Iterative Learning Control of a Left Ventricular Assist Device

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Abstract: Due to the lack of donor organs, the importance of left ventricular assist devices (LVADs) increases. State of the art is to operate the pumps with a constant speed (CS) leading to effects such as underpumping, ventricular suction or the backflow of blood from the aorta in the ventricle. The end-diastolic volume (EDV) is influenced by venous return, as well as diastolic function and systolic pressure development. Thus it is a good medical indicator of ventricular load. In this paper a norm-optimal iterative learning control (NOILC) algorithm is designed to shape the EDV of a pathological ventricle. In addition, further constraints such as for example a uniform filling of the ventricle and the prevention of pumping during the systole are considered. A simplified model of the systemic circulation and the pump is used to study the system response to changes in pulmonary vein pressure and to benchmark controller performance. The results show that the algorithm obtains an excellent tracking performance and prevents the dilation of the ventricle. The approach offers the physician the opportunity to control multiple physiological variables even though the optimal control output trajectory is not known. Future work will focus on the improvement of the controller performance regarding the rejection of instantaneous disturbances and the incorporation of variable cycle duration.

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1. INTRODUCTION

The human heart generates hydraulic power with a combination of muscular contraction and unidirectional valves to provide blood perfusion throughout the body. During diastole largely passive filling of the left ventricle (LV) is followed by a small atrial contraction before the inlet valve (mitral valve MV) closes. The subsequent ventricular contraction during the systole begins with an isovolumetric phase of rising ventricular pressure $P_{LV}$ until it exceeds the aortic pressure $P_{Ao}$ and the outlet valve (aortic valve AV) opens. As the muscular contraction weakens, the AV closes and the pressure decreases isovolumically. Finally, the MV opens as soon as $P_{LV}$ decreases below $P_{LA}$ and the process begins anew.

If a heart suffers terminal congestive heart failure, physiological perfusion of the body can not be maintained. The gold standard therapy in such a case would be the implantation of a donor heart, but due to its limited ability the usage of rotary blood pumps even for long term treatment increases as stated by Cheng and Slaughter (2014). As insufficiency predominantly afflicts the left ventricle, the pump is usually connected to move blood from the left ventricle to the aorta (Ao), i.e. in parallel to the aortic valve, and is thus called left ventricular assist device (LVAD). LVADs are typically applied with a constant rotary speed setting (CS) and thus not adaptive to changes in the hemodynamic conditions, e.g. an increased blood flow demand during exercise. Due to Salamousen et al. (2011) this might lead to underpumping, ventricular suction or backflow of blood from the Ao in the LV.

Various different control strategies for LVADS exists, e.g. physiological, fuzzy logic and optimal control algorithms, as stated by AlOmari et al. (2012). Many physiological control strategies for example aim at imitating the Frank-Starling mechanism by maintaining a linear relationship between mean flow and flow pulsatility, see Gaddum et al. (2014). While Ochsner et al. (2014) adapt the LVAD hydraulic power to the EDV of the left ventricle, Schrödel et al. (2016) regulate the LVAD angular velocity to obtain a fixed ratio between hydraulic and ventricular power. Most of these physiological control strategies have a very simple structure, but they only follow a single control objective. Due to the nonphysiological responses of LVADS to changes in preload, several control algorithms aim to detect suction or avoid abnormal pumping states in general, e.g. see Saito et al. (2010). Ferreira et al. (2009) combined such an algorithm with a fuzzy logic controller to provide the required flow and pressure perfusion and avoid suction. This control algorithm has the advantage that no model of the cardiovascular system (CVS) is required, but there is no Q possibility to weight objectives individually.

Boston et al. (1998) developed a controller to minimize a multi-objective penalty function, while AlOmari et al. (2011) used a model predictive control approach to track the variations in mean pulsatile flow and pump rotational
speed. Due to AlOmari et al. (2012) the main disadvantage of these two approaches is that they both require accurate predetermined mathematical models. Chang et al. (2011) designed a model-free adaptive controller regarding the heart rate based on previous measurements. Iterative learning control (ILC) algorithms are also based on the idea to use information from the previous cycle to design the control input for the current one. According to this, they take advantage of the repetitive nature of the CVS and require only minor model knowledge. Therefore, they are very robust against model uncertainties and slowly changing repetitive disturbances. Apart from that, it is possible to consider different objectives and weight them regarding their importance. Walter et al. (2015) use an ILC algorithm in combination with a PID-controller in order to calculate the required motor current to follow a desired flow trajectory, while Rüschen et al. (2017) use the same controller structure to minimize the left ventricular stroke work.

Addressing the aforementioned advantages of ILC algorithms an approach for left ventricular assist devices using a norm-optimal iterative learning control (NOILC) algorithm is presented hereafter. The end-diastolic volume (EDV) is a determining factor of systolic pressure development during contraction and influenced by as well venous return as diastolic function. According to this, the aim of this paper is to examine if it is possible to use an NOILC algorithm in order to control EDV. Additionally a uniform filling of the ventricle shall be ensured to generate a filling process as physiological as possible, while the LVAD removes blood from it. Furthermore, severe pump speed changes within one heartbeat and suction shall be prevented. Therefore, ventricular volume \( V_{LV}(t) \) is chosen as control variable. Based on its behavior, the controller adapts the angular velocity \( w(t) \), which causes a certain LVAD flow \( Q_{VAD}(t) \).

