Modeling and Experimental Identification of Peritoneal Cavity Pressure Dynamics During Oxygenated Perfluorocarbon Perfusion

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***Abstract*— This paper examines the problem of modeling the dynamics of the filling, drainage, and pressurization of the peritoneal cavity of a laboratory animal during perfusion. The paper is motivated by the potential of the peritoneal perfusion of an oxygenated perfluorocarbon (PFC) to provide a pathway for gas exchange in patients suffering from respiratory failure. Modeling cavity mechanics is important for avoiding excessive intracavity pressures that could potentially cause abdominal compartment syndrome during perfusion. Previous research in the literature examines elastic cavity behavior, but the problem of experimentally identifying models that couple this behavior with suction-assisted discharge remains relatively unexplored. Towards this goal, we performed large animal (namely, swine) experiments where we measured variables including peritoneal intracavity pressure, suction pressure, and PFC inflow. A simple state-space model fits data from the above experiment well. This model helps elucidate important preliminary insights into: *(i)* the role of active suction in facilitating discharge, *(ii)* the stiffening of the peritoneal cavity with perfusion, *(iii)* the linearity of cavity discharge behavior, *(iv)* the potential need to examine the impact of paralytics on cavity pressure dynamics as future work.**

I. INTRODUCTION

This paper examines the dynamic impact of the filling of the peritoneal cavity of a large animal (swine) with an oxygenated perfluorocarbon (PFC) on the animal's intra-abdominal cavity pressure. The paper is motivated by two main applications:

The first motivating application is **peritoneal dialysis** [1]. With this technique, a hyperosmotic solution, deficient in the ions and compounds to be removed from the bloodstream, is instilled into the abdominal cavity and allowed to dwell. The osmotic gradient then drives the transport of electrolytes and waste products across the peritoneal capillary walls, from the bloodstream into the dialysis fluid. In essence, peritoneal dialysis uses the lining of the abdominal cavity like a supplemental kidney.

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The second motivating application is the **peritoneal perfusion of oxygenated perfluorocarbons**. Perfluorocarbons (PFCs) are organic compounds containing carbon and fluorine. Typically nearly twice as dense as water, these compounds offer an extraordinary ability to dissolve both oxygen and carbon dioxide [2], [3], [4], [5]. Existing research in the literature shows that both the peritoneal and enteral perfusion of an oxygenated PFC (or alternative oxygen carrier) through the abdomens of laboratory rabbits, rats, and pigs provides a potential pathway for the diffusion-based transport of oxygen into these animals' bloodstreams [6]. This allows the abdomen to serve as a "third lung", in a manner similar to its use as a "third kidney" in peritoneal dialysis. The "third lung" concept is quite appealing because it provides a potential extra-pulmonary treatment modality for patients with respiratory failure. This is particularly valuable given the prevalence of different causes of respiratory failure, including acute respiratory distress syndrome (ARDS) induced by COVID-19, etc.

Care must be taken to effectively manage intra-abdominal pressure (IAP) in both peritoneal dialysis and potential future "third lung" interventions. Intra-abdominal hypertension (IAH) occurs at IAPs greater than or equal to $12mmHg$ [7]. While pressures exceeding this range may be well-tolerated, as may be the case in pregnant or obese patients, there is ultimately a point at which injury can occur. Specifically, this happens when, in a particular individual, the IAP is elevated to the point where it impedes venous return in the abdomen [8]. If this degree of IAP persists for a significant period of time, it can lead to abdominal compartment syndrome (ACS) [9], [10]. Measuring, characterizing, understanding, and fully reporting the prevalence of IAH and its dynamic dependence on intra-abdominal volume (IAV)/IAP is therefore extremely important for critical care applications [8], [9].

The literature already examines the relationship between IAP and IAV in applications such as abdominal paracentesis, laparoscopic pneumoperitoneum, and dialysis (e.g., [11], [12]). One topic examined by the literature is the characterization of elastic cavity behavior under the effect of different body habitus, patients' positions during perfusion, and respiration [13], [14], [15]. Some studies postulate an affine elastic relationship between IAV and IAP of the form $P = CV + B$, where P denotes cavity pressure, V denotes

perfused volume, C is the elasticity of the peritoneal cavity (a positive constant), and B is the baseline pressure inside the cavity [13], [16], [17], [18]. More recent studies: *(i)* argue that the above affine relationship is only accurate at lower IAP values (e.g., IAP $< 15mmHg$) and *(ii)* postulate an exponential cavity stiffening law for higher IAPs [9], [19]. These more recent studies describe cavity stretching and pressurization as going through three phases with increasing perfused volume, namely: a linear reshaping phase, a curvilinear stretching phase, and an exponential pressurization phase. Consequently, after a critical point, even a small change in perfused volume can make a drastic difference in IAP. This is consistent with our findings in this paper, where cavity stiffening occurs at higher perfused volumes.

