

# Detection of Movement toward Randomness by Applying the Block Decomposition Method to a Simple Model of the Circulatory System

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It is known that Shannon's information entropy, compressibility and algorithmic complexity quantify different local and global aspects of synthetic and biological data. Applicability of these concepts to objects closer to the "clinical end" is less studied. A possible approach consists of encoding the state of a human body system represented by its system dynamics model using matrices and monitoring the behavior of the system through analysis of these matrices, with a potential extrapolation of the results to the clinical setting. This paper presents an attempt at using some concepts and tools, specifically the block decomposition method (BDM), coming from the new emerging field of algorithmic information dynamics, for the management of a patient, especially in the intensive care unit (ICU). It describes some aspects pertaining to the "pre-clinical" incipient stage and tries to outline eventual future clinical application.

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*Keywords:* system dynamics; models; algorithmic information; algorithmic information dynamics; block decomposition method; critical illness; intensive care

## 1. Introduction

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The human body can be approached as a system of high complexity. From the organ systems the body is composed of down to a cell, this complexity persists, at least when assessed by conventional methods and tools.

A medical practitioner has to deal with this complexity and make the correct decisions concerning diagnosis, monitoring and treatment. Pretty often this should be done in a time-constrained environment with an abundance of data coming from monitors and directly from the patient. All this is especially valid for a critically ill patient, for

whom, despite the vast array of intensive care unit (ICU) data, the relevant information that would integrally characterize the patient's state is missing.

It would be more than welcome to have a tool or tools that can provide this kind of information, helping the clinician to make more effective decisions.

There have been many different attempts to address this problem, the more recent ones being connected with the advances in computational science and practice.

One promising possibility consists of building computer models able to simulate human physiological processes and "personalizing" them to a particular patient. Once achieved, this can help with a number of aspects pertaining to management of a real patient. An example would be simulating different treatment regimens before applying them to a patient.

An additional potential benefit of this "modeling approach" would serve as a step toward the elaboration of a toolkit able to integrally characterize the model state and behavior, with the possibility to extrapolate this information to a real patient.

The work presented in this paper is an endeavor to apply knowledge from the field of algorithmic information dynamics (AID) [1, 2] to a model of the cardiovascular system. The ultimate goal is to deliver more qualified care to the human patient by identifying aspects that can help better characterize the model state and its dynamics.

## 2. Cardiovascular System Model

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A critically ill patient can be seen as a dynamic system whose state can change rapidly and dramatically. Conventionally, it will be considered that the direction of these changes moves away from "order" (or homeostasis) toward "disorder" (or chaos/randomness).

Since it is difficult to approach the human body integrally in all its complexity, for the purpose of this work two "objects of interest" were established:

- An object of limited scale "to think about": namely the circulatory system present in the human body as one of the vital ones, depicted hereafter through a simple system dynamics model.
- A toolkit "to think with," represented by some concepts and tools found in the field of AID, especially the block decomposition method (BDM).

The model described here (and with more details in Appendix A) was recently created by the author of this work and has a number of

analogous models [3–5]. This is a minimal lumped-parameter system dynamics (SD) model of the cardiovascular system [6], with the focus on cardiac output (CO) and factors influencing CO.

The focus on CO as the main output of this model is conditioned by the importance of CO in functionally defining hypoxia as one of the landmarks as well as cornerstones of the so-called critical state. It is well known that many living organisms can survive without oxygen for a very limited time. This is why the amount of O<sub>2</sub> delivered to the tissues is so important for an intensivist while managing a critical case in the ICU, and it can be calculated with a simple formula, using data available in a modern ICU:

$$DO_2 = CO * 1.34 * Hb * S_a O_2$$

where DO<sub>2</sub> (L/min) is the amount of oxygen delivered to the tissues during one minute, CO (L/min) is the cardiac output or the volume of blood pumped by heart in one minute, 1.34 (ml of O<sub>2</sub>) is the amount of oxygen one gram of hemoglobin can bind, Hb (g/L) is the hemoglobin level, and S<sub>a</sub>O<sub>2</sub> is the arterial blood O<sub>2</sub> saturation.

