Thoughts and Progress

Design of an Artificial Left Ventricular Muscle: An Innovative Way to Actuate Blood Pumps?

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Abstract: Blood pumps assist or take over the pump function of a failing heart. They are essentially activated by a pusher plate, a pneumatic compression of collapsible sacs, or they are driven by centrifugal pumps. Blood pumps relying upon one of these actuator mechanisms do not account for realistic wall deformation. In this study, we propose an innovative design of a blood pump actuator device which should be able to mimic fairly well global left ventricular (LV) wall deformation patterns in terms of circumferential and longitudinal contraction, as well as torsion. In order to reproduce these basic wall deformation patterns in our actuator device, we designed a novel kind of artificial LV “muscle” composed of multiple actively contracting cells. Its contraction is based on a mechanism by which pressurized air, inside such a cell, causes contraction in one direction and expansion perpendicular to this direction. The organization and geometry of the contractile cells within one artificial LV muscle, the applied pressure in the cells, and the governing LV loading conditions (preload and afterload) together determine the global deformation of the LV wall. Starting from a simple plastic bag, an experimental model based on the abovementioned principle was built and connected to a lumped hydraulic model of the vascular system (including compliance and resistance). The wall deformation pattern of this device was validated visually and its pump performance was studied in terms of LV volume and pressure and heart rate. Our experimental results revealed (i) a global LV motion resembling a real LV, and (ii) a close correlation between our model and a real LV in terms of end-systolic volume and pressure, end-diastolic volume and pressure, stroke volume, ejection fraction and pressure-volume relationship. Our proposed model appears promising and it can be considered as a step forward when compared to currently applied actuator mechanisms, as it will likely result in more physiological intracavity blood flow patterns. Key Words: Actuator—Artificial—Blood pump—Contraction—Heart—Left ventricle—Muscle—Pneumatics—Torsion—Wall deformation.

Blood pumps are primarily designed to take over the pump function of a failing heart. The main difference between the different pumps is the pulsatility. Pulsatile devices consist of a pusher plate-activated device or collapsible sacs that are compressed by pneumatic power (1, 2), whereas nonpulsatile devices are essentially motor-driven centrifugal pumps (2). Although the pump function of the pulsatile actuation mechanisms has already been proven, it does not account for realistic wall deformation, nor can intraventricular blood flow patterns be realized in these blood pumps.

In this study, we propose an innovative design of a blood pump actuator device that has the ability not only to function as a pump but also to mimic fairly realistic global left ventricular wall deformation patterns. It consists of an active wall structure mimicking the heart muscle, which efficiently exerts pressure on the blood. To demonstrate the actuation mechanism, we proposed a strategy to build the artificial left ventricular muscle with a simple plastic bag. We also built and tested an experimental model to demonstrate the pump performance of the artificial left ventricular muscle.

PHYSIOLOGICAL BACKGROUND

The heart is a four-chambered, muscular organ that continuously pumps blood through the body’s extensive network of arteries and veins. The left ventricle (LV) can be considered as its most important and powerful pumping chamber and is, therefore, probably its most frequently studied chamber. The LV wall consists of three layers: the epicardium, the myocardium, and the endocardium. The endocardium is the thin inner layer of endothelium, while the epicardium is an external visceral layer covering the heart. The myocardium, the middle layer, forms the bulk of the LV and is the layer that actually contracts. It consists mainly of cardiac muscle fibers embedded in connective tissue structure. The myocardium consists of interweaving bundles of cardiac muscle fibers spirally arranged around the circumference of the heart.
As a result of this anatomic architecture and the timing and sequence of electrical excitation, during contraction, the LV deforms as follows: (i) the diameter of the LV decreases, while (ii) the base (the atroventricular valve plane) is simultaneously pulled downward in the direction of the apex (iii) in a rotating manner (3). Due to this accompanying “wringing” effect, pressure is efficiently exerted on the blood enclosed within the chamber, thereby directing it upward toward the aortic outflow tract. As such, global ventricular wall deformation can be basically described in terms of (i) circumferential contraction; (ii) longitudinal contraction; and (iii) torsion.

CONTRACTION MECHANISM

In order to reproduce these basic wall deformation patterns in our actuator device, we designed a novel kind of artificial heart “muscle” composed of multiple actively contracting cells. Contraction is based on a mechanism by which pressure inside such a cell causes expansion in one direction and contraction in another direction. The contraction (systole) and relaxation (diastole) of a biologic muscle (Fig. 1 Panel I(a)) take place along the cardiac muscle fiber direction, while the contraction (inflation) and relaxation (deflation) in a single contractile chamber of this new artificial LV muscle (Fig. 1 Panel I(b)) take place perpendicular to its long axis. The final deformation of the LV wall is determined by the organization and geometry of the contractile cells within one artificial LV muscle, together with the governing LV pressure and the applied pressure in the cells. In the present active LV wall structure, only one layer of artificial muscle was used. However, a well-thought-out configuration of a number of artificial muscles could be used in future blood pumps to produce even more realistic deformations.

In its unpressurized state, a basic contractile cell can be considered as a flat cavity with a rectangular ground surface. When pressurized air is supplied to the cell, it inflates and becomes round (Fig. 1 Panel I(b)). Because the diameter of the inflated cell is less than the width of the deflated cell, both ends of the chamber move toward each other, and the cell starts to contract. The cell shortening \( \Delta L_{\text{cell}} \) then equals the deflated width minus the inflated diameter. As such, the contractile cell shortens and thickens simultaneously, which is, to some extent, similar to the kinematics of the cardiac tissue.

The global deformation patterns of the LV, that is, longitudinal and circumferential contraction and torsion, can be achieved by arranging the contractile cells under a certain angle (\( \alpha \)) relative to the LV long axis (centerline Fig. 1 Panel I(d)). As such, exclusively longitudinal contraction and \( \Delta L_{\text{long}} \) exclusively circumferential contraction \( \Delta L_{\text{circum}} \) and torsion in combination with longitudinal and circumferential contraction \( \Delta L_{\text{long}}, \Delta L_{\text{circum}}, \) and \( \beta_{\text{tension}} \) are obtained by arranging the contractile cells respectively at an angle \( \alpha = 90^\circ \), \( \alpha = 0^\circ \), and \( 0^\circ < \alpha < 90^\circ \) to the long axis of the LV. We found analytically that \( \beta_{\text{tension}} \) reaches a maximum of approximately \( 13^\circ \) at an angle \( \alpha = 39^\circ \) and, therefore, used this angle in our model (Fig. 1 Panels II and III). Also, the width of the deflated chamber was set to 15 mm.

In order to clarify the principle of the global LV wall deformation in our model, we assume a deflated artificial muscle (Fig. 1 Panel I(c)) that is rectangular in shape and consists of a number of contractile cells that are oriented at an angle \( \alpha \) between \( 0^\circ \) and \( 90^\circ \) with respect to the LV long axis. By rolling up the rectangular-shaped artificial muscle, a cylindrical LV model is obtained (Fig. 1 Panel I(d)) in which the seal (dotted line) is directed parallel to the LV long axis. When pressurized air is applied to each individual cell of the rectangular-shaped artificial muscle, contraction in the cells takes place in a direction perpendicular to its long axis. Because the cell’s long axis is oriented at a certain angle \( \alpha \) between \( 0^\circ \) and \( 90^\circ \) with respect to the LV long axis, the contraction of the cells results in a transformation of the initial rectangular to a parallelogram contour. In comparison with the rectangular, the parallelogram has become shorter \( \Delta L_{\text{circum}} \) (circumferential contraction), less high \( \Delta L_{\text{long}} \) (longitudinal contraction), and has rotated by an angle \( \beta_{\text{tension}} \) (Fig. 1 Panel I(c)). By rolling up the parallelogram-shaped artificial muscle, a cylindrical LV model is created (Fig. 1 Panel I(d)) in which the seal (dotted line) is oriented at the angle \( \beta_{\text{tension}} \) to the LV long axis. In this way, a cylindrical LV model is obtained in which longitudinal \( \Delta L_{\text{long}} \) and circumferential contractions \( \Delta L_{\text{circum}} \), as well as torsion \( \beta_{\text{tension}} \) are realized when activated.