The above literature provides an important foundation for the current work by highlighting key factors contributing to IAP, such as cavity stiffening with increasing perfused volume. However, the focus of this literature is predominantly on **static** relationships between perfused volume and cavity pressure. To model cavity pressure **dynamics**, one needs to create a coupled model of elastic cavity behavior, cavity discharge behavior, and the interplay between these behaviors. This is particularly important in scenarios where active suction (using, say, a vacuum pump) is used for assisting cavity drainage. To the best of the authors' knowledge, the problem of experimentally identifying dynamic models that couple elastic cavity behavior with suction-assisted discharge is relatively unexplored. The overarching goal of this paper is to address this gap through a combination of a perfusion experiment on a laboratory animal (namely, an adult swine) and subsequent model identification. Towards this goal, we present an experiment on a laboratory swine where oxygenated PFC was perfused through the animal's peritoneal cavity and discharged using active suction. The animal's abdominal pressure, bladder pressure, suction pressure, and PFC inflow were measured. The goal of this paper is to identify a mathematical model of the peritoneal cavity pressure dynamics using a simple state-space model, based on the above data.

The remainder of this paper is organized as follows. Section II describes the setup used for perfusion experiments. Section III presents the state-space model used for representing cavity dynamics. Section IV presents the approach used for model identification, the results of this identification effort, and the insights gathered from these results. Finally, Section V summarizes the paper's conclusions.

II. EXPERIMENTAL SETUP AND EXPERIMENTS

Fig. 1 provides a high-level schematic of the experimental setup used in this work. A companion paper describes this setup in more detail, focusing on its data acquisition and control capabilities [20]. The setup uses a peristaltic pump to perfuse perfluorodecalin, a widely-studied PFC, through a laboratory pig's abdominal cavity. Fluid enters and leaves the abdominal cavity through 36-French (i.e., $12mm$ diameter) venous cannulas terminating in custom-designed, 3D-printed, foam-covered plastic diffusers. A flow sensor is

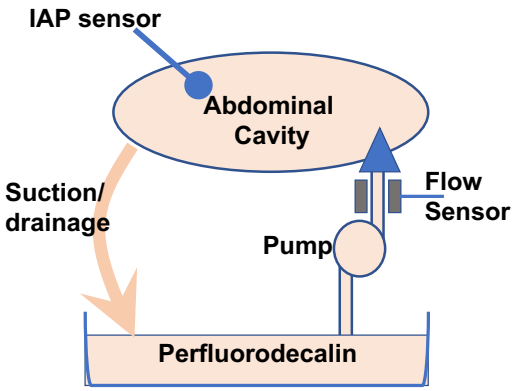

Fig. 1. Perfusion Setup

used for measuring the volumetric flowrate of PFC into the test animal. Fluid is drained from the animal using active suction from a manually-controlled vacuum pump. Finally, four different pressures relevant to this paper are measured:

1) Cavity pressure (measured via a catheter).
2) Bladder pressure (also measured via a catheter).
3) Suction pressure.
4) A "proximal pressure" in the PFC flow almost immediately downstream of the perfusion pump.

Four Institutional Animal Care and Use Committee (IACUC)-approved experiments have been conducted to date using the above setup. Collectively, the intent of these experiments is to provide insights into animal cavity pressure dynamics as well as the efficacy of the "third lung" concept in supplementing gas exchange by the lungs. Only the fourth animal experiment involved the direct measurement of the suction pressure used for PFC drainage, together with the use of foam-covered diffusers for perfusion. With this in mind, the focus of this paper is on taking a "deep dive" to characterize the cavity pressure dynamics seen specifically in this fourth experiment. Essentially, the goal of the paper is to postulate and parameterize a simple state-space model whose input variables are the perfusion flowrate and active suction pressure, and whose output is intra-abdominal pressure, or IAP. A neat (i.e., pure) mix of cis- and trans-perfluorodecalin was used in the experiment considered in this paper, as opposed to an emulsified PFC, in recognition of the degree to which PFC emulsification is typically associated with its intravascular applications. An adult yorkshire swine was used. The animal was anesthetized for the full duration of this animal experiment. Moreover, a paralytic was administered to the animal partway through the experiment. Only the animal test data gathered prior to the administration of the paralytic are used for this paper's model identification efforts, in recognition of the potential interplay between the paralytic and cavity pressure dynamics.