As can be deduced from the formula, CO is of crucial importance, and in its turn is determined by a number of factors (i.e., preload, after load, myocardial contractility and compliance, heart rate and rhythm), many of them being routinely monitored in the ICU.

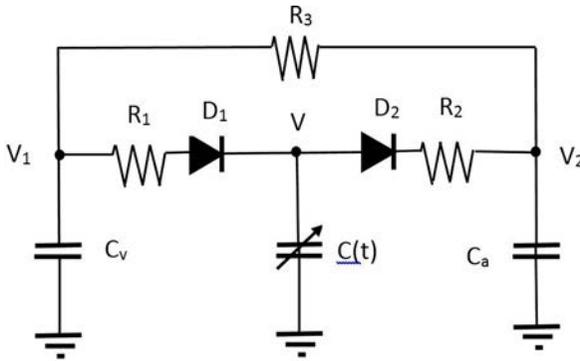
Behind the approach to the heart as “a pump,” from which the CO determinants are derived, there are simple hydraulic models backed up by the analogous metrics between the electrical domain and fluid dynamics, based on Ohm’s law ( $I = U / R$ ), where  $I$  (current A) is equivalent to flow rate (ml \* s<sup>-1</sup>),  $U$  (voltage V) is equivalent to fluid pressure (mmHg), and  $R$  (resistance Ohm) is equivalent to resistance of different segments of the circulation (mmHg \* s \* ml<sup>-1</sup>):  $Q$  (charge C)—volume,  $V$  (ml) and  $C$  (capacitance F)—compliance,  $C$  (ml \* mmHg<sup>-1</sup>). Elastance is the reciprocal of compliance ( $1 / C$ ).

The model hereafter referred to as the CVS model consists of three compartments: two passive ones for arterial and venous parts of the circulation and an active compartment for the left ventricle.

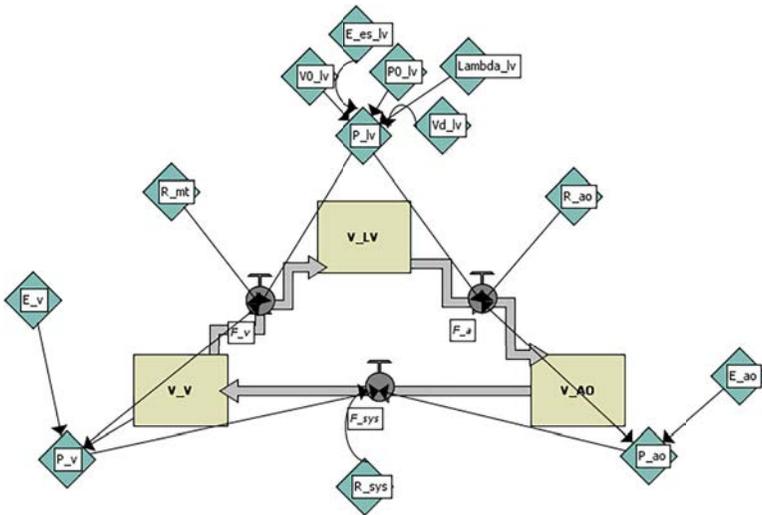
Figure 1 presents the electrical domain approach to the model.

Figure 2 depicts the system dynamics approach (by NetLogo System Dynamics Modeler [7], where several stocks and flows between them are presented) close to the hydraulic domain.

The model dynamics are governed by ordinary differential equations (ODE), which describe the dynamics and relation between three stocks (left ventricle volume, arterial segment and venous segment volumes) and their respective flows.



**Figure 1.**  $R_{1,2,3}$  denote the resistance for the three compartments: venous, left ventricle and arterial;  $V_{1,2,3}$  stand for volume;  $C_{a,v,(t)}$  denotes compliance;  $D$  represents the valves that ensure the unidirectional nature of the blood flow.



**Figure 2.**  $V_{LV}$ ,  $V_{AO}$  and  $V_V$  are stocks representing the left ventricle, arterial segment and venous segment, respectively.

