary that is based on physiological principles is explained in this chapter. Among many modelling strategies, the present model combines the benefits of both lumped and distributed parameter modelling. Lumped parameter modelling has many advantages, especially when it comes to modelling the overall dynamics of the cardiopulmonary system's constituent parts—that is, excluding the arterial tree system's hemodynamics. However, the distributed modelling method performs exceptionally well in explaining the phenomenon of wave propagation inside the artery tree system.

With that incorporate the base model with pleural pressure. Integrate a module to simulate pleural pressure dynamics, capturing the effects of respiratory mechanics on intrathoracic pressure. Pleural pressure refers to the pressure within the pleural cavity, the space between the lungs and the chest wall. It plays a crucial role in respiratory mechanics by influencing lung expansion and air movement during breathing. Pleural pressure can be positive, negative, or zero relative to atmospheric pressure, depending on the phase of the respiratory cycle and the conditions within the thoracic cavity.

As a result, the current model makes use of the advantages of the two modelling strategies that were previously stated.

The electrical analogy was chosen for this study among other analogies because it offers a consistent framework for describing both pneumatic (respiratory) and hydraulic (cardiovascular) systems. Furthermore, it provides an easy way to incorporate different mechanisms into cardiopulmonary dynamics and link model parameters to their physical interpretations.

A model includes: - A closed-loop lumped parameter model that consists of:

1. Cardiovascular model.
2. Respiratory model, which includes gas exchange and lung mechanics.
3. An open-loop model with distributed parameters that includes the left upper limb's.
4. Systemic arterial tree, which starts at the aorta and ends at the radial artery.

Essentially, the formulation of this model builds upon the most effective features of several modelling techniques and analogies to produce a comprehensive framework for comprehending and modelling the complex dynamics of the cardiopulmonary system.

A. CARDIOVASCULAR MODEL

The systemic and pulmonary circulation, the venous system, the cardiac valves, the direct ventricular interaction via the septum, the mechanics of the atrium and ventricles, and the impact of the pericardium on heart pumping efficiency are all included in the cardiovascular model's sub-models. The circulation model utilised in this study is one of the many models that have been reported in the literature; it is a modification of Sun et al (1997). In their model, they included a comprehensive representation of the heart and valves. The aforementioned model did not take into account the physiology of the respiratory system, intrathoracic pressure, or the impact of breathing on CVS in order to accurately replicate CP physiology. A simplified representation of the baroreflex, which solely controls cardiac duration, was also provided.

1. Heart rate, myocardial contractility, and peripheral resistance are all regulated by the baroreflex model.
2. Intrathoracic pressure is considered to serve as a respiratory-dependent pressure source, changing instantly in accordance with the stages of respiration (inspiration and expiration).
3. The CVS model is combined with a full respiratory model that includes gas exchange and lung mechanics to enable simultaneous simulation of respiratory dynamics.
4. A dispersed arterial tree model illustrates the anatomy of the systemic arterial system, which extends from the aorta to the radial artery.
5. The following characteristics of the existing lumped parameter cardiovascular model:
6. The heart is composed of four chambers and four valves.
7. The volume coupling of the pericardium.
8. The systemic and lung circulation; the time-varying intrapleural pressure; and the interventricular septum pressure coupling.

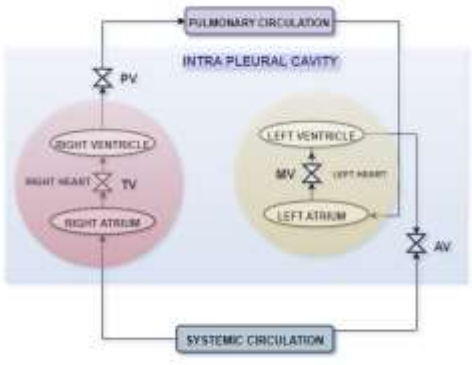

Fig. 3.2 Schematic diagram of cardiovascular system model (TV- Tricuspid valve, PV- Pulmonary valve, MV- Mitral calve and AV-aortic valve)

B. HEART MODEL

A lot of literature has been made about the mathematical representation of the heart. It has been shown as a simple signal generator (Grodins et al., 1967; Braunwald et al., 1967) and in more complex representations with changing elastance (Suga, 1969; Suga et al., 1973). Because of its direct link with pressure and volume (E=dP/dV), the time-varying elastance model of the heart has been used extensively in research endeavours (Chambell et al., 1982; Chung et al., 1997; Abdolrazaghi et al., 2010). As such, it provides a somewhat simple way of integrating experimental results concerning the pressure-volume relationship of heart chambers. As a result, the thesis's heart model embraces the idea of time-varying elastance.

