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Research Article

Determination of State Function Associated with Cardiovascular Dynamics

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Abstract

Every system can be described through state equations, which contain all the information on its internal dynamics. In this work we explain a basic model for the understanding of the state function associated with the cardiac wall, which could be analyzed for any specific pathology. The aim is to find the natural and forced behavior of the coronary system, treating it as an automatic control system, obtaining the pulse by pulse, that is, state to state, the response of the system applying the concept of Transition of States. Through the use of the Modern Control Theory and its tools such as the transfer function, state space and physiological dynamics, among others, it will be sought to determine the context situation of two classes of subjects, hypertensive patients and patients with normal blood pressure. Using models analogous to the cardiovascular system, we are interested in knowing the evolution of the response or output of the system for a given input and a set of given initial conditions. In addition, graphs in the state space will be new parametric indicators to keep in mind. The achieved results indicate that the use of this analysis developed in this work contributes to the understanding of cardiovascular functionality, provided that the parameters are adjusted for each particular case.

Keywords: Modeling; Status Function; Cardiac Dynamics; Control

Introduction

Bioengineering is a branch of Engineering that studies, among other things, the quantification of biological phenomena, such as the conductivity of blood and tissues, the mechanical response to an electrical stimulus or the study of bioelectric phenomena. Within the latter is framed the analysis of the cardiac signal, the electrocardiogram (ECG).

The electrocardiographic signal is the most studied biological signal in the world, despite this, there is no automated method that allows classifying the wave or signal to identify a normal heartbeat of which it is not [1].

In order to elaborate this work, we hypothesized that the heart is a stable system. We rely on homeostasis, which alludes to the tendency to maintain physiological balance by chemical compensation, and which allows, therefore, to consider the heart as a stable and feedback biological system [2]. This would validate the application of the general criteria of stability of a physical system, and the use of own computational procedures for the identification of anomalies.

The detailed study of cardiac dynamics is an essential tool when diagnosing heart disease. His analysis became more relevant from the incorporation of digital techniques that can be implemented with technology within the reach of an experimental laboratory, thus expanding the possibilities of diagnosis through computational algorithms.

Mathematical modeling is now widely applied in physiology and medicine as a support in the professional development of the scientist and clinical worker. A model is, by definition, an approximation of a system in terms of its representation [3]. The vascular system is a widely studied physiological system. Its hemodynamic characteristics, such as total peripheral resistance, total arterial compliance and the characteristic impedance of the proximal aorta allow us to understand the cardiovascular system [4].

Equations of state are first-order differential equations, simple to solve. In a system there may be internal dynamics (states) that instead of tending to zero or a limited value, increase their energy in the sense of making the system unstable. Considering the heart as a system with automatic control, we can analyze it from the point of view raised above.

Taking into account these concepts, the objective of this work is to apply the equations of state to the cardiovascular system with the purpose of finding cardiac function.

Materials and Methods

We have extracted records from the bank of normal and pathological patients of the Department of Biomedical Engineering of the Favaloro University (Buenos Aires – Argentina). We used in particular the registry of a population of eleven normotensive patients and that of fourteen hypertensive patients, from which records have been taken directly (systolic and diastolic pressures) and indirectly (pulse wave velocity and compliance).

Normo-tensos	PS mmHg	PD mmHg	PM mmHg	VOP m/s	Cm e-4cm/mmHg	L mmHg
1	93	57	69	10.39	4.03	36
2	127	80	96	12.25	4.15	47
3	104	66	79	9.05	5.03	38
4	120	89	99	10.78	3.43	31
5	91	75	80	10.94	3.14	16
6	97	70	79	11.11	3.57	27
7	118	66	83	9.28	5.36	52
8	85	51	62	7.80	6.40	34
9	96	61	73	8.83	4.08	35
10	117	77	90	9.80	3.62	40
11	119	64	82	10.69	4.00	55

Table 1: Normotensive patients.

Tables 1 and 2 list the data for two categories of patients, with normal blood pressure and hypertension (hypertension). Columns 2 and 3 show systolic and diastolic pressures, respectively. In column 4, the average pressure. Column 5 shows the pulse wave velocity. Column 6 lists the compliance calculated for each record. The values in column 7 indicate the inertia of the system, a parameter that is calculated by subtracting the diastolic pressure from the systolic pressure.

Hiper-tensos	PS mmHg	PD mmHg	PM mmHg	VOP m/s	Cm e-4 cm/ mmHg	L mmHg
1	146	96	113	15.21	2.00	50
2	106	84	91	14.58	2.21	22
3	116	65	82	10.07	3.49	51
4	157	89	112	11.15	3.76	68
5	166	98	121	14.25	2.68	68
6	164	92	116	16.26	2.22	72
7	127	82	97	11.44	3.76	45
8	155	92	113	17.16	2.48	63
9	155	70	98	10.83	3.70	85
10	139	100	113	11.28	4.04	39
11	134	84	101	10.27	4.82	50
12	114	75	88	11.31	3.12	39
13	117	78	91	14.07	2.31	39
14	125	83	97	14.78	1.68	42

Table 2: Hypertensive patients.

