MINIMUM FOREARM VASCULAR RESISTANCE IN ESSENTIAL HYPERTENSION AFTER ANTIHYPERTENSIVE THERAPY

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A b s t r a c t

The aim of the present study was to analyse the relationship between the blood pressure decrease and minimum forearm vascular resistance decrease due to different anti-hypertensive therapies. Patients with mild to moderate essential hypertension (n=40) were examined after placebo therapy and 3 months after the following therapy with Ca antagonists verapamil (n=10), nitrendipine (n=10), dilthiazem (n=10), and with the ACE inhibitor enalapril (n=10). From our results we conclude that anti-hypertensive therapy with all Ca antagonists and the ACE inhibitor decreased the minimum forearm vascular resistance. No differences among these drugs were observed.

K e y   w o r d s

Minimum forearm vascular resistance, Essential hypertension, Antihypertensive therapy, Ca antagonists, ACE inhibitor

INTRODUCTION

Vascular hypertrophy, which is inferred from an increase in minimum forearm vascular resistance (FVR$_{min}$), is present in patients with essential hypertension without therapy *(1)*. The development of structural changes in the systemic arteries and arterioles contributes to the continuation and progressive worsening of arterial hypertension *(2)*. The aim of the present study was to analyse the relationship between blood pressure (BP) decrease and (FVR$_{min}$) decrease due to a range of different antihypertensive therapies.

MATERIALS AND METHODS

The study was carried out on 40 male patients (age, 48±5 years; height, 181±5cm; weight, 86±8kg) with mild to moderate essential hypertension who had been treated with placebo therapy followed by administration of different anti-hypertensive drugs for 3 months. According to the medication used, the patients were distributed into four groups, three received Ca antagonists and the fourth an angiotensin-converting enzyme (ACE) inhibitor. Group EH V (n=10) received

verapamil at a single morning dose of 240 mg (slow releasing) every day; group EH N (n=10) was given nitrendipine at a dose of 20 mg twice a day; group EH D (n=10) was treated with dilthiazem at a dose of 90 mg (retard) twice a day. Group EH E (n=10) received enalapril at a single daily dose of 13.3±2.1mg.

Medical tests and examinations excluded any other disease and the diagnosis of essential hypertension was made according to the WHO criteria to rule out any form of secondary hypertension. All patients had been followed up at the clinic at least 3 years prior to this study.

We measured blood pressure and the maximum forearm blood flow after occlusion plethysmography (5 min; occlusion pressure, 300 mmHg) using a Fluoscript device. The forearm blood flow was measured and expressed in ml per 100 ml of tissue per min. The minimum forearm vascular resistance (FVRmin) was calculated from the mean arterial blood pressure value (MAP, mmHg) divided by the maximum forearm blood flow. These tests were carried out on 40 patients at the end of placebo treatment and on each of the four groups after 3 months of anti-hypertensive treatment.

The results were calculated as mean±SD for the four groups during the placebo phase. The statistical significance of differences between the placebo and treatment stages in each group was determined by the Wilcoxon test for paired data.

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

RESULTS

The following results are presented in *Table 1* and *Fig. 1*: FVR $_{min}$ (mmHg/ ml per 100 ml per min; mean±SD) and BP (MAP, mmHg; mean±SD) in each treated group during the placebo therapy phase; the decrease due to the therapy in MAP and FVR$_{min}$ (dP and dR, expressed as percentages) and the dP/dR ratio.

On the basis of our results we conclude that anti-hypertensive therapy with different Ca antagonists or with an ACE inhibitor decreased the minimum forearm vascular resistance. There were no observed differences associated with these drugs.

DISCUSSION

In the pathogenesis of essential hypertension, elevated blood pressure is connected with increased peripheral resistance. Many recent studies have shown that ACE inhibitors are very effective in preventing or inhibiting many of the morphological and functional changes associated with hypertension that take place in the media as well as in the endothelium of arteries and arterioles (3). In a clinical study, it has been shown that long-lasting treatment with ACE inhibitors induced relaxation in arterial smooth muscles and, therefore, an increase in arterial diameter and compliance in hypertensive patients. These vascular protective effects seem to be at least partly independent of the anti-hypertensive effect of these drugs since, in many cases, pure vasodilatators do not have similar effects (4,5). Radial artery compliance–pressure curves were not changed by placebo, atenolol or nitrendipine treatment, but they were changed in subjects treated by lisinopril, an ACE inhibitor (6).