III. PROPOSED CAVITY PRESSURE MODEL

This section proposes a simple state-space model of cavity pressure dynamics. The model has two control inputs,

namely: *(i)* the volumetric flowrate $Q_{in}(t)$ of PFC measured by the perfusion flowrate sensor (in liters per minute), and *(ii)* the active suction pressure $P_\infty(t)$ applied by the vacuum pump used for assisting fluid drainage, and measured at the suction canister (in $mmHg$). Both of these inputs can be adjusted by the perfusion setup's user as a function of time, either using a data acquisition and control interface (in the case of perfusate flowrate) or manually (in the case of suction pressure). All pressure measurements presented in this paper reflect gauge pressures, meaning that a pressure reading of zero reflects atmospheric conditions. We propose the following state-space model of cavity dynamics:

$$\begin{aligned}
\frac{dQ_f(t)}{dt} &= \mu_f(Q_{in}(t) - Q_f(t)) \\
\frac{dP_f(t)}{dt} &= \mu_p(P_\infty(t) - P_f(t)) \\
\frac{dV(t)}{dt} &= Q_f(t) - Q_{out}(t) = Q_f(t) - C_o\delta P(t) \\
P(t) &= \beta_1 V(t) + \beta_2 V(t)^2 + \beta_3 V(t)^3 \\
\delta P(t) &= P(t) - C_\infty P_f(t) \\
V(0) &= V_o
\end{aligned} \tag{1}$$

In the above state-space model, $P(t)$ represents peritoneal intra-cavity pressure, in $mmHg$. This pressure can potentially be nonzero when the perfused fluid volume is zero, reflecting the fact that the peritoneal cavity can exhibit a slight positive gauge pressure even prior to perfusion. We account for this fact by creating a state variable $V(t)$, in liters, whose rate of change equals the rate of change of cavity volume but whose initial value at the onset of perfusion equals some constant, V_o, that we allow being nonzero. One implication of this modeling approach is that the relationship between $V(t)$ and $P(t)$ can pass through the origin while still allowing for a nonzero cavity pressure at zero perfused volume. Another implication is that the true perfused volume is equal to $V(t)-V_o$. Hence, the characteristic curve showing the relationship between $P(t)$ and $V(t)$ is a shifted cavity characteristic curve, with the magnitude of the shift, V_o, selected such that the new $P-V$ curve passes through the origin. Intuitively, therefore, the constant V_o represents a potentially nonzero fictitious "initial volume" of PFC that allows intracavity pressure to be nonzero prior to perfusion. The resulting model uses a cubic relationship between the shifted volume $V(t)$, and cavity pressure, $P(t)$. By adjusting the coefficients, β_1, β_2, β_3, of this relationship, one can obtain either stiffening or softening cavity pressure-volume characteristics.

Both of the above model's inputs, $Q_{in}(t)$ and $P_\infty(t)$, pass through first-order low-pass filters prior to impacting cavity pressure dynamics. These filters are partly intended as approximate representations of various delays within the perfusion setup. For example, the volumetric flowrate provided by the perfusion pump may not be immediately seen by the peritoneal cavity due to the length and compliance of the setup's perfusion tubing. The continuous-time eigenvalues associated with these first-order filters are denoted by $-\mu_f$ and $-\mu_p$ for the flowrate-related and suction pressure-related filters, respectively. Furthermore, the resulting filtered input flowrate and filtered suction pressure are denoted by $Q_f(t)$ and $P_f(t)$, respectively. Air leakage within the setup may lessen the impact of active suction. We account for this by multiplying filtered suction pressure by a factor C_∞ when computing the drainage pressure difference, $\delta P(t)$, seen by the outlet cannula. The volumetric flowrate through this outlet cannula, $Q_{out}(t)$ is assumed to equal some linear discharge constant, C_o, times $\delta P(t)$. Finally, the rate of change of perfused volume equals the difference between the filtered inflow rate, $Q_f(t)$, and the outflow rate $Q_{out}(t)$. The main goal of this paper is to identify the parameters of the above model from perfusion data, as shown next.

