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TREATMENT WITH FOSINOPRIL DECREASES LEFT VENTRICULAR MASS IN PATIENTS WITH ESSENTIAL HYPERTENSION. Bernard MY CHEUNG, CP LAU. University Department of Medicine, University of Hong Kong, Hong Kong.

Hypertensive left ventricular hypertrophy (LVH) is a powerful risk factor for cardiovascular morbidity and mortality. Therefore, treatment of hypertension should reduce blood pressure and LVH. Meta-analysis suggested that angiotensin-converting enzyme inhibitors are effective in regression of LVH but few controlled studies have been performed.

32 patients not previously treated for hypertension were randomised to receive fosinopril (10-20 mg daily) or placebo for 12 weeks. All patients were given life-style advice. Echocardiography was performed at the beginning and end of the study. One-third of patients had LVH by echocardiographic criteria. Diastolic pressure was significantly reduced by fosinopril compared to placebo (-5.6 $\pm$ 2.2 mmHg vs 1.3 $\pm$ 2.3 mmHg, $p = 0.03$). Left ventricular mass index (LVMI) was also significantly reduced by drug treatment compared to placebo (-11.7 $\pm$ 4.6 g/m^2 vs 1.7 $\pm$ 4.1 g/m^2, $p = 0.04$). The reduction in LVMI was not significantly related to the reduction in blood pressure ($r = 0.24$, $p = 0.2$). Ejection fraction and renal function were unaffected in the treatment group. No serious adverse events occurred. This study showed that a relatively short period of treatment can reduce LV mass.

	placebo (n = 15)		fosinopril (n = 17)	
	baseline	final visit	baseline	final visit
diastolic blood pressure	95.9 $\pm$ 2.2 mmHg	98.1 $\pm$ 2.6 mmHg	96.6 $\pm$ 2.3 mmHg	91.5 $\pm$ 3.0 mmHg
left ventricular mass index	110 $\pm$ 9 g/m^2	112 $\pm$ 9 g/m^2	121 $\pm$ 6 g/m^2	109 $\pm$ 8 g/m^2

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DECLINE IN FREQUENCY OF DD GENOTYPE IN HYPERTENSIVE PATIENTS WITH AGE. Bernard MY CHEUNG, Raymond LEUNG, Kathryn TAN. University Department of Medicine, University of Hong Kong, Hong Kong.

There is mounting evidence that an insertion/deletion polymorphism in the angiotensin-converting enzyme (ACE) gene is associated with the risk of myocardial infarction. Hence, the ACE genotype was determined in 75 patients attending a hypertension clinic and 83 healthy controls. Genomic DNA was extracted from peripheral leucocytes and amplified by PCR using standard primers and conditions. Insertion (I) or deletion (D) alleles were identified after electrophoresis. A significant correlation between ACE genotype and age was found in hypertensive patients ($p = 0.04$)(Table). Furthermore, the relative frequency of the D allele was significantly lower in patients $\geq$50 years compared to those <50 years (0.22 vs 0.41 respectively, $p = 0.01$), especially in the sub-group of newly-diagnosed untreated hypertensive patients (0.05 vs 0.46 respectively, $p = 0.002$). However, the distribution of phenotypes and allelic frequencies in patients <50 years were not significantly different from controls ($p = 0.94$).

The observed decrease in frequency of the D allele in older hypertensive patients, particularly in the untreated, is consistent with premature death of hypertensive DD individuals due to cardiovascular disease.

	Age	n	DD	ID	II
Normal controls	<50	69	22%	36%	42%
	$\geq$50	14	43%	21%	36%
Hypertensive patients	<50	28	21%	39%	39%
	50-59	16	6%	44%	50%
	>60	31	3%	32%	65%