Table 1

The results of FVRmin (in mmHg/ ml per 100 ml per min) and the mean BP (MAP, mmHg) in the four groups after the therapy, and a decrease in MAP (dP,%), a decrease in FVRmin (dR,%) and the dP/dR ratio

	EH V	EH N	EH D	EH E
FVR min	4.7±1.6	6.6±1.5	4.1±1.5	4.6±1.5
MAP	118.8±7.1	115.7±5.6	121.1±9.1	109.7±8.7
dP	*12.4±7.1	*13.7±6.5	*17.5±7.9	*10.4±6.9
dR	*32.61±8.6	*36.2±7.6	*44.51±5.2	*41.1±15.3
dR/dP	2.6±1.4	2.6±0.5	2.5±0.9	4.1±1.6

FVRmin, minimum forearm vascular resistance; BP, blood pressure; MAP, mean arterial pressure; *, $P<0.05$ (statistical significance of differences between the groups in the treatment versus the placebo phases).
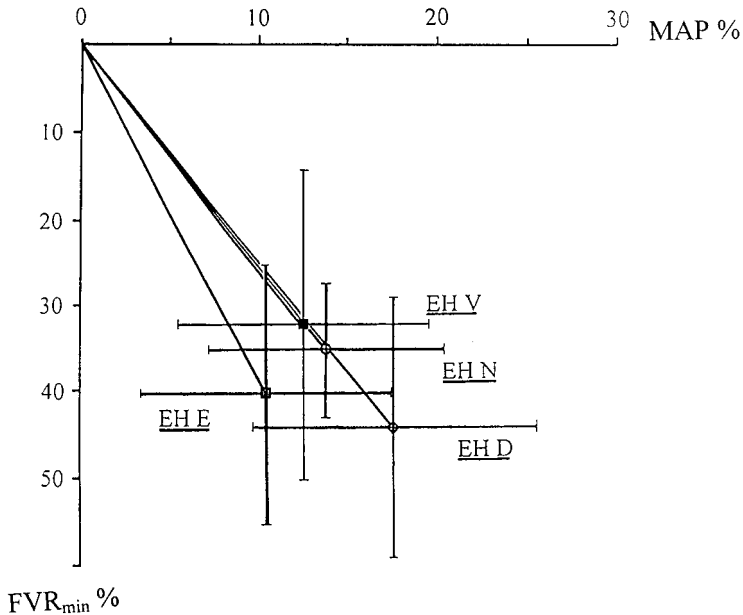

Fig.1

Relationship between the decrease in mean blood pressure (MAP %) and the decrease in forearm vascular resistance (FVRmin %) caused by the antihypertensive treatment by verapamil (EH V), nitrendipine (EH N), dilthiazem (EH D) and enalapril (EH E).

Our previous results *(7)* as well as the results from the present study did not confirm these conclusions completely. The findings of the present study showed that the FVR_{min} decreasing effect is not greater after ACE-inhibitor treatment than that after verapamil, nitrendipine and dilthiazem and also that Ca-antagonists, themselves, are very effective in remodelation of resistance vessels.

In agreement with our results, there are also the results from a clinical study by *Rossi et al. (1).* These authors prospectively investigated the relationship of plasma renin activity and aldosteron levels to carotid artery lesions in essential hypertension. They have found that primary hypertensive patients have a prevalence and severity of carotid artery lesions higher than normotensive subjects. The maximal degree of stenosis was more strongly predicted by pulse pressure and the known duration of hypertension. Their findings do not support the hypothesis that a high renin-sodium profile carries an excess risk of carotid artery lesion in primary hypertensives.

Dušek J., Siegelová J. Fišer B., Savin E., Martineaud J.P.

MINIMÁLNÍ CÉVNÍ REZISTENCE PŘEDLOKTÍ U ESENCIÁLNÍ HYPERTENZE PO ANTIHYPERTENZIVNÍ LÉČBĚ

S o u h r n

Cílem této práce byla analýza vztahu mezi poklesem krevního tlaku a poklesem minimální cévní rezistence v důsledku odlišné antihypertenzivní léčby. Vyšetřili jsme pacienty s mírnou až střední esenciální hypertenzí po léčbě placebem a po 3 měsících léčby Ca antagonisty verapamilem, nitrendipinem, dilthiazemem a ACE inhibitorem enalaprilem. Z našich výsledků vyplývá, že antihypertenzivní léčba Ca antagonisty a ACE inhibitorem snížila minimální cévní rezistenci a mezi těmito léky nebyl prokázán žádný rozdíl.

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