IV. SYSTEM IDENTIFICATION: APPROACH, RESULTS, AND INSIGHTS

This section fits the proposed state-space pressure dynamics model to approximately 600 seconds of experimental perfusion data. The state-space model in Eq. (1) is simulated with a time step $\delta t = 10$ seconds, assuming the initial condition $V(t) = V_o$. Optimization is used for estimating the parameters V_o, β_1, β_2, β_3, C_∞, C_o, and the discrete-time eigenvalues, $-\gamma_p$ and $-\gamma_f$, associated with an exact discretization of the first-order filters corresponding to the continuous-time eigenvalues $-\mu_p$ and $-\mu_f$, respectively. The optimization objective is to minimize the summation of the squared differences between predicted and measured cavity pressures over the perfusion time horizon, subject to the proposed cavity dynamics model as a constraint. This is a nonlinear, non-convex system identification problem, owing to the nonlinearity of the relationship between cavity pressure and volume and the resulting nonlinearity of the model's state equations. We solve this problem using a particle swarm optimizer and report the results here.

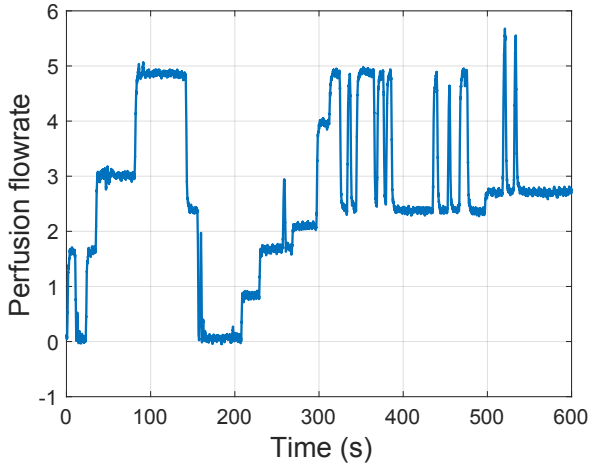

Fig. 2. Experimental perfusion flowrate (liters per minute)

Fig. 2 and Fig. 3 show the PFC perfusion flowrate and suction pressure used in this system identification study, respectively. Different perfusion flowrates are applied, ranging from

