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ANALYSIS OF BAROREFLEX FUNCTION BY MEANS OF MATHEMATICAL MODEL

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Abstract

Re-evaluation of all functions of baroreflex by means of a simple mathematical model of circulation was the aim of the present study. The following states are modelled: 1. Rest. 2. Immediately after baroreceptor denervation. 3. Several days after denervation. 4. Physical exercise before denervation. 5. Physical exercise several days after denervation. Despite the same cardiac contractility and the same vasodilatation in working muscles as before denervation the cardiac output is by one third lower after baroreceptor denervation. In conclusion, a model simulation revealed the common regulation of blood pressure and blood volume by baroreflex and kidneys as a primary function of baroreflex.

Key words

Baroreflex, Mathematical model, Baroreceptor denervation, Exercise

INTRODUCTION

It is generally believed that the primary function of baroreflex is a short-time stabilization of blood pressure. Re-evaluation of all functions of baroreflex by means of a simple mathematical model of circulation was the aim of the present study.

METHODS

The simple mathematical model of circulation consists of three equations:

CO = (AP – VP) / TPR, M = Ca * AP + Cv * VP, CO = k * VP, where CO = cardiac output, AP = arterial pressure, VP = venous pressure, TPR = total peripheral resistance, M = total volume of blood in arterial and venous beds, Ca = arterial bed capacity (0.006 l/mmHg), Cv = venous bed capacity (0.6 l/mmHg), k = constant proportional to myocardial contractility (*1*). The following states are modelled: 1. Rest (AP = 100 mmHg, CO = 5 l/min, M = 3.6 l = total blood volume – volume of blood in the heart and lungs). 2. Immediately after baroreceptor denervation (increase in blood pressure 50 mmHg caused by increased TPR). 3. Several days after baroreceptor denervation (return of blood pressure to the original level caused by natriuresis and by corresponding decrease in blood volume).

4. Physical exercise before baroreceptor denervation (increase of cardiac output to 20 l/min at constant blood pressure, k and TPR are calculated, other values equal to rest values). 5. Physical exercise several days after baroreceptor denervation (TPR and k equal to values at exercise before denervation).

RESULTS

The results are seen in

Table 1

	Rest			**Exercise**	
	Before denervation	***Immediately after***	***Several days after***	***Exercise before***	***Exercise after***
AP mmHg	100	150	100	100	72.6
CO l/min	5	4.5	3	20	13.1
M l	3.6	3.6	2.4	3.6	2.4
TPR mmHg*min/l	19	32.3	32.3	4.75	5.3
k l/(min*mmHg)	1	1	1	4	4
VP mmHg	5	4.5	3	5	3.3

Despite the same value of cardiac contractility indicator k and the same decrease of TPR caused by vasodilatation in working muscles as before denervation the cardiac output is by one third lower after baroreceptor denervation. This demonstrates the disadvantage of baroreceptor denervation and thus the main function of the baroreflex.

DISCUSSION

The elimination of baroreflex by cutting baroreceptor efferents in animal experiments elicits immediately an increase in blood pressure of approximately 50 mmHg. Blood pressure returns to the original level by means of natriuresis during a few days. The trade-off is a decrease of blood volume, which causes decrease of cardiac output during exercise. This indicates that the primary function of baroreflex is vasodilatation by suppression of the activity of vasomotor level nerves. Therefore blood volume is held on a sufficiently high level.

Baroreceptors cannot measure the absolute values of blood pressure. The baroreceptor resetting during the night serves recalibration (*2*). The set point is approximately 50 mmHg under the value of blood pressure at which the suppression of vasomotor activity is completely abolished. The combined action of baroreflex control and endocrine humoral blood pressure by the kidneys secure relatively low blood pressure at a blood volume sufficiently high for increased cardiac output during exercise. The relatively low blood pressure decreases the oxygen consumption of the heart muscle and protects the brain vessels. The latter function is more important. Blood pressure in a giraffe is much higher than in man and other

mammals without negative consequences. The importance of brain blood vessel protection demonstrates the localization of baroreceptors in vessels conducting blood to the brain. The brain perfusion is fine-tuned by autoregulation during several seconds. We have demonstrated this reaction in man several years ago (*3*). This autoregulatory reaction disappears in hypertensives (*4*). It is seen not only from our study, but is also demonstrated by the observation of inadequate perfusion of brain in hypertensives after abrupt decrease of blood pressure by antihypertensive treatment. The low baroreflex gain insufficiently protects the human body from increase in blood pressure caused by psychological stress or by obesity. We have demonstrated in children and young adults that low baroreflex gain and obesity are two independent risk factors for high blood pressure (*5*). This study also treated "the egg and the hen problem". A low baroreflex gain was also observed in white-coat hypertension, i.e. before the increased blood pressure remodelled the carotid sinus wall (6).

CONCLUSION

In conclusion, the model simulation revealed the common regulation of blood pressure and blood volume by baroreflex and the kidneys as a primary function of baroreflex. The second important function is protection of brain vessels against the loss of autoregulatory ability, which is important for adequate supply of the brain with oxygen and nutrition. Baroreflex maintains adequate protection against hypertension in primitive civilizations; however, in obesity and/or psychological stress the low baroreflex gain is an independent risk factor of hypertension.

A c k n o w l e d g e m e n t

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