COMPUTER SIMULATION OF HEART RATE AND BLOOD PRESSURE CHANGES, AND COMPARISON WITH CLINICAL DATA

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Introduction

The human body has highly complex physiological mechanisms to ensure that its physiological functions remain stable and are able to respond to peak demands. Physiologists and other clinical scientists have discovered much about the detailed functioning of the body, but its overall control is often difficult to predict.

This is where simulation techniques are helpful. Simulation allows physiological responses to be predicted, and then compared with actual clinical data. This enables different models to be assessed, and hence improves our understanding of the body's control mechanisms.

One example which researchers have considered, is that of blood pressure control. In our research, we have taken a published model for the control of blood pressure, discovered its limitations, and then improved the model so that it would more accurately predict actual clinical changes.

Control of blood pressure

Control of blood pressure is highly complex. An increase in heart rate will pump more blood from the heart around the body. This tends to increase blood pressure by increasing blood flow through the peripheral circulation. However, the body attempts to limit blood pressure, and so peripheral resistance can be lowered to reduce the increasing blood pressure. There are many good reviews covering this topic. Mary and Hainsworth discuss both control and methods of measurement (Mary and Hainsworth 1993).

Simulation of blood pressure control

Many research workers have investigated the control of blood pressure, and have used computer techniques to simulate this control. In our study, we used a model developed by de Boer (de Boer et al 1987). In this model there are two main feedback control pathways. Briefly, in one pathway heart rate controls cardiac output, and hence pulse pressure. This controls systolic pressure which completes the circle by controlling heart rate. In a second, and interlinked pathway, systolic pressure controls the peripheral resistance, diastolic pressure and the arterial time constant, which then in turn control

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pressure. These two pathways are also linked by factors which influence both heart rate (or RR interval) and the peripheral circulation. A greatly simplified diagram is illustrated in figure 1. In addition, respiration is a major factor in controlling changes in both heart rate and blood pressure. Some of the feedback is instantaneous, in that each heart beat produces an immediate effect upon blood pressure. There are also delayed effects built into the model. It is generally accepted that quick responses are associated with the parasympathetic nervous system, and slower responses with the sympathetic system, but slow responses also depend on some parasympathetic feedback.

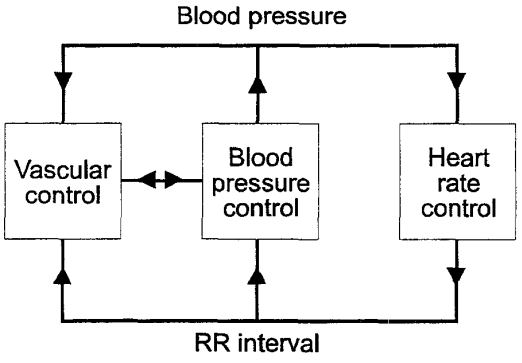

Figure 1. A simplified diagram of blood pressure and heart rate control.

Development of the model

We implemented all aspects of the model described above using Labview software running on a personal computer.

ECG and blood pressure data

The model was studied with data from normal subjects. Electrocardiograms (ECGs) were recorded from chest electrodes, and blood pressure with the non-invasive Finapres monitor, with the measurement probe attached to one finger. Respiration was recorded using a magnetometer instrument with chest mounted transducers (Griffiths et al 1983).

Subjects were asked to breath at ten breaths per minute, as this is known to accentuate the high frequency heart rate and blood pressure response.

Calculation of data from model

From the recorded data each RR interval was measured, and from each beat the systolic and diastolic pressures and arterial time constants were measured. The mean values of these were input to the model. In addition, the respiratory signal from the magnetometer recording was also input to the model.

From both the model and directly from the recorded data, spectral analysis was used to calculate the high frequency response (near 0.17 Hz, the respiratory frequency) and the low frequency response (expected to be close to 0.1 Hz).

Results

The data from the model were then compared with the data obtained directly, for each of the subjects. Using the original description of the model there were very large differences, especially in subjects with a high baroreflex sensitivity. Adapting the model to reduce the feedback, and introducing separate control of the low frequency and high frequency feedback dramatically improved the results, especially at the low frequency response. There was a 5 fold improvement in quantifying changes in RR and a 12 fold improvement in systolic blood pressure at low frequencies.

Discussion and conclusions

We do not claim that the original model, or our adaptation, gives an accurate description of blood pressure control. However, the model does shed some light on the feedback control, and gives some information on the level of feedback required to achieve results which compare with those from the real data. In addition, our results show that when feedback control for the low and high frequency pathways were handled separately, better results were obtained.

It must be remembered that simulation simply improves our understanding of the control process, and we make no claim that our model directly represents reality. However, the results do provide an insight into how heart rate and blood pressure are interrelated and controlled.

References

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