The pulsatile nature of the flow is conditioned by a driver function that mimics the myocardial contractions and a Heaviside step function that simulates valvular mechanism, based on the “open on pressure, close on flow” principle. The types of the main equations used in the model are as follows:

$$Q_t = \frac{P_1 - P_2}{R} \tag{1}$$

$$\frac{dV}{dx} = Q_{in} - Q_{out} \tag{2}$$

$$P = E * V \tag{3}$$

$$P_{es}(V) = E_{es}(V - V_d) \tag{4}$$

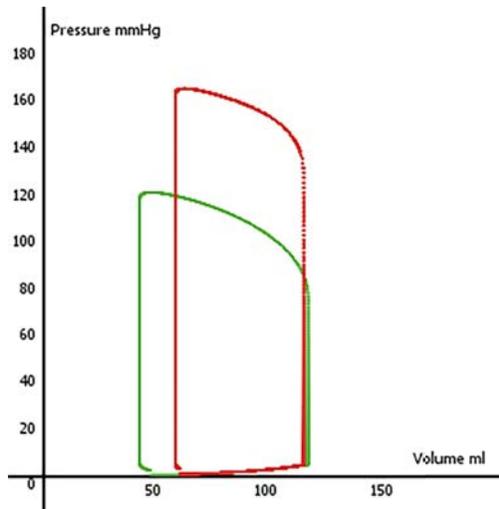
$$P_{ed}(V) = P_0(e^{\lambda(V-V_0)} - 1) \tag{5}$$

$$P(t) = E(t)(V(t) - V_d) \tag{6}$$

$$e(t) = \sum_{i=1}^N A_i e^{-B_1(t-C_i)^2} \tag{7}$$

Equations (1) through (3) are used for representation of the blood flow, variations in the volume and pressure. Equations (4) through (7) refer to the left ventricle as the active compartment, describing the same parameters as for the passive ones, as well as the time-varying elastance (*e*), which drives the model by mimicking myocardial contractility and contributing to the pulsatile nature of the blood flow. In Appendix A, there is a detailed description of these aspects.

The outputs of the model include stroke volume, CO and ejection fraction. Additionally, the model generates a pressure-volume loop (PVL), unique for a particular scenario/pathological state. Figure 3 depicts two examples of a “normal” PVL (green color) and in hypertension (red color). The PVL can provide valuable information for diagnosis, monitoring and treatment of a particular patient. The area inside PVL is considered to be indicative of myocardial oxygen



**Figure 3.** PVL (green—normal, red—hypertensive state).

demand [8], a parameter that cannot be directly assessed but can drastically influence the disease course, outcome and treatment.

For the current research, PVL provides the pressure parameter that is used along with the volume and CO in setting the arrays representing/encoding a particular behavior of the system/model.

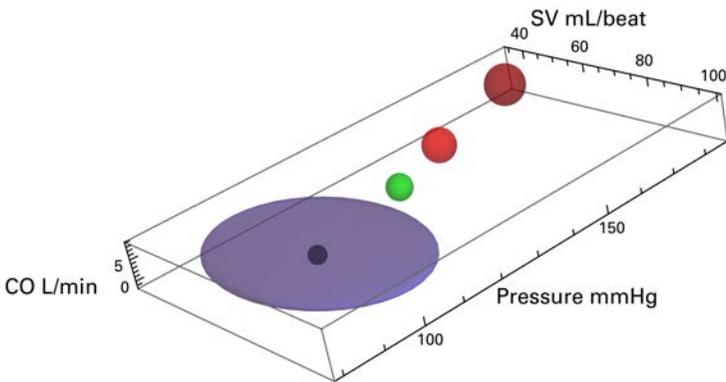
As stated previously, the model can visualize the influence of a number of factors (i.e., preload, afterload, myocardial contractility, heart rate, etc.) on CO (see [6]).

The effect of varying different model parameters can be observed in the resulting pressures and volumes (shown on the plot area of the model GUI [6]) and PVL, and last, in the left ventricle CO in normal and pathological conditions.

By setting model parameters to different values, it is possible to simulate a number of pathological states. For the purpose of this research, four such states are simulated and later analyzed using AID tools.

