CONTROL OF A NON-BLOOD CONTACTING CARDIAC ASSIST DEVICE

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ABSTRACT
Cardiac assist devices are currently being developed with the aim of providing physical pumping assistance to a weakened or failing heart. This paper concerns the concept and control of an assist device using an artificial-muscle wrap consisting of contractile bands. A heart simulator has been constructed in order to test the function and develop the control of these contractile bands. The simulator represents a section through a ventricle, and can physically replicate the dynamics of hearts in various states of health. With a prototype assist device wrapped around the simulator, the effect of any applied assistance can be quantified. We demonstrate that an impedance control strategy is suitable for control of an assist device, as this provides an assistance level that automatically adjusts as appropriate to the natural ability of the heart.

KEY WORDS
Cardiovascular, Heart Assist Device, Control, Modelling

1. Introduction
Coronary heart attacks or viral diseases can weaken the pumping ability of the heart muscle, reducing the effectiveness with which oxygen can be delivered to muscles and organs. As transplant hearts are rarely available, efforts are increasingly being directed to mechanically assist the heart.

Implantable pumps are becoming available, however the problems of immune-system rejection and clotting must be closely monitored [1]. These complications are the result of blood flowing through an artificial chamber. Alternatively, to avoid contact with the blood, direct cardiac compression (DCC) can assist the heart by providing a compressive pressure to its outer surface. A procedure known as cardiomyoplasty involves creating a wrap of latissimus dorsi muscle around the heart, which is stimulated to contract in synchronisation with the heartbeat [2]. This technique was shown to be successful in that the heart can be briefly assisted. However, skeletal muscle differs from healthy heart muscle in that it fatigues rapidly, therefore stimulation has to be carefully controlled [3]. Furthermore, the electrical impulses used to stimulate the muscle preclude the use of a pacemaker.

The difficulties associated with using human muscle have driven attempts to create a mechanically driven, "artificial muscle" assist devices [4]. Pneumatic pressure has been applied to animal hearts in-vivo by placing the heart within a pressure vessel [5], surrounding the heart with an inflatable cuff [6], or applying inflatable patches [7]. These trials have demonstrated that pneumatic actuation can successfully support weakened and failing hearts. The pneumatic actuators (cuffs, patches) have benefits of high power/weight and power/volume ratios. However, air hoses passing through the skin are a potential source of infection, and attachment to a pneumatic pressure supply severely limits mobility. Additionally, there is potentially difficulty in controlling pneumatic pressure due to the non-linear pressure/flow relationship of valves, and the visco-elastic silicone construction of the actuators.

Several new "artificial muscle" actuation technologies are being developed; a prototype cardiac assist device using electro-active polymers has been investigated [8]. These new technologies are at early stages of development, and are not yet suitable for a cardiac compression device.

2. Concept of Assist Device
This paper describes the concept and control of an assist device consisting of several independently-actuated contractile bands. Direct-drive, miniature D.C. motors are employed to control the circumference of flexible bands, placed around both ventricles of the heart, as shown in figure 1.
Compared to pneumatic actuation, the torque and position of these motors are easily controlled by computer with the use of miniature sensors, or self-sensing strategies [9]. They are suitable for use with an implantable battery power supply. Long-term device life is being determined by endurance trials. If necessary, brushless commutation could be used to increase motor life; brushless motors are currently in use within implantable pumps.

The human heart comprises two displacement pumps; the right side pumps deoxygenated blood through the lungs, and the left side pumps oxygenated blood from the lungs to the rest of the body (figure 2 shows a representation).

Diastole describes the period in which the ventricles relax and are passively filled by blood at a low pressure. The pressure required to expand the ventricles increases with volume since the myocardium is naturally elastic, (the passive diastolic pressure-volume relationship, PDPVR). The atria contract to provide a further increase in pressure that completes the filling process at end diastole.