This paper is structured as follows. Section 2 deals with the model of the cariovascular system and the pump used for preliminary testing. Furthermore, the cost function considering the different control objectives and the corresponding constraints are explained. The simulation results are described and analyzed in Section 3. Due to the fact, that the target of this publication is proof of concept, one simulation experiment is chosen to benchmark controller performance, while the second experiment is physiologically motivated. The conclusions are drawn in the final section.

2. METHODS

2.1 Cardiovascular and Pump Model

For preliminary LVAD testing a simplified model of the systemic circulation and the pump are used. The circulation model is a combination of a model to imitate the heart functionality based on the model of Leaning et al. (1983), a model of the systemic circulation similar to the model of Toy et al. (1985) and a model of the baroreceptor reflex of Colacino et al. (2007). For more details regarding the simulation model and the parametrization, see Schrödel et al. (2016). The following description of the circulation model closely follows the references above with minor alterations and additions for clarification.

The pulmonary vein (PV) pressure \( P_{PV}(t) \) is set to provide potential for the flow

\[
Q_{PV}(t) = \frac{P_{PV}(t) - P_{LA}(t)}{R_{PV}}
\]

across the resistance \( R_{PV} \) into the LA with the pressure \( P_{LA}(t) \). Blood exits the heart through both the AV and the LVAD outlet. In the Ao the flow is divided into a component \( Q_{AV}(t) \) describing the aortic windkessel (AW) effect and a component \( Q_{Ao}(t) \) that passes through the Ao, such that

\[
Q_{Ao}(t) + Q_{AV}(t) = Q_{AV}(t) + Q_{VAD}(t),
\]

where \( Q_{AV}(t) \) is defined as

\[
Q_{AV}(t) = P_{AW}(t)C_{AW},
\]

with the compliance \( C_{AW} \). The pressure in the Ao is calculated from the AW pressure \( P_{AW}(t) \) and the AW pressure drop \( \Delta P_{AW}(t) \) across the resistance \( R_{AW} \), i.e.

\[
P_{Ao}(t) = \frac{V_{AW}(t)}{C_{AW}} + R_{AW}Q_{AW}(t).
\]

The pressure difference between the aortic and the splanchnic (Sp) pressure

\[
P_{Sp}(t) = \frac{V_{Sp}(t)}{C_{Sp}},
\]

calculated by Sp volume \( V_{Sp} \) and Sp compliance \( C_{Sp} \), across the aortic inductance \( L_{Ao} \) yields to

\[
\dot{Q}_{Ao}(t) = \frac{1}{L_{Ao}}(P_{Ao}(t) - P_{Sp}(t)).
\]

Central nervous auto-regulation of aortic pressure with the aortic pressure sensor \( P_{CNS}(t) \) is approximated by a first order system, i.e.

\[
P_{CNS}(t) = \frac{P_{Ao}(t) - P_{CNS}(t)}{\tau_{CNS}},
\]

where \( \tau_{CNS} \) is the time constant. The control output in the CNS feedback loop is the time-varying resistance

\[
\Delta R_{Sp}(t) = k_{CNS}(P_{CNS}(t) - P_{Ao,0}(t))
\]

which uses the gain \( k_{CNS} \) to achieve the aortic pressure set point \( P_{Ao,0}(t) \).

Pressures in the LA and LV are calculated by

\[
P_{LA}(t) = E_{LA}(t)(V_{LA}(t) - V_{u,LA})
\]

and

\[
P_{LV}(t) = E_{LV}(t)(V_{LV}(t) - V_{u,LV}),
\]

where \( V_{LA} \) and \( V_{LV} \) are the volumes in the left atrium and ventricle and \( V_{u,LA} \) and \( V_{u,LV} \) the corresponding unstressed volumes at zero pressure. While the MV and AV ensure unidirectional flow, pumping is modeled with the time-varying elastances

\[
E_{LA}(t) = E_{dia,LA} + \varphi_{LA}(t)(E_{sys,LA} - E_{dia,LA})
\]

and

\[
E_{LV}(t) = E_{dia,LV} + \varphi_{LV}(t)(rE_{max}(t) - E_{dia,LV}),
\]

that use diastolic \( E_{dia,LA} \) and \( E_{dia,LV} \) and systolic \( E_{sys,LA} \) and \( E_{max} \) elastance parameters of LA as well as LV. The parameter \( E_{max} \) serves as a boundary condition and the parameter \( r \) is varied between \( 0 < r < 1 \) to model a pathological heart, i.e. with LVAD support. A healthy heart yields \( r = 1 \). The activation functions \( \varphi_{LA} \) and \( \varphi_{LV} \) are either set to a positive sine with amplitude 1 to represent systole or to 0 for diastole to model cardiac
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