Two parameters characterise the wall properties of each of the four cardiac chambers in our heart model: viscoelastic resistance and elastance. Each heart chamber's elastic properties are expressed in terms of the pressure-volume (P-V) relationship. An instantaneous elastance function, which offers a temporal representation of cardiac chamber contraction and relaxation, illustrates the P-V connection of the heart's peripheral walls (Heldt et al., 2002).

For the sake of this investigation, the left and right ventricles are divided by the flexible intraventricular septum. In response, it deflects in the direction of the lower-pressure chamber. There is no deflection and the septum stays flat when the pressures in the left and right ventricles (PLV and PRV), respectively are equal. The septum travels in the direction of the left ventricle (LV) when PLV is less than PRV and towards the right ventricle (RV) when PLV is more than PRV. The phenomenon of direct ventricular connection, which happens during each cardiac cycle, is characterised by the inclusion of septum movement (Santamore and Burkhoff, 1991; Olansen et al., 2000; Luo et al., 2007).

As a result, the direct pressure interaction between the RV and LV via the interventricular septum is represented by this model. Consequently, the RV elastic compartment (ev), the LV elastic compartment (err), and the septum elastic compartment (ES) make up the three elastic compartments in the model. Consequently, the following is the formulation of LV's time-varying functional elastance (eLV) with septum coupling.

$$eLVf= Es.eLV(eLV+Es)$$

The following equation expresses how the pressure interaction from the RV across the septum determines the LV pressure.

$$PLV= eLVf.VLV+eLV(eLV+Es)PRV$$

where the pressure in the left ventricle is represented by PLV and the pressure in the right ventricle by PRV. Similarly, by replacing the left ventricular parameters with their corresponding right ventricular values, the right ventricular functional elastance (eRvf) and pressure (PRV) are calculated. It's crucial to remember that the two atrial chambers are modelled separately and aren't connected by the septal wall. The reason for this is that there is very little direct connectivity between the two atria due to the relatively thick and less flexible septum between them. The amplitude and baseline components are included in a cosine function that describes the ventricular pericardium wall's time-varying elastance.

The left ventricle's time-dependent wall elastance is represented by the equation eLV[mmHg.ml-1], where ELVa stands for the amplitude factor, ELVb for the base factor, tee for the end ejection time, and tcr for the cardiac period. The Frank-Starling law states that variations in volume have an impact on the ventricle's elastance, which varies with time (Brutsaert and Paulus, 1977). This volume-dependent relationship results from the finding that contraction velocity and force both decrease with increasing preload. A scaling factor, F, is used to approximate this contractility-preload relationship. Left ventricular stiffness rises and left ventricular contractility decreases with increasing preload. This conceptual idea is included in the model by, as will be shown below, dividing the base factors (ELVb and ERVb) by F and multiplying the amplitude factors (ELVa and ERVa) of the left and right ventricular elastance by F.

$$FL=1-VLV \_ EDVmax$$

The left ventricular end-diastolic volume is denoted by VLV _ ED in the equation above, and the maximum reference volume of the cardiac chambers, or Vmax, stays fixed at 900 ml for the duration of the investigation. This decision is made to guarantee that the left ventricle functions as a suitable depiction of the Frank-Starling relationship.

$$tac= tcr- 0.5tee-0.02$$

$$tar= tcr-0.04$$

The equation below defines the left atrium's time-dependent elastance. The left atrium's amplitude factor is denoted by the symbol ELAa in this equation, its base factor is ELAb, its contraction start time is indicated by tac, and its relaxation start time is indicated by tar. Furthermore, atrial contraction and relaxation periods are denoted by the terms teer and teec, respectively. Additionally, tee, the ventricular end-ejection time, is a function of tac and tar values.

