

RELATIONSHIP BETWEEN PULSE PRESSURE AND BAROREFLEX SENSITIVITY IN PATIENTS WITH ESSENTIAL HYPERTENSION

SIEGELOVÁ J.¹, HOFÍREK I.¹, FIŠER B.³, DUŠEK J.¹, PLACHETA Z.¹, JANČÍK J.¹, DOBŠÁK P.¹, SVAČINOVÁ H.¹, VANK P.¹, TOMAN J.²

¹Department of Functional Diagnostic and Rehabilitation, ²First Department of Internal Medicine/Cardioangiology, St. Anne's Teaching Hospital, Faculty of Medicine, Masaryk University, Brno, ³ Ministry of Health, Prague, Czech Republic

Abstract

The aim of the study was to analyse the relationship of pulse pressure and the total gain of baroreflex in patients with essential hypertension who were treated with ACE inhibitors or Ca antagonists. Blood pressure was measured beat-by-beat (Finapres Ohmeda, USA, 5 min). Baroreflex sensitivity of the heart rate was determined by means of spectral analysis. The baroreflex component of the blood pressure response was determined by means of an original method developed in our department. A group of 30 patients was divided, according to pulse pressure values corrected by mean arterial pressure (MAP), into two subgroups: one with a lower corrected pulse pressure and the other with a higher pulse pressure. The treated hypertensives with a higher pulse pressure were older and had a lower gain of the baroreflex response of systolic blood pressure.

Key words

Pulse pressure, Baroreflex heart rate sensitivity, Baroreflex blood pressure component, Essential hypertension

INTRODUCTION

There is evidence suggesting that an increased pulse pressure is one of important cardiovascular risk factors in patients with essential hypertension (1). Another risk factor is an increased sympathetic activity in essential hypertension (2). A decrease in baroreflex sensitivity is also a measure of increased sympathetic activity (3). Baroreflex (heart rate) sensitivity (BRS) in patients with essential hypertension is generally low (4,5). A slight increase in BRS, but not normalisation of its values, was observed after monotherapy with Ca antagonists or ACE inhibitors, as compared with placebo-controlled treatment (6). This study was aimed at the analysis of inter-relationship between pulse pressure (PP) and a total gain of baroreflex in patients with essential hypertension treated with ACE inhibitors or Ca antagonists.

MATERIALS AND METHODS

Thirty patients with mild essential hypertension were examined under treatment with Ca antagonists or ACE inhibitors. The whole group of 30 patients was divided, according to PP values corrected to MAP, into two subgroups: one with low pulse pressure (PPL) and the other with high pulse pressure (PPH).

Blood pressure was non-invasively measured beat-by-beat (Finapres Ohmeda, USA, 5 min, 7). Baroreflex heart rate sensitivity was determined by means of spectral analysis (8). The baroreflex component of blood pressure response was determined by means of an original method developed in our Department (9). We put occlusive cuffs on both thighs and the pressure in them was increased abruptly to a supersystolic value and maintained for 5 min. After the occlusion was released, both systolic and diastolic pressure decreased by 10 to 30 mmHg. The curve of blood pressure return to the initial level showed a linear slope in its middle part. This slope (velocity of return, mmHg/s) corresponded to the blood pressure component of baroreflex gain.

STATISTICS

The results are summarized as means (SD). The statistical significance of differences was determined by the Wilcoxon test. Further analysis was then performed by using the analysis of variance (ANOVA).

This study was approved by the Ethics Committee of the Masaryk University Teaching Hospital.

RESULTS

The data on age and blood pressure values (systolic blood pressure /SBP/, diastolic blood pressure /DBP/, PP, MAP) in PPL and PPH subgroups of patients with essential hypertension are presented in *Table 1* and the results of baroreflex gain (BRS and blood pressure components) analysis are shown in *Table 2*.

We did not find any differences in BRS values between the PPL and PPH patients with essential hypertension who were treated with Ca antagonists and ACE inhibitors. On the other hand, the systolic blood pressure baroreflex component was significantly increased in the PPH patients.

Our results also showed that the patients in the PPH subgroup were significantly older and had a lower baroreflex component of systolic blood pressure.

Table 1

Age and blood pressure values (mean±SD) in two subgroups of patients with essential hypertension

Patient subgroup	Age (in years)	SBP (mmHg)	DBP (mmHg)	PP (mmHg)	MAP (mmHg)
PPL	57±9	135±21	75±1	62±14	118±16
PPH	65±9*	146±18	66±11	81±15*	119±13

SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MAP, mean arterial pressure; PPL, low-pulse pressure; PPH, high-pulse pressure; *, $p < 0.05$, PPL vs. PPH subgroups, Wilcoxon test.

Table 2

Results of baroreflex gain (BRS and blood pressure components) assessment in two subgroups of patients with essential hypertension

Patient subgroup	BRS	BP BRS	
	(ms/mmHg)	SBP (mmHg/s)	DBP (mmHg/s)
PPL	3.73 +3.06	1.05+0.78*	0.71+0.53
PPH	2.14+2.27	0.71+0.50	0.59+0.45

BRS, baroreflex sensitivity; BP BRS, blood pressure component of baroreflex gain; SBP, systolic blood pressure; DBP, diastolic blood pressure; PPL, low-pulse pressure; PPH, high-pulse pressure;

*p<0.05, PPL vs. PPH, Wilcoxon test.