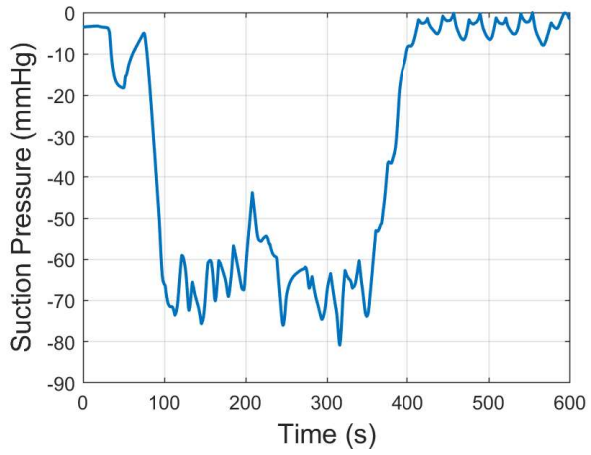

Fig. 3. Suction pressure

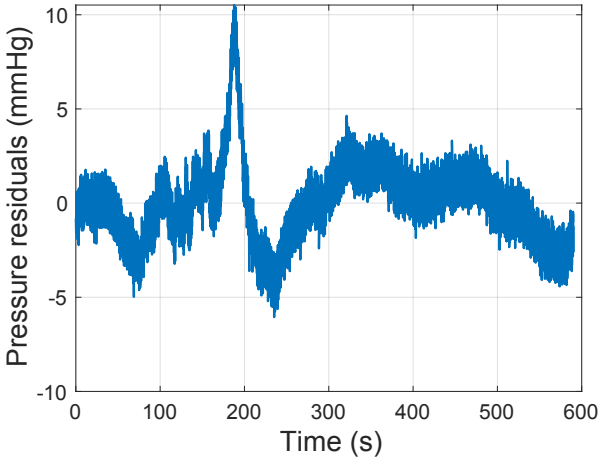

Fig. 5. Cavity pressure prediction residuals

0 to almost 6 liters per minute, and suction is used for fluid drainage roughly between the 100- and 400-second marks. The end result is a cavity pressure profile that rises rapidly due to perfusion, then drops rapidly due to reduced perfusion plus increasing suction, then finally converges to a slowly-rising, mid-range value. Fig. 4 compares the measured versus predicted cavity pressure profiles for this time window. A good match is achieved, with Fig. 5 showing small residual pressure prediction errors (defined as measured pressure minus predicted pressure), except during periods of very rapid pressure decline. A histogram of the pressure prediction residuals (Fig. 6) confirms that the residuals are, indeed, mostly small, with the exception of a "long tail" on the positive side of the histogram reflecting the incorrect prediction of pressure by 5-10$mmHg$ during transients. Moreover, a plot of the auto-correlation of the residuals (Fig. 7), for a 10-second time lag, suggests that while these residuals are not independent, identically distributed (iid), the unmodeled dynamics contained within these residuals are fairly small in impact.

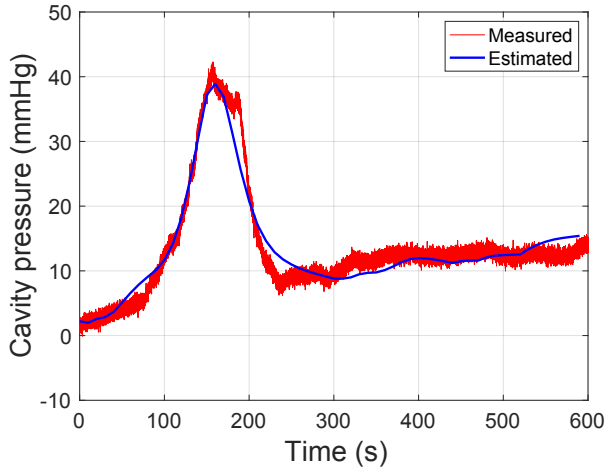

Fig. 4. Cavity pressure prediction

The parameter values associated with the above curve fit are $C_\infty = 1$, $V_o = 0.2044L$, $C_o = 0.0733(L/min)/mmHg$, $\beta_1 = 11.897mmHg/L$, $\beta_2 = -5.526mmHg/(L^2)$, $\beta_3 = 1.193mmHg/(L^3)$, $\gamma_f = 0.3101$, and $\gamma_p = 0.0228$ (for a 10-second sampling time). All of these values are interior optima, except for $C_\infty = 1$, which is boundary-optimal (where the simple bounds $0 \leq C_\infty \leq 1$ were imposed on the particle swarm optimizer). As shown in Fig. 8, these parameters correspond to a linear cavity discharge law, i.e., a discharge law that emphasizes linear viscous pressure drops over more traditional orifice discharge characteristics. A plot of the difference between the "proximal" pressure and cavity pressure versus PFC perfusion flowrate, omitted for brevity, shows that such a discharge law is indeed accurate for PFC *inflow*, thereby providing some support to the use of this law for modeling PFC *outflow*. Fig. 9 analyzes the estimated parameter values further by plotting the corresponding cavity pressure-volume characteristic curve. As expected from the literature, the cavity acts as an initially linear spring that becomes slightly more compliant then eventually much stiffer as perfused volume increases. The estimated value of V_o, together with this characteristic curve, implies that when the perfused volume is zero, abdominal cavity pressure sits at a reasonably small value of $2.2mmHg$ relative to atmospheric. Finally, the estimated values of γ_p, γ_f, and C_∞, collectively, imply that while the peritoneal cavity feels the full impact of both control input variables in steady-state, the transients associated with suction may potentially be much slower. Future research is needed to explain this observation, one possibility being a model fitting error, and another possibility being that slow tissue mechanics may play a significant role in allowing suction to achieve its maximum impact on PFC drainage.