Together with the time-dependent elastance, each heart chamber has a viscoelastance

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factor, represented by the letter "S," which takes into consideration the internal resistance in each chamber (Sun et al., 1995). The pressure inside each heart chamber affects this viscoelastance component, denoted as S_i in the equation below:

$$Si=0.0005Pi$$

In this case, the heart chamber (RA, RV, LA, or LV) for which the viscoelastic resistance is being calculated and it is indicated by the subscript i. The electrical equivalent of the right atrium (RA), for instance, together with the pericardium and intrapleural pressure, are shown in the following figure.

Typical electrical equivalent model representation of RA wall properties (Pvc Venacava Pressure, SRA- Right atrium viscoelastic resistance, eRA- Right atrium elastance, Ppc- Pericardium pressure and Ppl- Pleural pressure)

C. PARAMETER TUNING

We calculate resistance and inertance values by taking into consideration the anatomical characteristics of particular portions and applying the concepts of fluid dynamics. By using Doppler echocardiographic data from a study by Sun et al. from 1997, the valve parameters (R, L, and B) may be found. The flow patterns of each heart valve are then exactly replicated by carefully adjusting these parameters. Furthermore, the pulmonary circulatory elastances are iteratively adjusted until they give an average volume stored within the elastance that is consistent with predicted values and offer a reasonable depiction of hemodynamic waveforms.

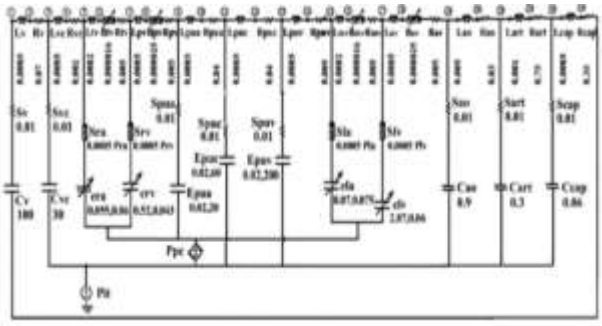

Fig 3.3. Electrical Equivalent model of CVS by Hemalatha et al

Based on the volume distribution of the heart, the initial elastance values (Eb) for the contracting chambers are determined. The degree of damping seen in the pressure waveforms determines which viscoelastic terms (S) are used. In our model, the pressure-dependent internal chamber resistance is given a low value (0.0005) as the proportionality constant in order to accommodate the significant flow-dependent components at the outflow tract of a contracting chamber.

IV. RESULTS AND DISCUSSION

Since the model meets the fundamental needs of the current investigation, it is regarded as the base model, as stated in the literature by K Hemalatha et al. In physiology, the heart's electrical activity initiates cardiac rhythm; this is not taken into account in this model. As a result, the model incorporates rhythmic activity by interpreting the heart chambers' elastance as a variable that varies with cardiac frequency.

The base model is used to get the model equations, and the Runge-Kutta numerical solution method is used to derive the solutions. The beginning circumstances are obtained from Hemalatha et al.,

$$F_L = 1-\left(V_{LV\_ED}/V_{\max}\right)$$

and MATLAB coding has been built to solve the relevant equations. The cardiac cycle has been set as 0.855sec to represent normal heart beat (70 Beats/Min). The simulations are generated with the resolution of 0.001secs.

The following are the elastance formulas implemented in this model:

$$e_{LV}=\begin{cases} 0.5F_L E_{LVa}\left(1-\cos\left(\frac{\pi t}{t_{ee}}\right)+\frac{E_{LVb}}{F_L}\right) & 0\le t\le t_{ee} \\ 0.5F_L E_{LVa}\left(1+\cos\left(\frac{\pi(t-t_{ee})}{0.5t_{er}}\right)+\frac{E_{LVb}}{F_L}\right) & t_{ee}<t\le 1.5t_{ee} \\ \frac{E_{LVa}}{F_L} & 1.5t_{ee}<t\le t_{er} \end{cases}$$

Where tee is the end ejection time, tcr is the cardiac period, ELVa is the amplitude factor, and eLVb is the base factor. eLV [mmHg.ml–1] indicates LV time dependent wall elastance. The time-varying elastance of the ventricle is affected by volume, according to Frank-Starling law. The volume dependence is created on the basis that contraction force and velocity decrease with increased preload. A first-order estimation of the contractility-preload relation is implemented by computing a scaling factor F. As preload increases, the LV contractility decreases and the LV stiffness increases. In this model, the previously mentioned idea is achieved by multiplying the left and right ventricle elastance amplitude factors (ELV a, ERV a) and dividing the base factors (ELV b and ERV b) by F.

