Design of a Muscle-powered Extracardiac Counterpulsation Device for Long-term Circulatory Support

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1 Background

While great strides continue to be made in the treatment of congestive heart failure using mechanical ventricular assist devices (VADs), several longstanding difficulties associated with pumping blood continue to limit their long-term use. Among the most troublesome has been the persistent risk of clot formation at the blood-device interface, which generally requires VAD recipients to undergo costly anticoagulation therapy for the duration of the implant. Another serious and persistent problem with long-term use of these pumps is the increased risk of infection associated with the use of percutaneous drivelines.

To address these issues we are currently exploring a new approach to blood pump design that aims to solve both these problems by avoiding them altogether. Toward that end, we propose to harness the body’s own endogenous energy stores in order to eliminate the need to transmit energy across the skin. Further, we intend to transfer the energy from this internal power source to the circulation without contacting the blood to obviate the thrombogenic risks imposed by devices placed directly into the bloodstream.

To power the implant we will employ a device developed previously by our group called a muscle energy converter (MEC), shown in Figure 1. The MEC is, in essence, an implantable hydraulic actuator powered by the latissimus dorsi (LD) muscle with the capacity to transmit up to 1.37 joules of contractile work per stroke [1]. By training the muscle to express fatigue-resistant oxidative fibers and stimulating the LD to contract in coordination with the cardiac cycle, the MEC captures and transmits this contractile energy as a high-pressure low-volume (5 cc) hydraulic pulse that can be used, in principle, to actuate an implanted pulsatile blood pump.

The goal of this research is to use the low-volume output of the MEC to drive a polymer-based aortic compression device for long-term circulatory support. In this context it is important to note that the idea of applying a counterpulsation device around the ascending aorta is not new. Indeed, this approach has been validated by clinical trials recently completed by Sunshine Heart Inc. showing that displacing 20 cc of blood at the aortic root has significant therapeutic benefits [2]. Unfortunately, while the pneumatic ‘C-Pulse’ device solves the blood-contacting problem, it suffers from the same limitations as traditional VADs—i.e., driveline infections. The device described here achieves the same volumetric displacement as the SSH device via geometric amplification of MEC outputs. Thus, through this mechanism we believe the low-volume power output of the MEC can be used to support heart failure patients while addressing the major limitations associated with long-term VAD use.

2 Methods

The active element of this compression device is a simple array of thin-walled polymer tubes connected side-to-side. These tubes have a circular cross-section when inflated and assume a flattened configuration when empty. Consequently, when the inflated tubes are arranged in a circular pattern the perimeter they form is \( nd \), where \( n \) is the number of tubes and \( d \) is the diameter of each individual tube. When a negative pressure is applied, the circumference of the circle enlarges to \( \pi nd/2 \). In essence then, each tube acts as a contractile element, the net effect of which is illustrated in Figure 2.

![Figure 1](https://example.com/fig1.png)

**Figure 1.** Computer rendering of a muscle-powered counterpulsation system implanted in a patient (stimulator not shown).

![Figure 2](https://example.com/fig2.png)

**Figure 2.** Design Schematic. A) Thin-walled tubes are arranged in a circle and drawn toward the center during inflation. B) Dimensions of a single contractile element.

Because the ascending aorta is a thick-walled vessel, a compression force applied uniformly around the circumference would preferentially increase circumferential hoop stress within the vessel wall, causing the aorta to buckle under the pressure. To avoid this problem, the design concept described above was modified to redirect the primary compression force away from the vessel walls and toward the center of the lumen. To accomplish this, seven tubes in the original 27-tube design were replaced by two thin polymer sidewalls positioned opposite to one another in the array as shown in Figure 3. In so doing, the contractile force of the tubes are made to draw these walls toward one another, resulting in a bi-directional displacement that gently...
compresses the aorta without generating damaging hoop stresses within the aortic wall. Finite element analysis was used to further explore the physical properties of this design.

Simulations of device performance were run with a linear solver and material properties selected from Autodesk’s repository. The stiffness of the walls and tubes were modulated and subjected to a range of actuation pressures. Device function and viability were evaluated based on four key parameters: pressure requirements, Z/X axis deformation, change in cross-sectional area, and flex life.

Figure 3. Hydraulic aortic compression device with sidewalls (1) drawn together by tubular contractile elements (2).

3 Results

Pressure requirements. To answer this first question we used the following energy balance equation: \( P_{\text{hydro}} \times V_{\text{hydro}} = P_{\text{blood}} \times V_{\text{blood}} \). Essentially, the volume of the fluid used in the compression device (5 cc) times the pressure at which the device is operating must, at minimum, equal the amount of blood displaced from the aorta (20 cc) times the diastolic blood pressure of the patient. Because heart failure patients can be hypertensive, a mean value of 180 mmHg was used to account for high-end power requirements. Solving the balance shows that the MEC must generate 720 mmHg (0.1 MPa) to actuate this counterpulsation device under hypertensive loading conditions. Because the LD/MEC complex can produce pressures over 2,700 mmHg (0.36 MPa) with a fully-conditioned muscle contracting at peak efficiency, we conclude that the working parameters for the device are more than sufficient to operate against both normal and elevated arterial pressures.

Z/X axis deformation. Two main parameters were investigated with respect to Z/X axis deformation: actuation pressure and material properties. The material properties of the tubes were changed independently of the material properties of the walls to determine how device performance would respond. Two main conclusions were drawn from these analyses. First, the Z/X ratio remains linear across varying pressures for any given tube/wall material pairing. And second, when the walls are made \( \geq 10 \) times stiffer than the tubes, the z-axis sidewalls move 2.5 times closer to the center than the x-axis tubular elements.

Change in area. The change in device cross-sectional area needed to displace 20 cc with a device length of 3.5 cm is 570 mm\(^2\). To evaluate how much area change a particular deformation causes, the area of the deformed (inflated) device is subtracted from the area of the relaxed (deflated) device. To calculate these areas, we treated both inflated and deflated shapes as ellipses since they have a major and minor axis. The starting device measures 31.87 mm in the Z-axis and 34.61 mm in the X-axis resulting in a starting area of 866.3 mm\(^2\). Using the same combinations of materials and pressures described above, we calculated area change as a function of actuation pressure and plotted the results in Figure 4. Here we observe the same material trends as described in the previous section, i.e., if the difference between wall stiffness and tube stiffness is large enough, the devices share the same area change function. These studies show that a device with stiff sidewalls achieves the minimum required area change of 570 mm\(^2\) with just 413 mmHg (0.055 MPa) inflation pressure, well within the functional capacity of the LD/MEC complex.

References