An assist device surrounding the ventricles could increase the PDPVR, which could impede filling and reduce stroke volume. However, there is also a benefit to be gained from preventing the heart from over-inflating: according to Laplace's law, the wall tension required to support a given pressure increases with ventricular volume. Dilated cardiomyopathy is a condition in which a patient's cardiac output is reduced as a result of an over-dilated ventricle. An implant is currently undergoing clinical trials to treat this condition: the CorCapTM [10] is an inelastic mesh that surrounds the ventricles and limits their maximum diameter. The device presented herein is also able to provide this function, furthermore its diameter is controllable to adjust with varying heart conditions.

The contraction of the ventricles and expulsion of blood is termed systole. This period begins with an iso-volumetric contraction, whereby the myocardium contracts, increasing the pressure within the ventricle until it is sufficient to open the aortic valve and expel blood (nominally 70-80 mmHg). The myocardium then continues to contract and the ventricular volume decreases as blood flows from the heart. Pressure rises to a maximum (nominally 120 mmHg) in this phase.

As the circumference of the ventricles decreases during systole, the heart also exhibits a twisting motion of around 20 degrees [11]. Skewed placement of some of the contractile bands would enable this twisting motion to also be augmented.

As the contractility of the myocardium decreases, the aortic valve closes and pressure falls rapidly at a constant volume. When the myocardium has fully relaxed, filling (diastole) begins again.

3. Control Techniques

Control of the assistance force applied to the heart is crucial to the device’s success. If a damaged heart is rested, studies have shown that cardiac muscle can regenerate [12].

The assistance required in-situ will vary greatly, sometimes beat-to-beat, depending on the fluctuating levels of natural heart function and bodily exertion. It is important to synchronise the assist compression with the systolic phase of the heart as any antagonistic motion could potentially injure the surface of the myocardium [13].

The coronary artery, which lies on the surface of the heart, is in potentially at risk of being occluded by an assist device. Restriction of blood to the myocardium would hinder regeneration, or could cause infarction. Therefore contact force must not be higher than necessary, particularly in diastole when most blood perfusion occurs.

The physiological factors described above must be considered when choosing a controller for an assist device. A control scheme that is used by some pneumatic devices is to apply a constant assist pressure during each systole phase. However, without feedback regarding the function of the heart, this type of controller could be applying more assistance than necessary, with potentially harmful results. What is required is a controller that can sense when the natural ability of the heart is insufficient, and provide assistance appropriately.

This specification is most similar to the strategy of the impedance control technique, developed for interaction between robot manipulators and non-rigid environments [14]. To paraphrase, this controls the motion of a manipulator (assist device) and in addition provides a "disturbance response" for deviations from that motion which has the form of an impedance. Thus, the impedance controller would control the assist device to follow the volume-time trajectory of a "normal" heart. If the heart lacks the natural ability to follow this desired trajectory then the assist controller will "encourage" it in relation to the amount of shortfall.

The transfer function for impedance control can be specified in the s-domain (where s represents differentiation with respect to time) as:

\[
\frac{F_s}{x_s} = Ms^2 + Cs + K
\]

(1)

Thus the controller accepts a position error from the desired trajectory, \(x_s\), and produces an assist force \(F_s\). The impedance is made from mass, damping and stiffness components M, C and K respectively. The impedance filter used for the assist application does not include a mass term. This is to maintain stability, since numerical
differentiation is on the limit of causality, and double-differentiation (the \(s^2\) term) amplifies any high-frequency noise present. Tuning these stiffness and damping parameters thus adjusts the tolerance of the controller to variation from the desired position trajectory.

The control scheme is hierarchical, with a high-frequency impedance control loop, and a low-priority routine that determines the high-level desired trajectory and allows for adjustment of the controller parameters.

4. Experimental Methods

A simulator has been constructed to evaluate the effectiveness of prototype compression assist devices. The simulator concerns just the left side of the heart as it is seen as more clinically important, and the majority of efforts to provide assistance concern assisting the left ventricle. The expansion and contraction of an axial slice through the left ventricle is represented as shown in figure 2. The cylindrical section is represented in a hardware heart simulator device. This comprises six moveable posts, vertically oriented and each 10mm high, arranged to form a regular hexagon. The diameter of this hexagonal simulator is computer-controlled to mimic the beating motion of the ventricle. This simulator enables a prototype assist device to be mechanically tested on a physical, beating, model of a heart. The force produced by the assist device is measured with a custom-designed force sensor placed between the assist band and the heart simulator.