Throughout the study, the maximal reference volume of the heart chambers, or Vmax, is chosen to be 900 ml in order to provide a valid interpretation of the Frank-Starling relation on LV functioning. In the equation, VLV_ED represents

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the left ventricular end diastolic volume. The time-dependent left atrial elastance is contained in the equation. In equation, the letters ELA a, ELA b, tac, and tar represent the LA amplitude factor, LA base factor, and atrial start of contraction and relaxation, respectively. The names "teer" and "teec" stand for atrial contraction and relaxation periods, respectively. where the ventricular end-ejection time (tee) determines the maintenance of tac and tar.

$$t_{ac} - t_{cr} - 0.5t_{ee} - 0.02$$
$$t_{ar} = t_{cr} \quad 0.04$$

The base model is used to get the model equations, and the Runge-Kutta numerical solution method is used to derive the solutions. The beginning circumstances are obtained from Hemalatha et al., and MATLAB coding has been built to solve the relevant equations. The cardiac cycle has been set as 0.855sec to represent normal heart beat (70 Beats/Min). The simulations are generated with the resolution of 0.001secs.

The power source for the model's electrically analogous cardiovascular system is changing elastance. Figures 4.1 and 4.2 mimic the right and left atrium and ventricular elastances and display them. Elastance, in the context of the heart chambers, refers to the chamber's ability to stretch and contract in response to changes in pressure. It is a key parameter in cardiovascular physiology and is often used to describe the contractile properties of the myocardium. Elastance is the reciprocal of compliance, representing the stiffness or rigidity of the heart chamber walls. During the cardiac cycle, the elastance of a heart chamber, particularly the ventricles, dynamically changes to efficiently pump blood throughout the circulatory system. In systole, elastance increases as the myocardium contracts, generating the necessary pressure to eject blood into the arteries. Conversely, during diastole, elastance decreases, allowing the heart chamber to relax and fill with blood. Understanding and quantifying elastance are crucial for assessing cardiac function and diagnosing conditions such as heart failure, where alterations in elastance can impact the heart's ability to effectively pump blood. The proposed model simulates all expected changes in cardiac chamber elastances during diastole and systole. The generated elastance matches well with the experimental elastance plot reported in Guyton 2016.

