Modeling and Simulation of Arterial Pressure Control

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Abstract: - Based on physiological studies and using experimental results published in specialty revues, the authors have created a mathematical model designated to simulate the arterial pressure control by arterial baroreflex. The model was validated by making comparison between the experimental data and the simulation results. The model has reproduced normal and pathological situations in concordance to experimental data.

Key-Words: - Heart rate variability, baroreflex, process control, modeling and simulation.

1 Introduction
The circulatory system is one of the most complicate physiological structures of the human beings. It is, also, the vital system that occupies the first place in human morbidity causes. So, it is naturally to be the subject of a great number of intensive studies that try to predict the evolution and the death risk for cardiovascular diseases.

In this paper the authors have realized the mathematical model of the baroreflex system, which control the arterial pressure (AP). Unfortunately, the experimentally data were not enough and some subsystems were modeled using uncompleted information. In another cases we have estimated the mathematical model using the physiological description.

The identification of models was made by using the physiological experiments data published in specialty revues. The model was tested also for pathological situations. In this cases the parameters were modified to simulate: hypertensive subject, tetraplegique subject and diabetes neuropathy or other neurological malady.

2 Mathematical Model
Neural and hormonal complex mechanisms achieve the short-term and long-term control of arterial pressure. The rapid reflex regulation of AP is realized by arterial baroreflex as a negative feedback control system.

The mathematical model was designed by the authors ([1], [2], [3], [6]) to study cardiovascular system control by arterial baroreflex. The structure of this model is represented in fig. 1.

One of the most important components of control system for arterial pressure is the baroreceptor. This is a complex structure who generates information for short-term regulation of AP. Arterial baroreceptors are mechano-sensors placed in aortic, sino-carotidian and other arterial walls and they respond to changes in stretch of these walls. Arterial baroreceptors start to discharge impulses as response to increase of AP. The firing frequency is a function of input pressure and baroreceptor anatomic characteristics and this function is nearly linear over the normal AP range (fig. 2). To simplify the model we ignored the "reset" property, which enables the adaptation of baroreceptor on long term.

This firing frequency generates motor outputs in medulla nervous centers. These motor outputs drive on sympathetic and parasympathetic (vagal) nerves and they act synergically in AP control.

The motor outputs increase parasympathetic nerve activity (PNA) and decrease sympathetic nerve activity (SNA). Both act on sino-atrial node (SAN) and modify the intrinsic pacemaker frequency of SAN.

The target variables for AP control are cardiac frequency ($F_C$), stroke volume ($V_s$) and peripheral resistance ($R_P$). The increase of AP generates the decrease of $F_C$ caused by the decrease of SNA and the increase of PNA. Also, increase of AP determines the decrease of $V_s$ and $R_P$, caused by decrease of SNA. The decrease of AP generates inverse responses in target variables.
For the baroreceptor we have used the transfer function with the structure presented in [3], [4], [5]:

\[
H_{\text{baroreceptor}}(s) = \frac{k_b(s^2 + b_1s + b_0)}{s^2 + a_1s + a_0}
\]  

(1)

From (1) it is possible to determine the state equations attached to the baroreceptor:

\[
\begin{align*}
x_1(t) &= x_2(t) \\
x_2(t) &= -a_0x_1(t) - a_1x_2(t) + P_A(t)
\end{align*}
\]

(2)

where

- \(P_A(t)\) - represents arterial pressure [mmHg];
- \(F_B(t)\) - represents the frequency of output signal of baroreceptor [Hz];
- \(k_b, a_0, a_1, b_0, b_1\) - parameters of baroreceptor.

The delay block is represented in time domain by the relation:

\[
F(t) = F_B(t - \tau)
\]

(3)

and has the transfer function:

\[
H_{\text{delay}}(s) = e^{-\tau s}
\]

(4)

The block which corresponds to sympathetic nerve activity (SNA) is represented by a first order model with the transfer function:
The time domain relation is:

$$Y_s(t) = -\frac{1}{T_{sn}} Y_s(t) + \frac{k_{sn}}{T_{sn}} [F_{os} - K_{bs} F(t)]$$  \hspace{1cm} (6)$$

The same form is used to model the parasympathetic nerve activity (PNA):

$$H_{PNA}(s) = \frac{k_{pn}}{T_{pn}s + 1}$$  \hspace{1cm} (7)$$

$$Y_v(t) = -\frac{1}{T_{pn}} Y_v(t) + \frac{k_{pn}}{T_{pn}} [F_{ov} + K_{bv} F(t)]$$  \hspace{1cm} (8)$$

where $F_{os}$ and $F_{ov}$ represent the basal (natural) frequency of sympathetic nerve activity and respectively parasympathetic nerve activity. The constants $k_{sn}$, $k_{pn}$, $K_{bs}$ and $K_{bv}$ are proportionally factors, while $T_{sn}$ and $T_{pn}$ are time constants.