The pressure within the left ventricle is the sum of the passive pressure produced by the myocardium, \(P_{pd}\), the active pressure of the myocardium \(P_{mc}\), and the assist pressure \(P_{assist}\). The assist pressure can be calculated from the measurement of the force sensor. With the polygonal geometry of the simulator, the force recorded normal to the simulator surface is equal to the tension of the band. The ventricular pressure produced for a particular circumferential assist tension is given by:

\[
P_{assist} = \frac{T}{r_{ext} h}
\]

Alternatively, it is possible to perform a simulation without an assist device, by simply routing the output from the controller into the pressure sum (performed in software). This simulates an ideal assist device, that instantly applies the pressure requested by the controller. This was used to compare controller types independently of the effects of the assist device.

![Figure 2 Block diagram of the control scheme](image-url)
The passive myocardium is modelled by a non-linear spring, with a rest volume of 40ml. These properties are determined by the ventricle's wall thickness and elasticity - various cardiac conditions can be modelled by altering this PDVPR function.

The pressure produced by the contracting myocardium is a function of its contractility and also the ventricular volume and pressure at onset of systole (preload). These factors relating to the circulatory system have been left out of the simulation in order that the effect of assist pressure could be identified more clearly. For these studies, the pressure produced by the myocardium has been represented by a time-varying pressure corresponding to an average, healthy heart [15]. Impaired cardiac function was produced by simply scaling down the pressure-time function produced by the myocardium.

The changing volume of the ventricle following the onset of systole is given by the flowrate/pressure relationship that is the impedance of the vascular system as seen at the entrance to the aorta. This is here modelled with a Windkessel function, which models the vascular compliance and resistance to flow with a simple resistor-capacitor network. The model parameters can be changed to represent various vascular conditions (e.g. stiff, constricted vessels), and the function provides a good approximation of total flow quantity and mean flow rate [16].

The simulator only represents one section of the ventricle, of height, h. In calculating the total output flow, it is assumed that assist bands would be repeated to cover the entire ventricle, and the pressure $P_{\text{assist}}$ would be applied throughout.

Onset of systole is triggered by a pacemaker. In this laboratory case, a constant output rate of 70bpm is set, however the pacemaker can sense the natural rhythm of the heart from electrodes if required. The diastolic period - ventricular filling - depends on the venous pressure, which in turn depends on the previous history of cardiac output function, and the condition of the vascular system. It is possible to include a physiological circulatory system model in the simulator, to "close the loop" however that has not been modelled for this study since the assist device only operates in systole. During diastole, the simulated ventricle is controlled to fill at a constant rate to a predetermined end diastolic volume.

The heart simulator was used to model the effect of adding assistance to hearts in various stages of health - normal (100% function), and weakened conditions of 70% and 50% function. Impedance control and a constant applied-pressure controller were compared in simulation using the assumption of an ideal assist device. Impedance control was then applied to a D.C. motor-based assist device, which was wrapped around the beating heart simulator.

5. Results

Figure 3(a) shows example traces of volume and assist pressure from a simulation of a weakened heart; the active myocardial pressure is 70% of normal. The reduced pressure within the ventricle resulted in slower emptying through the Windkessel model of circulatory system impedance. Therefore the overall stroke volume decreases.

The effect of adding cardiac assist is also shown on figure 3(a) - in this case, impedance control was applied to the heart via an assist device. Throughout the contraction, the error between the actual instantaneous volume and the desired volume trajectory was applied to the impedance controller. This calculated an assist pressure dependent on the size and rate of change of the volume error and resulted in a contractile force being applied by the assist device. The resulting increase in pressure increased the flowrate out of the ventricle in the heart model.
impedance controller. The thinner trace shows the pressure applied to the simulator by the assist device. An ideal assist device would instantly apply the exact pressure requested by the controller. However, we found a delay in the reaction capability of the assist device. This was particularly noticeable in early systole, where the velocity of the heart is a maximum and the assist device must "catch up". This result indicates that this type of assist device requires advance notification of systole, in order to accelerate up to the necessary contraction speed.

