# Numerical Simulation of Cardiovascular Response with VAD Support

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Abstract—This paper studies the cardiovascular response under heart failure condition supported by two types of ventricular assist devices (VADs): the positive displacement pump and the impeller pump. The cardiovascular system is modeled using the concentrated parameter method developed previously, by considering flow resistance, vessel elasticity and inertial effects of blood in individual conduit segments. The dynamic modeling of displacement and impeller pumps is represented by VAD inlet/outlet flow-rate changes. Results show that in each simulation, the diseased ventricle is effectively unloaded, and the physiological variables of arterial pressure and systemic flow are adequately maintained. The numerical model is under further development for more sophisticated studies of the interaction of the native cardiovascular system with VAD support and other cardiac prosthetic devices.

*Index Terms* — Numerical Simulation, Cardiovascular Dynamics, Heart Failure, Ventricular Assist Device, Prosthetic Device.

#### NOMENCLATURE

- C Compliance
- CQ Flow coefficient
- E, e Elastance
- L Inertia
- P Pressure
- Q Flow rate
- R Resistance
- T, t Time; heart period
- V Volume, velocity

### **Subscripts**

- 0 Initial value; offset value
- ao Aortic valve
- i Inlet
- la Left atrium
- lv Left ventricle
- mi Mitral valve
- o Outlet

р	Pulse
par	Pulmonary arterioles
pas	Pulmonary artery sinus
pat	Pulmonary artery
рср	Pulmonary capillary
ро	Pulmonary valve
pvn	Pulmonary vein
ra	Right atrium
rv	Right ventricle
sar	Systemic arterioles
sas	Systemic aortic sinus
sat	Systemic artery
scp	Systemic capillary
svn	Systemic vein
ti	Tricuspid valve
vad	Ventricular assist device
T1	Beginning instance of T wave in ECG signal

T2 Ending instance of T wave in ECG signal

# I. INTRODUCTION

Ventricular assist devices (VADs) have greatly helped in the treatment of heart failure since their first surgical implantation in the 1960s, either as a bridge to transplantation, or as a bridge to recovery, while lately there is also mention of permanent implantation (destination therapy) [1]. Currently there are two kinds of VADs available for clinical usage: the impeller pump types; and the displacement pump types. Extensive analytical, numerical and experimental investigations have been carried out to study various aspects of the VAD design and analysis, such as [2-15]. These numerical and experimental investigations greatly enrich our understanding of VAD interaction with the native cardiovascular system. However, the hemodynamic effects of the VAD are affected by many factors, such as: the VAD design itself; the VAD control mechanism; VAD motion pattern; VAD cannulation location etc. Further numerical, experimental and clinical studies are required in order to optimize the VAD design. In this paper a numerical investigation is carried out to study the cardiovascular response under the support of two types of VADs: impeller pump; and displacement pump.

# II. METHODS

The numerical model includes two parts: one for the native cardiovascular system; and the other for the VAD. Fig. 1 shows the schematic of the whole system. Impeller type or

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displacement type VADs are connected in parallel with the native left ventricle, by cannulating the inlet of the VAD in the left ventricular apex, and the outlet of the VAD in the ascending aorta.



Fig. 1 Schematic of the system configuration

#### A. Model for the native cardiovascular system

The detailed model for the native cardiovascular system has been introduced in a previous paper [16]. The system is modeled in three main parts: heart; systemic circulation loop; and pulmonary circulation loop. The heart is modeled as a four-chamber pump with variable elastance and four heart valves that control the blood flow direction. The systemic and pulmonary circulation loops are each separated into aortic sinus/pulmonary artery sinus, artery, arteriole, capillary and vein segments. In every segment the individual component is modeled by considering the local resistance to blood flow, elasticity of blood vessels, and inertia of blood. The combined effect of venule, vein and vena cava is modeled as the vein segment. The artery segment represents the general characteristics of the aorta, the main and smaller arteries. The aortic sinus describes the general feature of blood vessel around the aortic root. The heart valve dynamic operation is specifically modeled by considering the various factors of pressure difference, frictional force, vortex effect etc acting on the valve leaflets, which helps to give a more accurate description of cardiac response.

B. Model for the VADs





Various designs of the impeller pump and displacement

pump VADs are in use. The VAD motion mechanisms vary with the individual design. The current research aims to address the general cardiovascular hemodynamics under VAD support. Thus emphasis is placed on describing the inherent flow characteristics of the VADs in a manner that is independent from device implementation details. Therefore in the present study the VAD flow performance is directly specified: the VAD inflow and outflow changes are defined by referring to previously published VAD motion dynamics [2, 3]. In all cases the VAD motion control mechanism is assumed to work perfectly. More elaborate pump models based on dynamics analysis of these types of pumps will be presented in future studies, but they will not change the fundamental nature of cardiovascular system response presented here. Fig. 2(a) and (b) show the specified flow rate changes in the VADs.

The impeller pump does not have variable chamber volume, thus the volume flow rate at the inlet and the outlet of the VAD are the same ( $Q_{vad,i} = Q_{vad,o}$ ). Usually the impeller pump works with constant rotating speed and produces a constant flow-rate in the VAD to support the failing heart.

$$Q_{vad,i} = Q_{vad,o} = Q_0 \tag{1}$$

Pulsatile VADs work in several modes: fixed rate; fill to empty; and synchronized counter-pulsation. Compared to the other two modes, the synchronized counter-pulsation helps to achieve a greater reduction in pressure and volume of the heart, thus reducing myocardial wall tension and the determinants of myocardial oxygen consumption, while the increased diastolic arterial blood pressure improves coronary blood flow [4]. Thus the synchronized counter-pulsation is chosen as the representative pattern in this study.

Due to the variable VAD chamber volume, the flow-rates at the inlet and the outlet of the displacement pump type VAD are not equal during the heart cycle. As shown in Fig. 2 (b), the pump discharges to the ascending aorta during diastole, while it is filled during systole. Thus for the VAD inlet:

$$Q_{vad,i} = \begin{cases} 0.5Q_{p,i} \left( 1 - \cos \frac{2\pi \cdot t}{T_{T2}} \right) & 0 < t < T_{T2} \\ Q_0 & T_{T2} < t < T \end{cases}$$
(2)

and for the VAD outlet:

$$Q_{vad,o} = \begin{cases} Q_0 & 0 < t < T_{T2} \\ 0.5Q_{p,o} \left( 1 - \cos \frac{2\pi \cdot (t - T_{T2})}{T - T_{T2}} \right) & T_{T2} < t < T \end{cases}$$
(3)

The detailed values for  $Q_{p,i}$  and  $Q_{p,o}$  are inter-related so that their time-integrals during one full heart cycle are equal. These time-integrals form the main part of cardiac output when the native ventricle is dysfunctioning.

## C. Parameter settings

Values for the hemodynamic variables in the native cardiovascular model are the same as those used in [16]. Elastance values for the heart chambers are assigned by referring to parameter selection in [17, 18]. Parameter settings

for systemic and pulmonary loops are based on [17, 18]. In choosing the coefficients for the VAD dynamics, first the range of cardiac output with and without VAD support under normal, weakened and possible exercise conditions are identified based on physiological text books [19] and clinical results [5-7]. Then the coefficients for the equations describing the VAD dynamics are varied over a reasonable range to produce the compatible cardiac output changes in the different situations. Other parameters such as heart period were chosen based on general knowledge in physiological textbooks.

# III. RESULTS

Based on the mathematical models described above, a computer program is developed in C to simulate the dynamic changes in the cardiovascular system under various healthy, diseased, and VAD supported conditions. In the study, first the normal values are assigned to the cardiovascular variables to reveal the physiological response in healthy conditions. Next the left ventricular characteristics are changed to simulate the pathological condition of LV failure. Based on the LV failure condition, each of the VAD types is individually coupled with the diseased cardiovascular model, to study the dynamic response of the native diseased cardiovascular system under VAD support. The heart period is chosen to be 1 second in the healthy and VAD support simulations (60 beats per minute). In the simulations, the system often reaches periodic solution after 10 to 15 heart cycles of calculation. The converged solution in the period from the 36th to the 37th second is chosen in every simulated case for comparison of results. Figs. 3 to 7 illustrate the changes of important physiological variables such as pressure in the left ventricle and the artery in the different cases of simulation.

# A. Response in Native Cardiovascular System under Healthy and Left Ventricular Failure Conditions



Fig 3. Cardiovascular response under healthy condition



Fig 4. Cardiovascular response under heart failure condition

First the cardiovascular response under healthy conditions is simulated. For this purpose the normal physiological values are adopted in the model as in [16], and the VAD model is deactivated in the global model. Fig. 3 shows the simulation results for pressure and flow rate changes in the systemic loop under healthy conditions. The corresponding pressure-volume relation is illustrated in Fig. 7. In Figs. 3 and 7 it is observed that the corresponding simulation results agree well with the typical drawings for cardiovascular response shown in textbooks such as [19]. The left ventricular pressure is in the range of  $0 \sim 120 mmHg$ , and the aortic pressure changes between  $80 \sim 120 mmHg$ . Periodic peak flows exist in the mitral and aortic flows, which with forward and regurgitant flows reach an average flow-rate of about 5l/min. The left ventricular volume change is approximately from 50ml to 130ml, with a stroke volume of 80ml and ejected volume of 70ml. The 10ml volume difference is due to the regurgitant flow in the mitral and aortic valves (about 5ml each).