Similarly, we choose for the stroke volume the approximated model:

$$H_v(s) = \frac{k_v}{T_v s + 1}$$  \hspace{1cm} (9)$$

$$V_s(t) = -\frac{1}{T_v} V_s(t) + \frac{k_v}{T_v} [F_{os} - K_{bs} F(t)]$$  \hspace{1cm} (10)$$

where $k_v$ is a proportionally factor and $T_v$ is a time constant.

Also, for peripheral resistance, it is possible to use the model:

$$H_R(s) = \frac{k_r}{T_r s + 1}$$  \hspace{1cm} (11)$$

$$R_p(t) = -\frac{1}{T_r} R_p(t) + \frac{k_r}{T_r} [F_{os} - K_{bs} F(t)]$$  \hspace{1cm} (12)$$

where $k_r$ is a proportionally factor and $T_r$ is a time constant.

The model was completed with coupling relations:
- for arterial pressure:

$$P_A(t) = Q_c(t) \times R_p(t)$$  \hspace{1cm} (14)$$

3 Simulation Results

The parameters of the mathematical model can be modified to reproduce a healthy subject behavior or to simulate a cardiac disease.

To avoid the transitory-state of the model, all the registrations were made after 200 seconds. The simulation results are presented in the next figures.

Instantaneous cardiac frequency for a healthy subject is represented in figure 3 and his spectral analyses is showed in figure 4. Figure 5 shows the average of AP and figure 6 shows his spectral analysis for the same subject.
Fig. 5. The instantaneous average of arterial pressure at healthy subject.

Fig. 6. Spectral analysis of the instantaneous average of arterial pressure at healthy patient.

Fig. 7. Instantaneous cardiac frequency at hypertensive subject.

Fig. 8. Spectral analysis of heart rate at hypertensive subject.

Fig. 9. The instantaneous average of arterial pressure at hypertensive subject.

Fig. 10. Spectral analysis of the instantaneous average of arterial pressure at hypertensive patient.
Fig. 12. Instantaneous cardiac frequency at subject with inhibition of sympathetic nerve activity.

Fig. 13. Spectral analysis of heart rate at subject with inhibition of sympathetic nerve activity.

Fig. 14. Arterial pressure at subject with inhibition of sympathetic nerve activity.

Fig. 15. Spectral analysis of the instantaneous average of arterial pressure at subject with inhibition of sympathetic nerve activity.

Fig. 16. Heart rate at subject with baroreceptor deafferentation.

Fig. 17. Spectral analysis of heart rate at subject with baroreceptor deafferentation.
The model was tested also for pathological situations. In these cases the parameters were modified to simulate: hypertensive subject, tetraplegique subject (corresponding to inhibition or complete denervation of sympathetic nerve) and diabetes neuropathy or other neurological malady (corresponding to baroreceptor deafferentation).

Instantaneous values for heart rate, average of arterial pressure and its spectral analyses for hypertensive subject are represented in fig. 7-10. In the case of inhibition of sympathetic nerve activity the results of simulation are presented in figures 11-14 (heart rate, average of AP and the spectral analysis). The results of simulation for the case of baroreceptor deafferentation are presented in fig. 15 (instantaneous heart rate) and in fig. 16 (spectral analysis).

4 Conclusion

Based on physiological studies and using experimental results published in specialty revues, the authors have created a mathematical model designated to simulate the arterial baroreflex of arterial pressure control. Unfortunately, the complexity of physiological phenomena can't be for moment, described by this model.

The model was validated by make comparison between the experimental data and the simulation results (forms and spectral analyses of signals). Physiological spectral components for healthy subjects are:
- ultra very low frequency (UVLF) and very low frequency (VLF) correspond to thermoregulation, reninangiotensine system, physical effort, etc.; normal values: 0.003-0.040 Hz.
- low frequency (LF) corresponds to sympathetique and parasympathetique nerve activity, baroreflex, etc.; normal values: 0.040-0.150 Hz.
- high frequency (HF) corresponds to parasympathetique nerve activity, respiration, etc.; normal values: 0.150-0.400 Hz.

These components were reproduced by the model for the healthy subject. Also, the model has generated the modified spectral components for the pathological situation simulated, in concordance with experimental values.

References: