

## PULSE PRESSURE AND CAROTID BLOOD FLOW IN ESSENTIAL HYPERTENSION

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### Abstract

There is some evidence to suggest that increased pulse pressure in patients with essential hypertension may be an important indicator of cardiovascular risk. The aim of the present study was to assess brain blood perfusion in patients with essential hypertension treated with ACE inhibitors or Ca antagonists. Thirty male patients involved in the investigation were examined in two consecutive parts of the study, each using a different criterion for the assessment of brain perfusion on the basis of carotid blood flow and carotid artery resistance (R, measured in mmHg x min/ml). The criterion used first was the value of pulse pressure correlated with the mean blood pressure. There was no difference in artery resistance between the patients with low-pressure pulse and those with high-pressure pulse. The second criterion was the value of systolic blood pressure (SBP). The 14 patients with SBP lower than 140 mmHg had a lower R value than the 16 patients with SBP higher than 140 mmHg. Our results showed that the value of pulse pressure has no validity as a diagnostic indicator in patients suspected to have structural changes in cerebral arteries.

### Key words

Pulse pressure; Essential hypertension; Carotid blood flow

### INTRODUCTION

The main function of baroreceptors is the regulation of blood pressure at the level of cerebral arteries. The baroreceptors are localised in the carotid sinus and the aortic wall near the origin of common carotid arteries. In essential hypertension, baroreflex sensitivity is impaired (1). A relationship between the risk of mortality and morbidity and high blood pressure in patients with hypertension has been studied, but relatively little data is available on brain perfusion in these patients. In addition, in essential hypertension, elevated pulse pressure (PP) and increased sympathetic activity enhance the patients' risk of cardiovascular disease (2, 3). Baroreflex (heart rate) sensitivity (BRS) in patients with essential hypertension is generally low (4,5).

A slight increase in BRS, but no return to normal values, was observed after a monotherapy with Ca antagonists or ACE inhibitors, as compared with placebo treatment (6). In our recent study of the treatment of essential hypertension with ACE inhibitors and Ca antagonists, patients with elevated PP showed a decrease in baroreflex sensitivity; elevated PP was diagnosed in older patients (7).

The aim of this paper was to study blood flow in carotid arteries and brain perfusion in patients with essential hypertension treated with ACE inhibitors and Ca antagonists.

#### MATERIALS AND METHODS

Thirty male patients with mild essential hypertension treated with Ca antagonists or ACE inhibitors were included in the study. Their blood pressure values were measured, beat-by-beat, by a Finapres Ohmeda, USA (8) and pulse pressure values were calculated. Blood flow in the common carotid artery (CC) was assessed by Doppler echography (SONOS 5500, Hewlett Packard, USA). Brain perfusion was evaluated on the basis of blood vessel resistance (R) measured in the neck region of the common carotid artery. The R values were calculated as  $R = \text{MAP} / \text{blood flow in CC}$ .

The study was carried out in two parts. In the first, on the basis of PP values correlated with the mean arterial pressure (MAP), the patients fell into two subgroups: 15 patients with higher pulse pressure (PPH) and 15 patients with lower pulse pressure (PPL). In the second, the 30 patients were divided according to systolic blood pressure (SBP) into subgroup A (14 patients, SBP lower than 140 mmHg) and subgroup B (16 patients, SBP higher than 140 mmHg).

The local Ethics Committee of St. Anne's Teaching Hospital approved of the study and all patients signed an informed consent.

The results were summarized as mean  $\pm$  SD values. The statistical significance of differences was determined by Wilcoxon's test.

#### RESULTS

Cardiovascular parameters obtained in part one for all 30 patients are shown in *Table 1*. MAP, SBP and DBP values were similar in both subgroups and there was no difference between carotid blood flow and R values. In patients with essential hypertension, the magnitude of PP values at the same MAP did not affect the blood pressure resistance in the CC.

In part two, the patients were divided according to systolic blood pressure. Subgroup B patients had higher values of PP and MAP. Although there was no difference in CC blood flow between the subgroups, the R value was significantly higher in subgroup B (*Table 2*).

#### DISCUSSION

The anti-hypertensive therapy with either ACE inhibitors or Ca antagonists generally lowers blood pressure but with noticeable inter-individual differences. Our results showed that, when systolic blood pressure is not lowered under the recommended value of 140 mmHg, elevated levels of PP and MAP are found and a higher resistance of the CC is present.

Table 1

Cardiovascular characteristics of patient subgroups defined by pulse pressure values correlated with the mean arterial pressure

Sub-group	Age (years)	SBP (mmHg)	DBP (mmHg)	PP (mmHg)	MAP (mmHg)	flow CC (ml/min)	R (mmHg x min/ml)
PPL	57±9	135±21	75±11	62±14	118±16	712±145	0.174±0.050
PPH	65±9 *	146±18	66±11	81±15*	119±13	728±171	0.176±0.053

PP, pulse pressure; MAP, mean arterial blood pressure; PPL, patients with low-pulse pressure; PPH, patients with high-pulse pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; CC, common carotid artery; R, resistance in CC. \*, PPL versus PPH at p<0.05.