DISCUSSION

An increase in pulse pressure is a risk factor for cardiovascular morbidity and mortality (10). The magnitude of pulse pressure has been found to depend on the left ventricular ejection and properties of the arterial wall, which determines both the compliance and the transmission characteristics of the arterial system (1). Thus an increase in pulse pressure can occur by means of an increase in the velocity of ventricular ejection, a reduction in viscoelastic properties of the arterial wall, or a modification in the timing of reflected waves (11). More recently, the Framingham study has shown that the risk of coronary artery disease is inversely related to diastolic blood pressure at a systolic blood pressure value of at least 120 mmHg. This suggests that a higher pulse pressure is an important component of this risk (1).

Sympathetic overactivity is another important risk factor for cardiovascular mortality (2). The results from other laboratories, together with the results of the present study, can contribute to the elucidation of problems related to two questions: Why is BRS low in essential hypertension and what are the changes in baroreflex that are responsible for a treatment-induced BRS increase? An attractive hypothesis that low baroreceptor sensitivity is compensated by increased responsiveness of the smooth muscles of an hypertrophic arteriolar wall (12) is not supported by the evidence available. A long-lasting therapy with the ACE-inhibitor enalapril decreased blood pressure and normalised the forearm blood flow after 5 min of ischaemia, which indicated regression of hypertrophy of the resistance vessels (13). However, in patients treated with enalapril, BRS remained low at about half of the value shown by age-matched normotensive controls (14).

The subgroup of treated hypertensives with high pulse pressure had a higher mean age. In this group, a lower baroreflex component of systolic blood pressure was detected.

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Siegelová J., Hofířek I., Fišer B., Dušek J., Placheta Z., Jančík J., Dobšák P., Svačinová H., Vank P., Toman J.

VZTAH MEZI TEPOVÝM TLAKEM A BAROREFLEXNÍ SENSITIVITOU U PACIENTŮ S ESENCIÁLNÍ HYPERTENZÍ

Souhrn

Cílem studie byla analýza vztahu mezi pulzovým tlakem a celkovým ziskem baroreflexu u pacientů s esenciální hypertenzí léčených ACE inhibitory nebo Ca antagonisty. Krevní tlak byl měřen po jednotlivých pulsech (Finapres Ohmeda, USA, 5 min). Baroreflexní sensitivity srdeční frekvence byla stanovena pomocí spektrální analýzy. Baroreflexní složka reakce krevního tlaku byla stanovena původní metodou vyvinutou na naší katedře. Celá skupina 30 pacientů byla rozdělena podle hodnot pulzového tlaku korigovaného podle MAP na dvě poloviny: soubor s nižším korigovaným pulzovým tlakem a s vyšším pulzovým tlakem. Léčení pacienti s hypertenzí s vyšším pulzovým tlakem jsou starší a mají nižší zisk baroreflexní reakce systolického krevního tlaku.

REFERENCES

1. *Dart AM, Kingwell BA.* Pulse pressure – a review of mechanisms and clinical relevance. *J Am Coll Cardiol* 2001; 37: 975s–984s.
2. *Mancia G.* The sympathetic nervous system in hypertension. *J Hypertens* 1997; 15: 1553–1565.
3. *Grassi G, Seravalle G, Gattaneo BM et al.* Sympathetic activation and loss of reflex sympathetic control in mild congestive heart failure. *Circulation* 1995; 92: 3206–3211.
4. *Grassi G, Gattaneo BM, Seravalle G, Lanfranchi A, Mancia G.* Baroreflex control of sympathetic nerve activity in essential and secondary hypertension. *Hypertension* 1998; 31: 68–72.
5. *Siegelová J, Fišer B, Dušek J, Al-Kubati M.* Baroreflex-Sensitivitaetsmessung bei Patienten mit essentieller Hypertonie: Einfluss von Enalapril. *Nieren und Hochdruckkrankheiten* 1995; 24: 20–22.
6. *Siegelová J, Fišer B, Dušek J.* Baroreflex heart rate sensitivity in essential hypertension: the effect of Ioptin SR 240 therapy. In: Varró V, de Chantel R, eds. 22nd Congress of International Society of Internal Medicine, Bologna: International Proceeding Div. Monduzzi Editore, 1994: 99–102.
7. *Peñáz J.* Photoelectric measurement of blood pressure, volume and flow in the finger. In: Digest of the 10th Int. Conf. Med. Biol. Engineering, Dresden, 1973: 104.
8. *Robbe HWJ, Mulder LJM, Ruddel H, Langewiz WA, Mulder G.* Assessment of baroreceptor reflex sensitivity by means of spectral analysis. *Hypertension* 1987; 10: 538–543.
9. *Savin E, Siegelová J, Fišer B, Bonnin P.* Intra- and extracranial artery blood velocity during a sudden blood pressure decrease in humans. *Eur J Appl Physiol* 1997; 76 :289–293.
10. *Safar ME.* Pulse pressure in essential hypertension: clinical and therapeutical implications (editorial review). *J Hypertens* 1989; 7: 769s–776s.
11. *Jokiniitty JM, Majahalme SK, Kahonen MAP, Tuomisto MT, Turjanmaa VMH.* Pulse pressure is best predictor of future left ventricular mass: 10 years of follow-up. *J Hypertens* 2001; 19: 2047s–2084s.
12. *Zimmerman BG.* Adrenergic facilitation by angiotensin: does it serve a physiological function? *Clin Sci* 1981; 60: 343–348.
13. *Folkow B.* The Fourth Volhard Lecture: cardiovascular structural adaptation: its role in the initiation and maintenance of primary hypertension. *Clin Sci Mol Med* 1978; 4: 3s–22s.
14. *Siegelová J, Fišer B, Al-Kubati M, Dobšák P, Dušek J.* Noninvasive determination of baroreflex sensitivity in hypertensives treated for five years with enalapril. *Scripta Med* 1997; 70: 239–244.