The development of the minimal model used in this research is based on a minimalist approach, where the model is kept as simple as possible unless the addition of complexity will result in a significant improvement in physiological accuracy. The basic building blocks of the model are the passive and active elastic chambers and the governing equations for flow between these chambers. The functions of the basic model building blocks are investigated individually before assembling these components to create a full closed-loop model. This approach ensures that the individual contributions of each component are known when analyzing the performance of the complete model.

Figure 4 shows the kinetics of the model in a virtual model phase space for the four states, which correspond to distinct clinical conditions and which are further analyzed.



**Figure 4.** The three-dimensional phase space for the CVS model: position of some states presented as spheres of different colors: normal (dark blue), pre-hypertension (green), hypertension (red), congestive heart failure with

hypertension (dark red). SV is the stroke volume, CO is the cardiac output. The blue ellipsoid shows the range that is considered normal. Its flat appearance is due to the difference in the metrics of axes ( $x$ ,  $y$ —volume (mL) and pressure (mmHg)—hundreds, versus units for  $z$ —CO (L/min)).

### 3. Algorithmic Information Dynamics

AID [9–11] is an emerging field of complexity science based on algorithmic information theory (AIT), which comprises the literature based on the concept of Kolmogorov–Chaitin complexity and related concepts such as algorithmic probability, compression, optimal inference, the universal distribution, Levin’s semi-measure and others.

Central to AIT is the definition of algorithmic (Kolmogorov–Chaitin or program-size) complexity (Kolmogorov, 1965; Chaitin, 1969) [10]:

$$K_T(s) = \min\{|p|, T(p) = s\},$$

that is, the length of the shortest program  $p$  that outputs the string  $s$  running on a universal Turing machine  $T$ .

AID strives to search for solutions to fundamental questions about causality: why a particular set of circumstances leads to a particular outcome. In this aspect it essentially differs from traditional statistics.

As an applied science, AID is a new type of discrete calculus based on computer programming and aimed at studying causation by generating mechanistic models to help find first principles of physical phenomena, building up the next generation of machine learning [2, 9].

In the AID toolkit, there is a special tool for providing reliable estimations to uncomputable functions, namely the online algorithmic complexity calculator (OACC) [2, 13], which provides estimations of algorithmic complexity and algorithmic probability for short and long strings and for two-dimensional arrays better than any other traditional tool, none of which can capture any algorithmic content beyond simple statistical patterns.

The OACC uses the BDM method [12, 13], which is based upon algorithmic probability defined by the coding theorem method (CTM) [14, 15]:

$$\text{BDM} = \sum_{i=1}^n \text{CTM}(\text{block}_i) + \log_2(|\text{block}_i|).$$

The OACC is available as an online version [2] as well as standalone packages in R [12] and a number of other languages, including the Wolfram Language [16], and it is used for respective calculations for the scope of the current work.

#### 4. Applying the Online Algorithmic Complexity Calculator to the CVS Model

In order to make datasets generated by the model when run under a certain scenario appropriate for OACC, the following mapping procedure was applied:

For each of the three model outputs mentioned previously (i.e., pressure, stroke volume and CO) a binary array with three cells was used, with a resulting  $3 \times 3$  matrix with rows for the outputs and columns for the respective output expression (deviation):

Pressure	0 / 1	0 / 1	0 / 1
Volume	0 / 1	0 / 1	0 / 1
Cardiac output	0 / 1	0 / 1	0 / 1

The deviation is denoted by “0” if it is in the physiological range and with “1” if it deviates from normal range. The first cell from left to right corresponds to “minimal” deviation, left and middle cell to “medium,” and all three cells filled with ones depict the maximal deviation of the respective output of the model. Thus the outputs within the physiological range will generate an all-zero matrix. Such matrices were generated for the four pathological states/scenarios in question.

The threshold values for establishing the degree of deviation are based on the accepted normal range [17, 18] of the respective parameter and the magnitude of the deviation from this range as shown in Table 1. The resulting outputs for selected scenarios/clinical states are presented in Table 2.