One challenge associated with the above model is the fact that it over-predicts pressure dynamics after the 600s mark by a substantial margin, as shown in Fig. 10. This over-prediction cannot be merely attributed to the fundamental difference between model *fitting* versus model *validation*. In

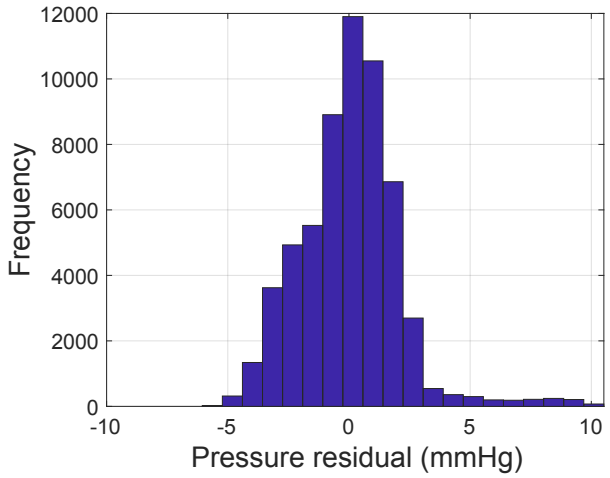

Fig. 6. Histogram of cavity pressure prediction residuals

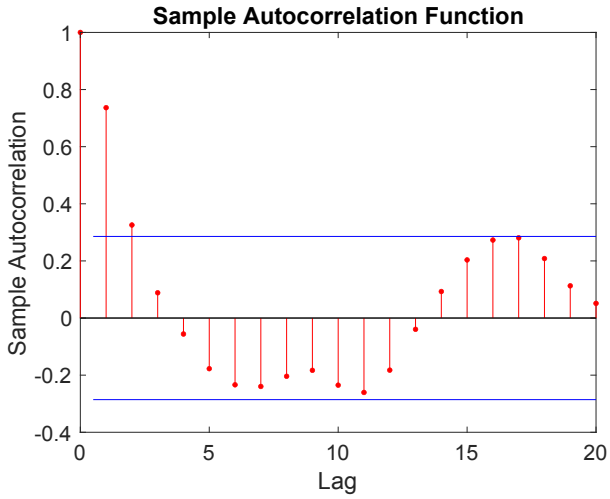

Fig. 7. Auto-correlation of cavity pressure residuals

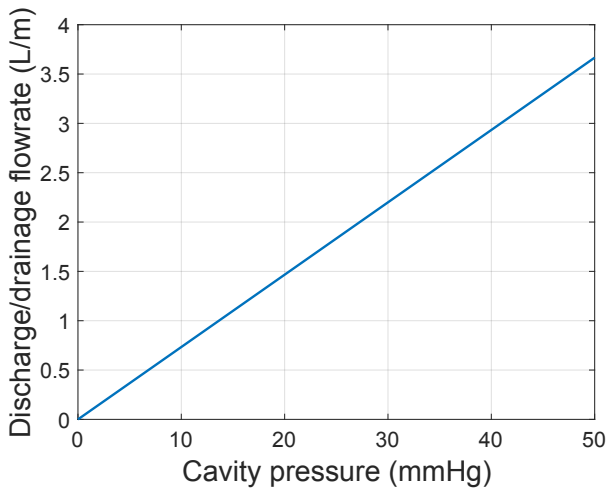

Fig. 8. Estimated cavity discharge characteristics

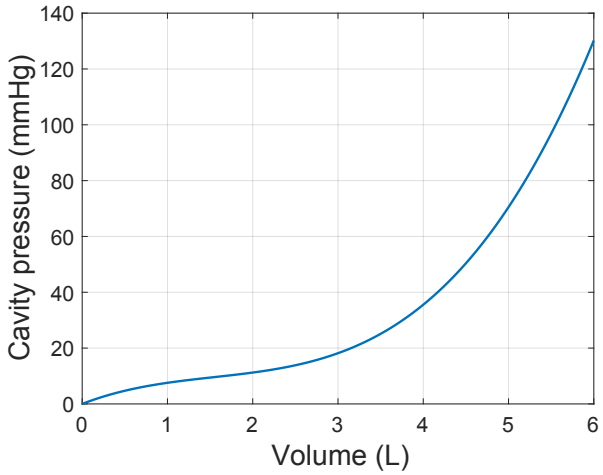

Fig. 9. Estimated cavity pressure-volume characteristics

fact, attempting to fit the proposed model to the full data range in Fig. 10 results in a much poorer fit for the 0-600s range, together with some improvement in fitting the 600s-1000s range. This suggests that the cavity pressure dynamics are fundamentally different during these two ranges of time. An examination of the animal experiment records reveals that a paralytic was administered to the animal, for the first time, approximately around the 600s mark. A significant drop in measured cavity pressure compared to our model's predictions is a potentially plausible consequence of the administration of paralytics. This suggests one important pathway for future work, namely, the modeling of the impact of paralytics on cavity pressure dynamics. Fig. 11 highlights another important pathway for future work by showing a model-based prediction of the perfused volume of PFC (inside the peritoneal cavity) as a function of time, for the first 600 seconds of perfusion. This is a signal that can potentially be measured versus time (say, by measuring the PFC fluid levels in the perfusion setup's various canisters), thereby providing an additional pathway for model validation beyond what is included in this paper.