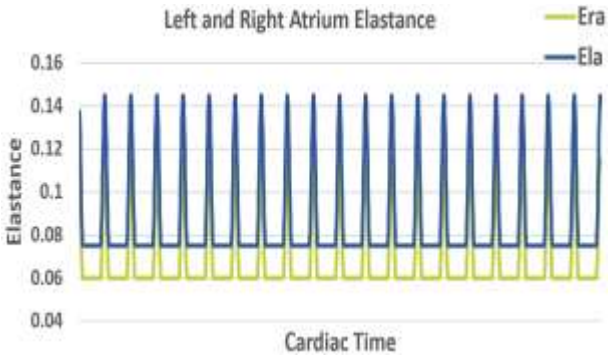

Figure 4.1 Right Heart Elastance

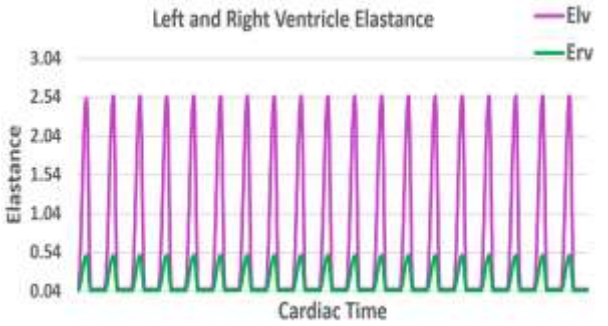

Figure 4.2 Left Heart Elastance

The elastance of the right and left cardiac chambers represents their distinct contractile properties and functions within the cardiovascular system. The right cardiac chamber, specifically the right ventricle, typically exhibits lower elastance compared to the left cardiac chamber, which includes the left ventricle. This difference is attributed to the lower pressure requirements for pumping blood to the pulmonary circulation compared to the systemic circulation. The right ventricle is adapted to handle lower resistance and pressures associated with the pulmonary circuit, allowing for efficient blood flow to the lungs. In contrast, the left ventricle experiences higher pressures and must generate greater force to propel oxygenated blood into the systemic circulation. This dissimilarity in elastance reflects the unique hemodynamic demands on each side of the heart, emphasizing the specialized roles of the right and left cardiac chambers in maintaining circulatory equilibrium.

Figure 4.3 shows simulation of the left ventricular pressure and volume plot is necessary for examining the impact of arterial stiffness on left ventricle function. The simulated PV loop displays importantphysiological occurrences as filling, isovolumetric relaxation, Isovolumetric contraction and ejection are reflected in the simulation which is shown in Figure 4.4

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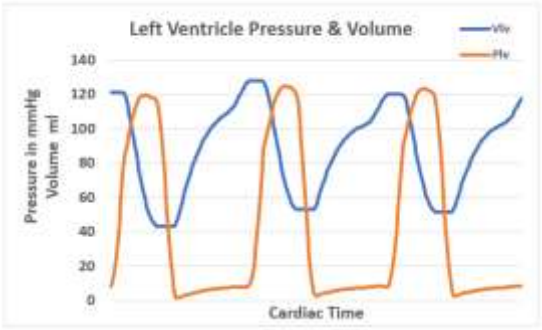

Figure 4.3 Left Ventricle Pressure and Volume

The Pressure-Volume (P-V) loop of the left ventricle (LV) is a critical tool in cardiovascular physiology and diagnostics, providing valuable insights into the dynamic changes occurring during the cardiac cycle. This loop graphically represents the relationship between the pressure within the LV and its volume throughout systole and diastole. Analyzing the P-V loop offers crucial information about the contractile function, efficiency, and overall performance of the left ventricle. Parameters derived from the P-V loop, such as end-systolic volume (ESV), end-diastolic volume (EDV), stroke volume (SV), ejection fraction (EF), and cardiac output (CO), provide quantitative measures of cardiac function. Changes in the P-V loop shape and size can indicate alterations in myocardial contractility, valvular function, and hemodynamic status.

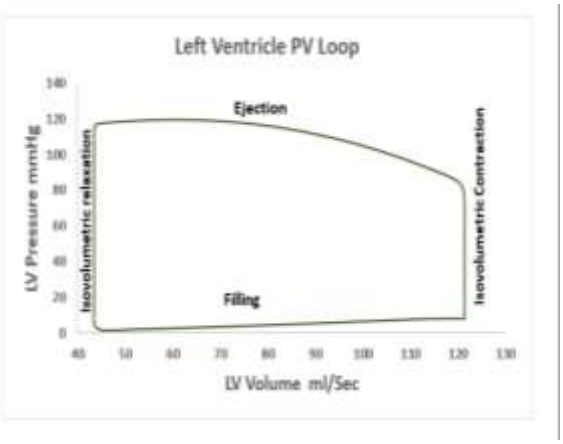

Figure 4.4 Left ventricle PV loop

The link between stroke volume and end diastolic volume is described by the Frank-Starling law of the heart, commonly referred to as Starling's law and the Frank-Starling mechanism. According to the rule, while all other variables stay constant, the heart's stroke volume rises in response to an increase in the amount of blood in the ventricles just prior to contraction (the end diastolic volume). As more blood enters the ventricle, the blood strains the heart muscle, increasing the force with which it contracts. The Frank-Starling mechanism enables synchronization of the cardiac output with the venous return, arterial blood supply, and humoral length without the need for outside control.