Model Output	Normal Range	Minimal Deviation	Medium Deviation	Maximal Deviation
Pressure (mmHg)	90–119	120–129 or 81–90	130–139 or 70–80	$\geq 140$ or $< 70$
Stroke volume (ml)	$\geq 70$	61 – 70	50 – 60	$< 50$
CO (L / min)	4.5–8.0	3.5–4.49	3.0–3.49	$< 3.0$

**Table 1.** CVS model outputs and the criteria for defining the magnitude of their deviation.

Model Output	Normal	pre-HT	s1-HT	s2-HT	CHF & HT
Pressure (mmHg)	119	125	135	145	100
Stroke volume (ml)	72.6	71.4	70.4	65.0	45.2
CO (L / min)	5.08	5.0	4.86	4.55	3.16

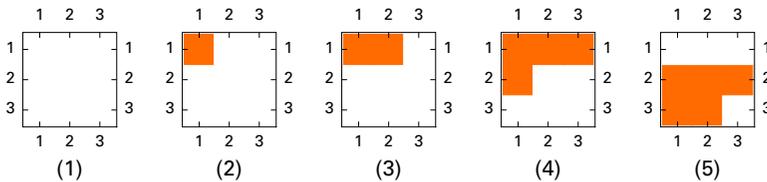
**Table 2.** CVS model output for certain scenarios/clinical states: normal, pre-hypertension (pre-HT), stage 1 hypertension (s1-HT), stage 2 hypertension (s2-HT) and congestive heart failure with hypertension (CHF&HT).

In this way, the matrix for the scenario corresponding to a clinical case with stage 1 hypertension will be:

$$\begin{matrix} 1 & 1 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{matrix}$$

Using data provided by the CVS model under the following scenarios: (1) normal, (2) pre-hypertension, (3) stage 1 hypertension, (4) stage 2 hypertension and (5) congestive heart failure with hypertension, the resulting matrices were generated and used for further analysis (see Figure 5).

Using the OACC, these arrays were analyzed with the results shown in Table 3.

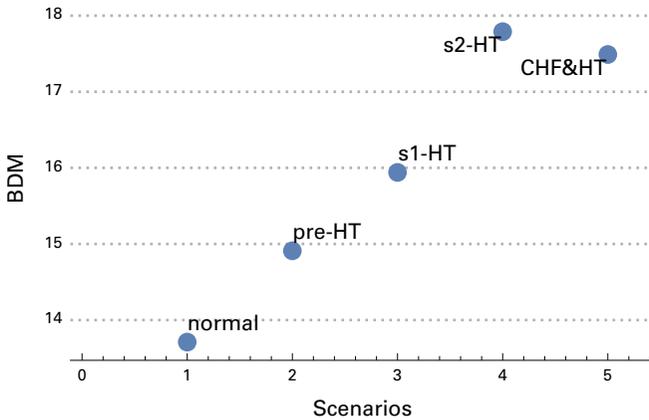


**Figure 5.** Matrices for the simulated scenarios: (1) normal, (2) pre-hypertension, (3) stage 1 hypertension, (4) stage 2 hypertension, (5) congestive heart failure with hypertension.

Model Scenario / Clinical Situation	BDM Algorithmic Complexity	Shannon Entropy	Compression Length (using gzip)
Normal	13.7134	1.5827	104
Pre-hypertension	14.9145	1.7639	104
Stage 1 hypertension	15.9416	1.8578	136
Stage 2 hypertension	17.7910	1.9395	144
Congestive heart failure and HT	17.4911	1.9395	136

**Table 3.** Results of applying three tools (BDM, Shannon entropy and compression length) to certain scenarios simulated by the CVS model.

The BDM seems to provide the most finely grained results among the three tools. Results for the BDM value for the simulated scenarios/clinical situations are presented in Figure 6 as a plot.



**Figure 6.** BDM value for CVS model scenarios: (1) normal, (2) pre-hypertension (pre-HT), (3) stage 1 hypertension (s1-HT), (4) stage 2 hypertension (s2-HT), (5) congestive heart failure with hypertension (CHF&HT).

Applying the approach for setting up the arrays described previously (i.e., sequentially replacing zeros in the original array that denote a normal physiological state with ones, depending on the magnitude of the deviation of a certain parameter) essentially diminishes the number of  $3 \times 3$  arrays that can be obtained by simple permutation. The final set will exclude the arrays where there is a 0 to the left of a 1.