The pathological condition of left ventricular failure is simulated by reducing the value of the maximum elastance in the left ventricle to one-fifth of the normal value. Figs. 4 and 7 illustrate the simulated results for pressure, flow rate and volume changes in the systemic loop under the LV failure condition. Generally the computed cardiovascular response in the LV failure condition follows the same trend as that in the healthy condition, but the values for the physiological variables change greatly. In Fig. 4(a) it is observed that the aortic pressure range is reduced to  $46 \sim 68mmHg$  and the peak systolic left ventricular pressure is reduced to 68mmHg. This greatly impaired perfusion condition will adversely affect the function of important organs in the cerebral, renal and hepatic sub-systems. Also the pressure in the left atrium is elevated to about 14mmHg, almost increased 50% from the normal value of about 9mmHg. Fig. 4(b) illustrates that computed peak flow rates across the mitral and aortic valves are reduced to about 60% of the normal values. Fig. 7 indicates corresponding prominent changes to chamber volumes. The left ventricular volume varies between 164ml and 210ml, greatly elevated from the healthy range of  $50 \sim 130 ml$ , and with a corresponding decrease in the difference between the minimum and maximum volumes. These changes suggest that the ventricular volume difference has been reduced from the healthy 80ml to 46ml, with corresponding increase in cardiac rate and cardiac volume (LV dilation), and overall decrease in cardiac output.

# B. Response with VAD Support

To compare the cardiovascular responses under different kinds of VAD assisting actions, each of the VAD models introduced above is coupled to the native diseased cardiovascular model one at a time. The VAD flow-rate of 70 ml/s is chosen as the standard situation under which the various VAD configurations are compared. Figs. 5 and 6

illustrate the changes of pressure and flow rate in the systemic loop under heart failure condition assisted with impeller and displacement pump respectively. Fig. 7 illustrates the pressure-volume loop in the different situations.



Fig 5. Cardiovascular response for heart failure assisted with impeller pump VAD



Fig 6. Cardiovascular response for heart failure assisted with displacement pump VAD



Fig 7. P-V loop for the simulated cases

Comparison of pressure response in Figs. 5(a) and 6(a) shows that with either type of VAD support action, the arterial pressure can be restored from the diseased condition in Fig. 4 to the normal range of  $80 \sim 120mmHg$ . Similarly the left atrial pressure is restored from the diseased condition of about 14mmHg to the normal value of about 9mmHg, although the choice of VAD type and the VAD motion profile affect the pulsatility and amplitude of the pressure waveforms. Comparison of left ventricular pressure in Figs. 5(a) and 6(a) with that in Fig. 4(a) shows that, peak systolic left ventricular pressure is decreased to about 40mmHg with the impeller pump and to about 30mmHg for the displacement pump. Decreasing of ventricular pressure allows the ventricular muscle to relax, and in some situations to partially recover.

Comparison of representative flow-rate changes in the simulated VAD assistance cases is shown in Figs. 5(b) and 6(b). The mitral flow is generally restored to the same level as in the healthy condition. As the left ventricular pressure is lower than the aortic pressure throughout the heart cycle, the aortic flow becomes zero throughout the heart cycle, so that in effect the VAD takes over overall perfusion. This is undesirable, as in optimal VAD operation, along with assisting physiological perfusion, the VAD should also maintain mitral and aortic flows above zero [8], otherwise stasis may develop near the heart valves, and induce acquired valve stenosis [9]. This is a common shortcoming for the in-parallel connected VADs.

The pressure-volume loops for the left ventricle under the native and various VAD support configurations are compared in Fig. 7. It is observed that, either the impeller pump support, or the displacement pump support can restore the ventricular volume from the greatly dilated condition in ventricular failure to the normal range. At the same time the VADs unload the ventricle by decreasing the ventricular pressure. The displacement pump type VAD produces ventricular volume difference of about 80ml, very near to that in the healthy condition; while under the impeller pump type VADs the left ventricle has a reduced volume difference (about 40ml). Reduced ventricular volume change may "cause deleterious morphological changes in both the tissue and cell levels with ventricle re-modeling" [10]. Thus the numerical results indicate that the displacement pump support is more advantageous to the ventricular recovery than the impeller pump.