![Pressure-volume loop](image)

**Figure 4: Reduction in cardiac output**

Pressure-volume loops are commonly used to illustrate mechanical function of the heart; the area enclosed by the loop is equal to the mechanical work done in that heartbeat. In this study, the instantaneous pressure-volume co-ordinates of the modelled left ventricle were plotted for each heart cycle. Point A corresponds to the beginning of systole, from here (moving anti-clockwise) the pressure within the ventricle rises to B, and the ventricle starts to reduce in volume as blood is expelled. Point C marks the end of contraction, the end-systolic volume, and then diastolic filling occurs from D to A. Note that in this simplified simulation, systole starts from the same point (A) for each test condition.

Figure 4 shows how the cardiac output decreased when the natural heart function was reduced to 70% of normal pressure (dotted line). The reduced pressure decreases the height of the P-V loop, and the resulting decrease in stroke volume decreases the width of the loop. Thus the loop area, representing mechanical output power, is reduced in this weakened state. From this level, when impedance control was applied to the assist device, the systolic pressure and stroke volume both increased (dashed line). The increase in loop area shows the energy provided by the assist device in one cycle.

A comparison between the strategies of constant assist pressure and impedance control was performed in simulation, by removing the physical assist device from the heart simulator rig, and assuming an ideal assist device instead. The increase in cardiac output power (P-V loop area X heart rate / 60) that was provided by assistance is shown in table 1.

<table>
<thead>
<tr>
<th>Cardiac Function</th>
<th>Natural Power</th>
<th>Impedance Control</th>
<th>Constant Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>100%</td>
<td>0.809</td>
<td>0.000</td>
<td>0.321</td>
</tr>
<tr>
<td>70%</td>
<td>0.424</td>
<td>0.180</td>
<td>0.214</td>
</tr>
<tr>
<td>50%</td>
<td>0.232</td>
<td>0.252</td>
<td>0.179</td>
</tr>
</tbody>
</table>

*Table 1: Power of assisted hearts in simulation, Watts*

When applying a constant pressure over the systolic period, cardiac output power in our simulation increased for all heart conditions, even when the heart's natural function was normal. In that particular situation, the additional pressure produced by the assist device would cause an unhealthily elevated blood pressure. As the heart weakened, the same assist pressure was applied, however the additional power provided actually decreased, due to the smaller stroke volume at lower natural heart function.

In contrast, when using an impedance control strategy, the assist pressure increases with a weakening heart. This increases the stroke volume and therefore the additional pumping power provided is shown to increase. When the heart is functioning normally, that is to say that it is meeting the current pumping output demands of the circulatory system, then the assistance power drops to zero.

6. Conclusions

Control strategies for a non-blood contacting cardiac assist device have been presented and illustrated experimentally. The impedance control strategy has been demonstrated to provide stable, predictable and controllable interaction with a beating heart. Impedance control automatically adjusts the assist level applied instantaneously depending on the heart's natural ability. This minimises unnecessary contact pressure, which could damage the surface of the heart. Further benefits are that the power supply is conserved and the operational life of the device is extended. The assistance applied can be controlled at a higher level by altering the desired volume-time trajectory, and by altering the stiffness and damping coefficients of the controller.

Key to designing and developing this control strategy has been the experimental heart simulator. During this study, several assumptions and simplifications were made to produce a basic circulatory system model. There is considerable scope to add detail to this model in software, while using the same hardware to interact with developmental assist devices. There is also potential to
increase the detail of the physical representation of the simulation. Each of the heart simulator's 6 sections can be independently controlled, allowing for realistic simulations of regional weakness; regional wall motion abnormalities are commonly caused by myocardial infarctions [17].

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