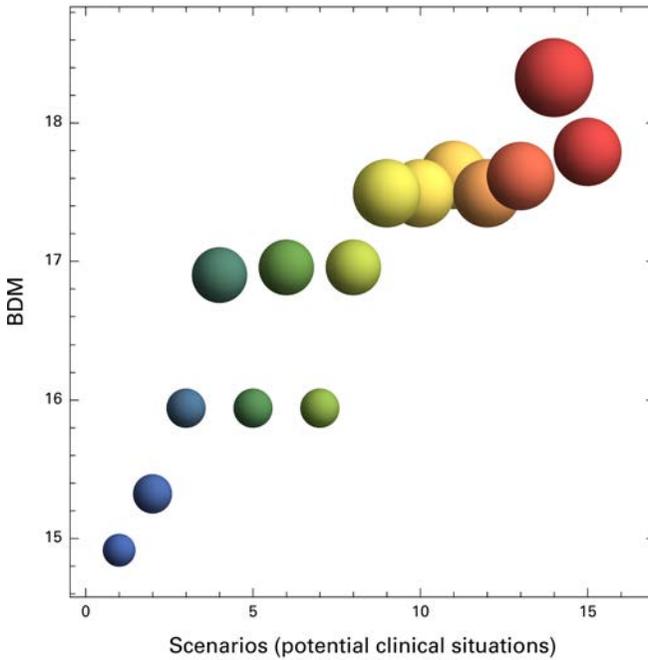
Calculating the BDM for this limited number of arrays (which can potentially describe distinct clinical situations) and plotting the values gives a general view of the “BDM value plane” for the particular CVS model, as shown in Figure 7. This is in line with Figure 4, where the clinical states simulated by the CVS model for the scope of this research are presented.

As denoted by Figure 6, the BDM value increases with the advancement of the hypertension state according to clinical criteria [18], and higher BDM values can be seen, depending on how far away from the normal range the system is.

## 5. Conclusions, Limitations and Future Work

Summarizing the given findings, it can be concluded that the block decomposition method (BDM) accurately highlights the dynamics of a particular model that simulates the progress of a clinical state, namely hypertension (HT), from the “normal range” to advanced disturbances (or from “order” to “disorder”). A similar but reversed path

can be seen in case of HT correction/treatment by manipulation of vascular resistance (e.g., decreasing it in a clinical setting with vasodilators).



**Figure 7.** BDM space of the CVS model.

It can be noticed that the BDM value goes slightly down in case of CHF associated with HT. This scenario is different from the “natural course” of HT and represents a combination of two underlying physiological mechanisms: increased vascular resistance (as in HT) and decreased myocardial contractility (as in CHF caused, for instance, by a myocardial infarction). It seems that in this more complex case, the way of encoding the behavior of the model applied in this research (i.e., by a  $3 \times 3$  array) is not able to address these “subtleties” and higher-dimension arrays may be required.

This paper presents a very general view of the problem and is limited mostly to the diagnostic/monitoring segment, where the BDM may be of value. Even a higher-value BDM might help in guiding the treatment when there is a need to manipulate a number of parameters (e.g., vascular resistance and myocardial contractility by vasodilators and inotropes, respectively), which would be the case when dealing with CHF and associated HT. These aspects need further investigation.

The main limitations of this study detected so far include:

- Coarse granularity of the CVS model. Although the model captures the main hemodynamic features of human circulation, there might be some details that will give slightly different results in case of more complex models. This can change details of the “path toward or away from randomness” in a particular case, with probably less influence on the “trend toward or away from randomness” itself.
- In this research, the model runs for every scenario with a fixed heart rate (HR) of 70 beats/min. Varying the HR will make the situation more complex, moreover with different values for volume status and vascular elastance. For the purpose of this research, in order to keep it simple, the HR was intentionally not altered. Including the HR, which directly influences the cardiac output (CO) ( $HR * SV = CO$ ) and/or other CO determinants, will require at least a higher-dimension array (i.e., at least four dimensions).
- A major limitation of the current paper is that the matrices are too small to believe they are capturing much content. Papers [11, 13] show that any matrix representation does approach the complexity of the automorphism group of that matrix. An increase in dimensionality of the matrix (e.g., by adding new parameters) may be expected to address this limitation and could serve as a potential path for further research.
- Results described in this paper are valid for this particular model, for the scenarios simulated by the model, and the way of setting up the arrays. Results for other models, scenarios or approaches may differ.