V. CONCLUSIONS

This paper models the dynamics of peritoneal intra-cavity pressure in a laboratory pig, and fits this model to experimental perfusion data. A good fit is obtained for experimental data prior to the administration of a paralytic. This curve fit highlights the linearity of cavity discharge versus pressure, as well as the stiffening of the cavity with increasing perfused volume. Future work will validate this model further using additional experimental data, in recognition of the importance of ensuring the validity of the model for multiple perfusion events per animal and/or multiple animals. One important area of potential emphasis in future work is the direct measurement of fluid volume in the perfusion setup as a validation signal, and another area of emphasis is the experimentally-validated modeling of the impact of paralytics on cavity pressure dynamics.

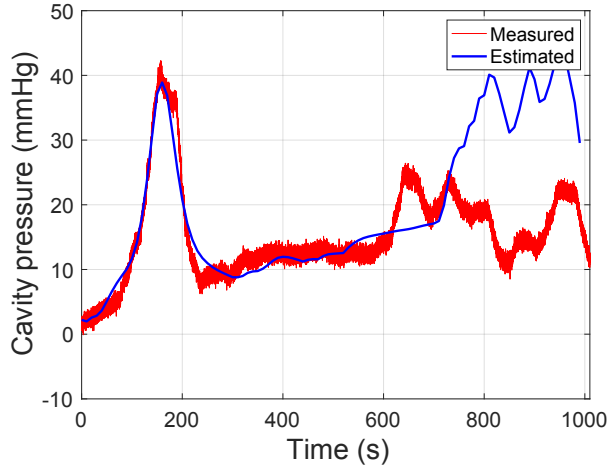

Fig. 10. Predicting cavity pressures over longer time horizons

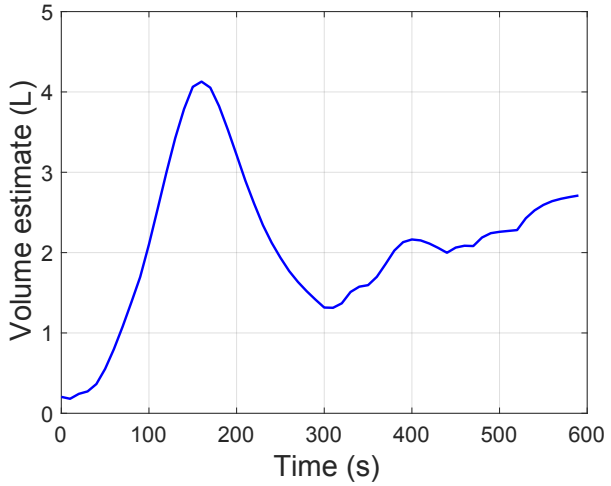

Fig. 11. Estimated perfused PFC volume

ACKNOWLEDGMENTS

The research in this paper was conducted under IACUC #0121006 at The University of Maryland Medical School, Baltimore, MD (UMB). Support for this research was provided by the Mechanical Engineering Department at The University of Maryland (UMD), UMD startup funding for Dr. Hosam Fathy, an internal grant from The University of Maryland Device Development Fund, a gift of 20 liters of PFC from Fluoromed, and a National Science Foundation (NSF) EAGER grant to Dr. Hosam Fathy, Dr. Joseph Friedberg, and Dr. Jin-Oh Hahn (NSF CMMI Award #2031251, #2031245). Any opinions, findings and conclusions, or recommendations expressed in this material are those of the author(s) and do not necessarily reflect those of NSF.

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