The area enclosed in the P-V loop corresponds to the external work generated by the left ventricle. Impeller pump VAD support produces less volume change, more pressure change, and eventually less ventricular contribution to blood-pumping work than that using displacement pump VAD. These numerical results suggest that for some patients the pulsatile VAD would induce the larger and almost physiological left ventricular volume change, and lower left ventricular pressure; but in other cases physicians may decide that the smaller impeller VADs, which induces higher ventricular pressure and less ventricular volume change, would be better suited for the particular patient's recovery.

# IV. DISCUSSION

This paper compares the hemodynamic response in the cardiovascular system under two types of VADs support: the impeller pump and the displacement pump. Comparison of the cardiac responses in the two VAD support configurations reveals that under both configurations the VAD can work to restore the arterial pressure and cardiac output from the diseased condition to healthy values.

From the results of impeller pumps it is observed that these induce only small variations in the arterial pressure and flow rate response. In the results of displacement pumps, pulsatility in arterial pressure and cardiac output similar to that in the healthy condition were generated, and the left ventricle is more adequately unloaded. These coincide with corresponding

results in similar research in Drakos et al.'s in vivo study [11] on 18 domestic pigs, and Koenig et al's in vitro study [12] in an adult mock circulation loop. According to previous research, pulsatile flow has an important effect on the circulation physiology: it promotes perfusion in the kidney and liver, and promotes microcirculation at the cell level that is quite important in early treatment of acute heart failure [13, 14]. In this sense the displacement pump has unmatched superiority over the impeller pump in maintaining adequate pulsatility that is beneficial to physiological perfusion of the peripheral organs and normal metabolism. In addition, in the displacement pump there are no high speed moving parts, thus avoiding the pitfalls of cavitation and damage to the blood cells that usually exist in the impeller pump. However, this does not mean that the impeller pumps are inferior. Except for their lack of pulsatility effect, impeller pumps have other advantages in assisting circulation. Minimum energy and power requirement, and simplicity of system configuration, are important factors in VAD design. The impeller pump is an axial or centrifugal impeller rotating in the pump housing, and eliminates the need for heart valves, resulting in the simpler system configuration and better mechanical reliability in the two types of VADs studied. The non-pulsatile nature of the impeller pump flow demands the minimized pump power requirement, while the displacement pump requires more power input.

Impeller pumps and the displacement pumps are usually considered for in-parallel flow connection to the native ventricle. Thus in maintaining the normal arterial pressure of  $80 \sim 120 mmHg$  and cardiac output of 70 ml, the VADs work under 100 % assistance condition. In the ideal situation the VAD should provide maximal augmentation of cardiac output while still keeping aortic valve opening [8]. This is not always achievable with in parallel connections because the existence of the aortic flow depends on the remaining strength of the diseased ventricle to produce a ventricular pressure higher than the arterial pressure produced by the VAD. In many cases of ventricular failure the in parallel connected VADs take over the total pumping action and completely unload the native ventricle [8, 9]. Under such situations the aortic valve is permanently closed [15]. This may cause stasis of blood on the ventricular side of the permanently immobilized aortic valve cusps. Such stasis of blood, sometimes despite anticoagulants, favors development of thrombi in this region, and in the long term leads to commissural fusion and acquired aortic stenosis [9]. In the situation of VAD for bridge to transplantation and permanent implantation, this effect can be allowed; but for the situation of bridge to recovery this complication needs to be carefully addressed. The stasis potential is an inherent disadvantage for the in parallel connection configuration of VADs.

The shortcoming of this paper is that the VAD dynamics are based on descriptions of the flow effects of the VAD assisting action but not the effects of preload and afterload on VAD operation. Also the VAD motion profiles used in this research are typical representative profiles. Elaborate VAD motion profiles (including motion curves, motion time delay, systole/diastole ratio etc.) may change the detailed response. This may cause small changes to the results, but will not qualitatively change the overall nature of the results or conclusions. The detailed VAD design can be modeled in order to obtain more accurate results and to investigate the individual contribution to cardiovascular dynamics. These are the subjects of future investigations.

## V. CONCLUSION

This paper compares the hemodynamic response under two types of VADs assisting action through numerical investigation. Results show that the impeller pump produces small pulsatility in arterial pressure and flow-rate. The displacement pump generates distinguishable pulsatility in arterial pressure and flow-rate. From a purely hemodynamic point of view, the impeller pump requires less power input than the displacement pump. The simulations results agree reasonably well with the published experimental results. The developed model can be extended to study the cardiovascular response under various VAD assistance conditions, such as under different VAD cannulation configurations and different degrees of heart failure situations.

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