This research is a ramification of a larger project aiming at elaboration of a set of tools to facilitate decision-making in the intensive care unit (ICU), particularly in case of cardiovascular problems. For this, more sophisticated models that are to be “personalized” for a specific patient (this is done mainly by using traditional machine learning techniques) are being created and potentially used for simulations of a particular treatment regimen before being applied to a real patient. In this context, the BDM could serve as a valuable guiding tool by monitoring the cardiovascular system dynamics at an “integral” level. This is to be done with future work.

At this point, the focus will be on exploring the BDM “behavior” for a larger set of parameters and scenarios and selecting the most appropriate model parameters to be used for BDM estimation.

An even “more profound” analysis would be through more sophisticated approaches like causal deconvolution [2] and the algorithms based on minimal algorithmic information loss [19], which may be one of the avenues for future research.

**Appendix**

**A. Cardiovascular System Model**

The following is a description of a NetLogo System Dynamics (SD) model close to NetLogo standards. The model is available for download [6].

**How It Works**

This is an SD model consisting of three compartments: two passive ones for arterial and venous parts of the circulation and an active compartment for the left ventricle.

The model dynamics are governed by ordinary differential equations (ODE) that describe the dynamics and relation between three stocks (left ventricle volume, arterial segment and venous segment volumes) and respective flows.

The pulsatile nature of the flow is conditioned by a driver function that mimics the myocardial contractions and a Heaviside step function that simulates valvular mechanism, based on the “open on pressure, close on flow” principle.

By changing different parameters (i.e., volume status, vascular resistance, myocardial contractility, vascular elastance, etc.), effects can be observed on stroke volume (SV), ejection fraction (EF) and cardiac output (CO). With every set of parameters, respective pressures and volumes are plotted and a pressure-volume diagram is generated. This can help in understanding principles of CO physiology, particularly the influence of separate factors or their combination.

The ODE are as follows:

For left ventricle:

$$\frac{dV_{lv}}{dt} = \frac{(P_v - P_{lv}) k}{R_{mt}} - \frac{(P_{lv} - P_{ao}) k}{R_{ao}}. \tag{A.1}$$

For arterial segment (aorta):

$$\frac{dV_{ao}}{dt} = \frac{(P_{lv} - P_{ao}) k}{R_{ao}} - \frac{(P_{ao} - P_v) k}{R_{sys}}. \tag{A.2}$$

For venous segment:

$$\frac{dV_v}{dt} = \frac{(P_{ao} - P_v)}{R_{sys}} - \frac{(P_v - P_{lv}) k}{R_{mt}}. \tag{A.3}$$

Where:

$V_{lv}$  (ml) is the left ventricle volume,

$V_{ao}$  (ml) is the arterial segment (aorta) volume,

$V_v$  (ml) is the venous segment volume,

$P_v$  (mmHg) is the pressure in the venous segment,

$P_{lv}$  (mmHg) is the pressure in the left ventricle,

$P_{ao}$  (mmHg) is the pressure in the aorta,

$R_{mt}$  (mmHg\*s\*ml<sup>-1</sup>) is the mitral valve resistance,

$R_{ao}$  (mmHg\*s\*ml<sup>-1</sup>) is the aortic valve resistance,

$R_{sys}$  (mmHg\*s\*ml<sup>-1</sup>) is the systemic vascular resistance,

$k$  is a scalar used to account for some extracardiac regulatory influence.

The general concept of this model uses the analogous metrics between the electrical domain and fluid dynamics, based on Ohm's law ( $I = U/R$ ), where  $I$  (current A) is equivalent to flow rate (ml\*s<sup>-1</sup>),  $U$  (voltage V) is equivalent to fluid pressure (mmHg), and  $R$  (resistance Ohm) is equivalent to resistance of different segments of the circulation (mmHg\*s\*ml<sup>-1</sup>):  $Q$  (charge C)—volume,  $V$  (ml) and  $C$  (capacitance F)—compliance,  $C$  (ml\*mmHg<sup>-1</sup>). Elastance is the reciprocal of compliance ( $1/C$ ).

$$\text{Blood flow} = \frac{P_1 - P_2}{R}, \quad (\text{A.4})$$

where  $P_{1,2}$  are pressures in vicinity segments and  $R$  represents resistance to the flow at the respective segment's junction.