Table 2

Cardiovascular characteristics of patient subgroups defined by systolic blood pressure values

Sub group	Age (years)	BP (mmHg)	DBP (mmHg)	PP (mmHg)	MAP (mmHg)	flow CC (ml/min)	R (mmHg x min/ml)
A	61±7	124±12	67±9	59±10	107±8	748±141	0.147±0.027
B	60±11	153±14*	73±13	82±14*	129±10*	695±169	0.198±0.057*

PP, pulse pressure; MAP, mean arterial blood pressure; PPL, patients with low-pulse pressure; PPH, patients with high-pulse pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; CC, common carotid artery; R, resistance in CC. \*, A versus B at P<0.05.

In hypertensive patients, the relationship of blood pressure to blood flow in the carotid artery is related to structural changes in the brain arteries due to high arterial pressure (9). Structural changes in the resistance vessels lead to a better response to high pressures but to a poorer response to hypotension. This is presumably because autoregulatory mechanisms are compromised, causing ischaemic brain damage (10). In our previous studies we observed that cerebral circulation did not function properly in hypertensive patients who had not been treated for this condition (11, 12).

Our results suggest that the PP value magnitude cannot be considered an indicator of structural changes in cerebral arteries. The MAP value, on the other hand, is closely related to the state of brain blood flow.

The approach used in the present study enabled us to evaluate the effect of treatment on the resistance vessels, especially carotid arteries. It provided better results than a mere measurement of blood pressure that varies and therefore cannot serve as a reliable indicator of the severity of disease, as shown in subjects with “white coat” hypertension.

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## TEPOVÝ TLAK A PRŮTOK KRVE KAROTICKÝM ŘEČIŠTĚM U ESENCIÁLNÍ HYPERTENZE

### Souhrn

Zvýšený pulsový tlak u pacientů s esenciální hypertenzí by mohl být důležitým ukazatelem kardiovaskulárního rizika. Cílem studie byla analýza perfúze mozku u 30 mužů s esenciální hypertenzí léčených ACE-inhibitory nebo vápníkovými antagonisty. Pacienti byli vyšetřováni ve dvou etapách za použití odlišných kritérií pro posouzení průtoku a odporu krve v arteria carotis communis jako indikátoru perfúze mozku. V první etapě byla hodnota pulsového tlaku korelována se středním arteriálním tlakem. Nebyly zjištěny rozdíly u pacientů s vyšším a nižším pulsovým tlakem. Hodnota systolického krevního tlaku (140 mmHg) byla použita jako druhé kritérium. 14 pacientů se systolickým krevním tlakem nižším než 140 mmHg mělo nižší odpor než 16 pacientů s vyšším systolickým krevním tlakem. Naše výsledky ukázaly, že hodnota pulsového tlaku není významná pro diagnostiku strukturálních změn v mozkových arteriích u pacientů s hypertenzí.

### REFERENCES

1. *Mancia G.* The sympathetic nervous system in hypertension. *J Hypertens* 1997;15:1553–1565.
2. *Dart AM, Kingwell BA.* Pulse pressure – a review of mechanisms and clinical relevance. *J Am Coll Cardiol* 2001;37:975–984.
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 Isoptin SR 240 therapy. In: Varró V, de Chantel R, eds. 22<sup>nd</sup> Congress of International Society of Internal Medicine, Bologna: International Proceeding Div. Monduzzi Editore, 1994:99–102.
7. *Siegelová J, Hofírek I, Fišer B, Dušek J, Placheta Z, Jančík J, Dobšák P, Svačinová H, Vank P, Toman J.* Relationship between pulse pressure and baroreflex sensitivity in patients with essential hypertension. *Scripta medica* 2002;2:101–104.
8. *Peñáz J.* Photoelectric measurement of blood pressure, volume and flow in the finger. In: Digest of the 10<sup>th</sup> Int. Conf. Med. Biol. Engineering, Dresden, 1973:104.
9. *Baumbach GL, Chillon JM.* Effects of angiotensin-converting enzyme inhibitors on cerebral vascular structure in chronic hypertension. *J Hypertens* 2000;18(suppl 1):7–11.
10. *Strandgaard S, Paulson OB.* Cerebral blood flow in untreated and treated hypertension. *Neth J Med* 1995;47:180–184.
11. *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.
12. *Siegelová J, Fišer B, Dušek J, Savin E.* Carotic peripheral vascular resistance in treated and non-treated hypertensives. *Arch Physiol Biochem (Formerly Arch Inter Physiol Biochem Biophys)* 1997;105:246–247.