PRACTICAL GUIDELINES FOR BUILDING THE ARTIFICIAL LV MUSCLE

In order to build a cylindrical three-dimensional artificial LV muscle, a step-by-step plan can be followed. First, reform the rectangular contour of the pattern depicted in Fig. 1 Panel I(c) (deflated) into a parallelogram with an angle \( \alpha \). Make sure to add at the top a horizontal cell that is connected to every single cell. In this way, air can be spread among all the cells in the LV wall. After that, draw the cell
I. CONTRACTION PRINCIPLE

Biological Muscle

- DIASTOLE
- SYSTOLE

Artificial LV Muscle

- DEFLECT
- INFLATED

II. GLOBAL LV WALL DEFORMATION

Longitudinal Contraction - Circumferential Contraction - Torsion

III. PUMP PERFORMANCE
pattern on a double film of plastic, for example on a simple plastic bag. Weld the plastic films together according to the cell pattern to obtain the two-dimensional (2-D) artificial muscle. Then, to achieve a cylindrical artificial LV muscle, fold the 2-D artificial muscle and weld the left and right sides together. Next, close the cylindrical artificial LV muscle wall near to the LV apex by bringing the wall together to one point and then welding this all together. Finally, insert an air tube in the top cell. The artificial LV muscle is now ready to be activated. When blowing into the air tube, the artificial LV muscle will contract and the three global deformation patterns can be visually observed.

As the biologic LV cavity is better represented by a truncated ellipsoid than by a cylinder, we subsequently built an ellipsoidal artificial LV muscle instead of a cylindrical one. As anticipated, the three basic wall deformation components could also be visually observed in the more realistic ellipsoid artificial LV muscle (Fig. 1 Panel II).

Weld quality is crucial for the well functioning of the active LV wall structure. Weld properties are determined by the type and thickness of the thermoplastic film as well as the welding temperature, pressure, and time. The thermoplastic film used in our artificial LV muscle was polyethylene with a thickness of 0.05 mm and was welded with a vacuum sealer (Krups Vacupack 2 Plus, Krups, Peeria, IL, USA). The welding time and pressure were adjusted by trial and error in order to obtain qualitative welds.

**PRELIMINARY FUNCTIONAL TESTS**

Besides fairly realistic wall deformation, this new artificial LV can also achieve the pump function performance of a real LV. In order to prove this, we modified the prototype presented above in three aspects. First, in order to obtain realistic performance in terms of pressure and flow, we reinforced the model using eight instead of only two layers of polymer. Secondly, in order to increase the systolic and diastolic speed, we lowered the airflow resistance by modifying the chamber pressure supply: the single collector tube is replaced by a number of individual tubes supplying pressurized air to each chamber individually. Thirdly, in order to simplify the sealing of the experimental model, we opted to supply the pressurized air at the apex instead of at the atroventricular valve plane. After the modifications (Fig. 1 Panel III), we connected our pump to a lumped hydraulic windkessel model of the vascular system (including compliance and resistance) and measured pressure (disposable transducer DTX/PLUS, Becton Dickinson Critical Care Systems, Franklin Lakes, NY, USA) and flow (Tubing Flow Sensor ME-PXL, Transonic Systems Inc., Ithaca, NY, USA). In this preliminary study, wall deformation patterns were only visually observed (Video Clip S1). Our experimental results show that the model is able to generate realistic data in terms of pressure and flow. Our experimental results show that the model is able to generate realistic data in terms of end-systolic volume (54 mL) and pressure (115 mm Hg), end-diastolic volume (156 mL) and pressure (~4 mm Hg), stroke volume (102 mL), ejection fraction (65%), mean outflow (6.8 L/min), heart rate (67 BPM), peak positive and negative dP/dt (1072 and −1134 mm Hg/s), and pressure–volume relationship. The systolic ejection phase, however, is suboptimal due to the relatively simple afterload model that generates unphysiological reflected pressure and flow waves.

**CONCLUSIONS**

We have developed an innovative blood pump actuator device that mimics the global kinematics of
the LV during the cardiac cycle and produced a
demonstrative model by making use of handicraft
material and tools. Besides fairly realistic wall defor-
mation, our experimental results show that this
new artificial LV is also able to generate realistic
data in terms of volume and pressure. However,
unlike the biologic system, in which contraction
occurs along muscle fiber direction, the long axis of
the contractile chambers in our artificial LV muscle is
perpendicular to the shortening direction. Another
difference with the biologic system is that, in our
artificial LV muscle, air is added during systole and
removed during diastole, whereas the volume of the
biologic heart muscle remains virtually constant
during the cardiac cycle. It is to be acknowledged
that biocompatibility might be a complex issue with
our design, given the complex internal surface and
folding of the ventricle. Nevertheless, our proposed
artificial LV muscle appears promising and can be
considered as a step forward when compared with
currently applied actuator mechanisms, as it will likely
result in more physiological intracavity blood flow
patterns.

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Supporting Information

Additional Supporting Information may be found in
the online version of this article:

**Video Clip S1.** Movie of wall deformation patterns of
the artificial left ventricular muscle. The movie is in
Windows Media Player format.

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