In the search for modeling the cardiac system using Modern Control Theory, we consider the heart as an electrical system (Windkessel Model) with automatic control [5].

Figure 1: Windkessel model.

The model consists of a parallel connection of a resistor and a capacitor. The resistance Rp represents the total peripheral resistance and the capacitor C represents the compliance of the vessels. Another resistive element between the pump and the air chamber, Rc, which simulates the resistance of blood flow due to the aortic or pulmonary valve. L is an inertial element in parallel with the characteristic resistance, Rc. With these arrangements, the model has the inertia of the entire arterial system at low frequencies, and at high and medium frequencies allow the characteristic resistance to intervene [6].

The state of a system in the initial time t is the amount of information in t_0 that together with the one intrada u[t,] ∞ determines in a unique way the behavior of the system, that is, determines the output y(t) for all $t>t_0$ [7]. There is a tau time constant which is the time when blood pressure drops to 1–e (approx. 37%) of its value at the beginning of the diastole; tau (τ) is the time constant of the fall of diastolic isobolometric pressure.

The model that manages to characterize cardiovascular dynamics in a simple way is the Windkessell electric model, which associates the cardiac system with its electrical analog [8], Figure 1. This model contains all the variables to be used in the development of our model in state space.

$$P = P_0 . e^{\frac{-t}{\tau}} \text{ -------(1)}$$

Where t is the time, P_0 the value of blood pressure at time= 0 and τ, the time constant.

The moment of the -dP/dt is considered as zero time and the instant when the pressure falls to one third of the value it had in time zero is detected. The time difference is tau and is equal to the product of R.C, where R is the peripheral resistance of all arterioles and C, arterial compliance or change of blood vessel volume per unit of pressure change. The decrease in tau implies a lower buffering capacity of pulsatility, which is well defined by equations of state.

$$\frac{dP}{dt} = \frac{1}{\tau}.P_0.e^{\frac{-t}{\tau}} = \frac{1}{\tau}.P \text{ ----------- (2)}$$

Is the equation that gives us the electrocardiographic signal.

A small τ (R.C) is typical of a less cushioned circulatory system because blood pressure during diastole drops abruptly. In hypertensive patients, arterial compliance is decreased and is a parameter that allows us to deduce that there are structural changes in the arterial wall.

Using the Branwell and Hill equation forcompliance,

$$Cm = \frac{1334.Dm}{2.r.VOP^2} \text{ -------(3)}$$

Where factors such as pulse wave velocity, blood viscosity and artery diameter are involved, data that served for the preparation of table 1.

The velocity of the pulse wave was calculated graphically using the following formula,

$$VOP = \frac{D}{\mathrm{D}x.T} \text{ -------(4)}$$

Where the distance separating both transducers intervenes, the distance on the x-axis between the two lower peaks of the carotid and femoral pressures [9] and in addition, the sampling interval, one beat one second. ≈

To achieve our goal we must describe the dynamics of the cardiovascular system, finding the input-output relationships in the space of states, considering the heart as a stable biological system with feedback [10].

Matricialmente,

$$\begin{matrix} \mathbf{x} = \mathbf{A.x} + \mathbf{B.u} \text{ ® Ecuación de Estado} \\ \mathbf{y} = \mathbf{C.x} + \mathbf{D.u} \text{ ® Ecuación de Salida} \end{matrix} \text{ ------------(5)}$$

Figure 2: System block diagram.

d where,

$\mathbf{x}$ ® Vector de Estado

$\mathbf{u}$ ® Vector de Entradas

$\mathbf{A}$ ® Matriz de Estado

$\mathbf{B}$ ® Matriz de Entrada

$\mathbf{C}$ ® Matriz de Salida

$\mathbf{D}$ ® Matriz de Transmisión Directa

The matrices of the Equation of State and Output of the systems (both patients):

--------(6)

RC= τ= 0.73.

To find the state function of cardiac dynamics we start from the System State Statement(5), and analyze it in Laplace space.

$$s\mathbf{X}(s) - \mathbf{X}(0) = \mathbf{AX}(s) + \mathbf{BU}(s) \text{ --------(7)}$$

To finally,

$$s\mathbf{X}(s) - \mathbf{X}(0) = \mathbf{AX}(s) + \mathbf{BU}(s) \text{ ------------(8)}$$

Anti-transforming the system we get,

$$\mathbf{x}(t) = e^{\mathbf{A}t}\mathbf{x}(0) + \int_0^t e^{\mathbf{A}(t-\tau)}\mathbf{Bu}(\tau)\,d\tau \text{ -----------(9)}$$

The matrix is called the $e^{\mathbf{A}t}$ Transition Matrix of States; it governs the trajectories of states in a finite time interval t.

Note that the system response has two components, the natural response which is the zero input response, due to the initial conditions, and the forced response, the zero state response, due to the input. The total answer is then the sum of both components.

$$\mathbf{x}(t) = \mathbf{x}_\mathbf{N}(t) + \mathbf{x}_\mathbf{F}(t) \text{ -----------(10)}$$

Results

Below are the calculated values for natural and forced responses:

Patient 8 – Table 1 – Lower mean pressure

$$\mathbf{x}_\mathbf{N}(t) = \begin{bmatrix} \frac{328}{897785} e^{\left(\frac{-50}{73}\right)t} \\ \frac{-3651}{24419752} e^{\left(\frac{-50}{73}\right)t} \end{bmatrix}$$

$$\mathbf{x}_\mathbf{F}(t) = \begin{bmatrix} 1 - e^{\left(\frac{-50}{73}\right)t} \\ \frac{73}{73259256} e^{\left(\frac{-50}{73}\right)t} \end{bmatrix}$$

Patient 5 – Table 2 – Higher Mean Pressure

$$\mathbf{x}_\mathbf{N}(t) = \begin{bmatrix} \frac{246}{2251355} e^{\left(\frac{-50}{73}\right)t} \\ \frac{-45925}{1025717338} e^{\left(\frac{-50}{73}\right)t} \end{bmatrix}$$

$$\mathbf{x}_\mathbf{F}(t) = \begin{bmatrix} 1 - e^{\left(\frac{-50}{73}\right)t} \\ \frac{365}{1025717338} e^{\left(\frac{-50}{73}\right)t} \end{bmatrix}$$

Figure 3: Natural Response – Space of States.

Figure 3 shows the response to states with zero excitation. It can be seen in the graph that for a heartbeat, the states of the patient with normal pressure, in black, are much higher than those of the patient suffering from arterial hypertension (red color).

Figure 4 represents the input response, non-zero excitation. It is visualized in the graph that this response requires very low states for the normotensive patient (black), however, the states of the hypertensive patient are high at the beginning of each pulsation.

Both responses (natural and forced) on the part of the hypertensive patient, in red, are characteristics of the behavior of a much more rigid heart wall compared to the normotensive patient.

Figure 4: Forced Response – Space of States.

Conclusions

Working in the Space of States from the equations of state and output allowed us to find the state, natural and forced functions that govern cardiovascular dynamics and to be able to graph them.

It allows the analysis of the intrinsic variables of the system to be studied and also to carry out a qualitative analysis of the model.

All this information that is accessed from the use of the control tools, serves as a reference, a complementary observation when making a diagnosis or treatment by the medical professional.

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Bibliography

1. Tompkins WJ. "Biomedical Digital Signal Processing". Prentice-Hall, Englewood Cliffs, New Jersey (1993).
2. Alvarez Picaza C., *et al*. "Modeling of the Cardiac System in the Space of States applying Controlability Criteria". 1st Congress of Bioengineering – Costa Rica (2009).
3. Cobelli C and Carson E. "Introduction to Modeling in Physiology". Academic Press, Elsevier. The Netherlands (2008).
4. Kind T., *et al*. "Estimation of Three- and Four-Element Windkessel Parameters Using Subspace Model Identification". *IEEE Transactions on Biomedical Engineering* 57.7 (2010): 1531-1538.
5. Alvarez Picaza C., *et al*. "MIMO system transfer functions in the study of cardiac dynamics". SABI 2020: XXII Congreso Argentino de Bioingeniería y XI Jornadas de Ingenieria Clinica (2020).
6. Alvarez Picaza C., *et al*. "Determination of models for the analysis of the control and stability of dynamical systems". Proceedings of the XIX Argentine Congress of Computer Science – Mar del Plata (2013): 1333-1342.
7. Ogata K. "Modern Control Engineering". 5ta ed., Ed. Spain: Pearson Prentice Hall (2007).
8. Kerner DR. "Solving Windkessel Models with MLAB".
9. Bia D., *et al*. "Vascular smooth muscle of the great arteries: site of local control of arterial damping function?". *Revista Española de Cardiología* 56.12 (2003): 1202-1209.
10. Monzón JE., *et al*. "Dynamic modeling of the vascular system in the state-space". Proceedings of the 32th Annual International Conference of the IEEE-EMBS (2010): 2612-2615.