Preload and afterload play pivotal roles in left ventricular (LV) dysfunction, influencing the heart's ability to pump blood effectively. Preload, represented by the end-diastolic volume, is the initial stretching of the myocardium before contraction. In LV dysfunction, increased preload, often associated with conditions such as fluid overload or regurgitant heart valves, can lead to impaired ventricular filling and exacerbate heart failure. Afterload, defined by the resistance the ventricle must overcome to eject blood, is represented by arterial pressure during systole. Elevated afterload, as seen in conditions like hypertension, places additional strain on the LV, compromising its ability to pump blood efficiently and contributing to myocardial hypertrophy. The interplay between increased preload and afterload in LV dysfunction creates a detrimental cycle, further impairing cardiac performance and exacerbating heart failure symptoms. Hence, the preload and afterload variables are simulated and plotted in Figure 4.5, Figure 4.6, Figure 4.7 and Figure 4.8.

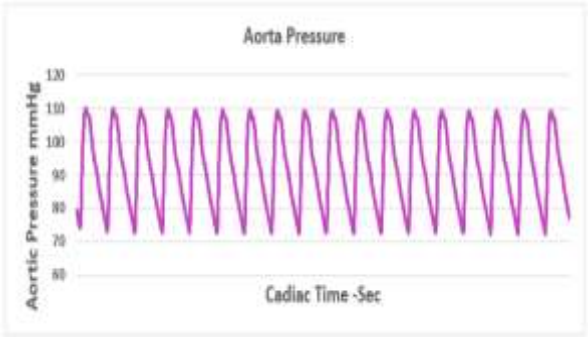

Figure 4.5 Aortic pressure

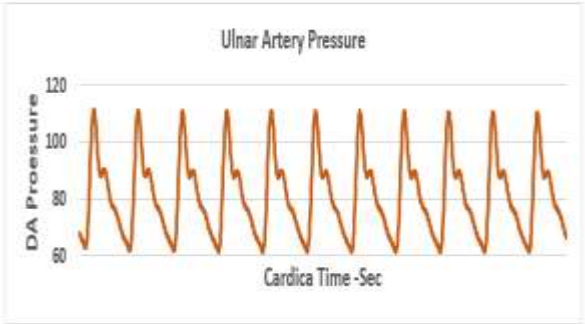

Figure 4.6 Ulnar Artery pressure

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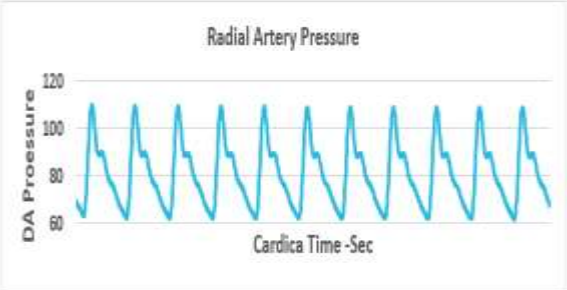

Figure 4.7 Radial Artery pressure

The simulation clearly shows the significant physiological occurrence like dichotic notch in all arterial patterns. The next significant event related to Frank-Starling mechanism is venous return. Since the model is a closed loop model, the venous return is directly simulated and it well settles within 5 to 6 cardiac cycles (shown in Figure 4.8)

Figure 4.8 Venous Return (Flow ml/sec)

V. CONCLUSION

Our computational study of left ventricular (LV) failure employed an advanced numerical model to investigate the interplay between various physiological factors. The model successfully replicated LV dysfunction and captured key aspects of the cardiovascular system. Careful validation ensured the model's reliability, and simulations in the MATLAB environment allowed dynamic examination of the system's behavior under different parameters. This approach not only enhanced our understanding of LV function but also demonstrated its potential as a powerful tool for cardiovascular research.

Post-simulation analysis revealed complex interactions between LV dysfunction and other critical parameters. By comparing simulations, we identified trends that contribute to a more comprehensive understanding of factors influencing LV function. These findings highlight the value of computational studies for unraveling the intricate relationships within the cardiovascular system and pave the way for future investigations.

While the computational approach provided valuable insights into LV dysfunction, limitations exist. Despite its complexity, the model simplifies some physiological processes, and certain values may be approximations. Additionally, the study focused on a specific aspect of respiration. Further exploration of other respiratory patterns presents opportunities for future research. Overall, this study establishes a framework for advancing our understanding of LV dysfunction, and future model modifications and extensions have the potential to significantly improve our knowledge of cardiovascular dynamics and their clinical applications.

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