Pressure is calculated as the product of elastance ( $E$  mmHg\*ml<sup>-1</sup>) and volume ( $V$  ml):

$$\text{Arterial (aortic) pressure} = E_{ao} * V_{ao}, \quad (\text{A.5})$$

where  $E_{ao}$  is aortic elastance.

$$\text{Venous pressure} = E_v * V_v, \quad (\text{A.6})$$

where  $E_v$  is venous elastance.

Left ventricle pressure is calculated as follows:

$$P_{lv} = tve * E_{eslv} * (V_{lv} - V_{d_{lv}}) + (1 - tve) * P_{0_{lv}} * (e^{\lambda_{lv} * (V_{lv} - V_{0_{lv}})} - 1), \quad (\text{A.7})$$

where

$tve$  (dimensionless) is the left ventricle time varying elastance,

$E_{eslv}$  (mmHg\*ml<sup>-1</sup>) is the left ventricle end-systolic elastance,

$V_{lv}$  (ml) is the left ventricle volume,

$V_{d_{lv}}$  (ml) is the unstressed left ventricle volume,

$P_{0_{lv}}$  (mmHg) is the zero-volume left ventricle pressure,

$\lambda_{lv}$  ( $\text{ml}^{-1}$ ) is the left ventricle lambda (the curvature of end-diastolic pressure-volume relationship (EDPVR) function/line),

$V_{0_{lv}}$  (ml) is the zero-pressure left ventricle volume.

The driver function concerning time-varying elastance (tve):

$$\text{tve} = e^{(-80 * (\text{time} - 0, 27)^2)}, \quad (\text{A.8})$$

where time is the period of time of a cardiac cycle.

Stroke volume (ml/beat):

$$\text{SV} = V_{\text{ed}} - V_{\text{es}}, \quad (\text{A.9})$$

where  $V_{\text{ed}}$  is the end-diastolic volume and  $V_{\text{es}}$  is the volume at the end of systole.

Ejection fraction (%):

$$\text{EF} = \frac{V_{\text{ed}} - V_{\text{es}}}{V_{\text{es}}} * 100 * 1.1, \quad (\text{A.10})$$

where 1.1 is a scalar used for consistency of physiological values for  $E$ .

Cardiac output (L/min):

$$\text{CO} = \text{SV} * \text{heart rate} / 1000, \quad (\text{A.11})$$

where division by 1000 is for conversion of milliliters to liters.

With every tick, calculations of variables concerning stocks and flows are performed and values for volumes and pressures are shown on the plot. SV, CO and EF are reported by respective monitors. Generated pressures and volumes for the left ventricle are used for setting coordinates for turtles, which are accordingly placed on the world window, creating the pressure-volume diagram.

The initial parameters for a “normal” scenario are as follows:

$V_{lv}$ —130 ml

$V_{ao}$ —50 ml

$V_v$ —1000 ml

$E_v$ —0.0059 mmHg\* $\text{ml}^{-1}$

$E_{ao}$ —0.6913 mmHg\* $\text{ml}^{-1}$

$V_{0_{lv}}$ —0 ml

$V_{d_{lv}}$ —0 ml

$P_{0_{lv}}$ —0.1203 mmHg

$$R_{\text{mt}}—0.0158 \text{ mmHg} \cdot \text{s} \cdot \text{ml}^{-1}$$

$$R_{\text{ao}}—0.018 \text{ mmHg} \cdot \text{s} \cdot \text{ml}^{-1}$$

$$R_{\text{sys}}—1.0889 \text{ mmHg} \cdot \text{s} \cdot \text{ml}^{-1}$$

$$E_{\text{esiv}}—2.8798 \text{ mmHg} \cdot \text{ml}^{-1}$$

$$\lambda_{\text{lv}}—0.033 \text{ ml}^{-1}$$

$$\text{heart rate}—70 \text{ bpm}$$

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