Echocardiographically Detected Left Ventricular Hypertrophy: Prevalence and Risk Factors in Thai Elderly Men and Women

SOMRAK CHANTRA, M.D.*,
BANHARN BHUTHONG, M.D.*

Abstract

Objective: To find the prevalence of and risk factors associated with echocardiographically determined left ventricular hypertrophy (LVH) in the general Thai elderly men and women.

Background: The prevalence of LVH and risk factors is not at present available for precise assessment of the seriousness in the general Thai population. The prevalence of LVH may be influenced by race, obesity, age, sex and hypertension. Their effects on left ventricular mass (LVM) have not been defined in the general Thai population.

Method: A cohort of 157 subjects 60 years of age and over was studied. LVM was calculated using the modification of the ASE cube formula. Criteria for LVH were based on various LVM indexation using the published conventional partition values. Univariate and multivariate analyses with various variables were studied.

Result: M-mode echocardiographic studies of adequate quality were obtained in 125 (80%) of 157 participants. Prevalence of LVH depended on the different types of indexation. LVH defined by 1) unindexes LVM (≥259g in men (M), ≥166 g in women (F)) was 35(28%); 2) defined by LVM/BSA (≥131 g/m^2 in M, ≥100g/m^2 in F) 63 (50%); 3) (≥117 g/m^2 in M, ≥104 g/m^2 in F) 68 (54%); 4) (≥125 g/m^2 in both M and F) 43 (34%); 5) defined by LVM/ height (≥143 g/m in M and ≥102 g/m in F) 49 (39%); 6) (≥126 g/m in M and ≥105 g/m in F) 52 (42%); 7) defined by LVM/height$^{2.7}$ (≥51 g/m$^{2.7}$ in both M and F) 62 (50%); 8) (≥50 g/m$^{2.7}$ in M, ≥47 g/m$^{2.7}$ in F) 77 (62%). The following variables independently predicted LVM in descending order of statistical significance: BW, BMI, SBP, PP, height were the most significant ($p < 0.01$), whereas, DBP and gender made smaller contributions and age showed no correlation. In multivariate analysis only BW and PP showed significant correlation with LVM in the total population.

Conclusion: LVH is a common echocardiographic finding in Thai elderly (28-62%). Body weight and PP are major risk factors. These findings support weight reduction and PP control for prevention or regression of this condition. Indexing for BSA (LVM/BSA ≥117 g/m^2 in M, ≥104 g/m^2 in F) reduces LVH variability in underweight, normal weight and overweight subgroups as well as sexes.

Key word : LVH, LVMI, Echocardiogram, Thai Elderly

CHANTRA S & BHUTHONG B
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* Sakaeo Crown Prince Hospital, Ministry of Public Health, Sakaeo 27000, Thailand.

Left ventricular hypertrophy (LVH) is a major mechanism of cardiac adaptation to hemodynamic overloads on the heart or contractile failure of the myocardium[1,2]. Assessment of the presence and degree of LVH by accurate measurement of left ventricular mass (LVM) is therefore of great importance. Traditionally, electrocardiograph has been considered a reliable method and a more sensitive tool than chest roentgenography to detect LVH[3,4]. A large LVM is a significant predictor of cardiovascular morbidity in adults, independent of other established risk factors such as smoking, systolic blood pressure (SBP) and age[5,6]. In the Framingham study about 45 per cent of all cardiovascular deaths were preceded by LVH detected by electrocardiography (ECG-LVH), and the 5 year mortality rate in men with ECG-LVH was about 35 per cent, compared with 10-15 per cent in the absence of ECG-LVH[7,8]. LVH is the usual consequence of arterial hypertension, and it has been shown to appear within 12 years in about 50 per cent of patients with SBP above 180 mmHg[9].

Compared to ECG, echocardiography shows a higher sensitivity and an equally high specificity for the diagnosis of LVH[10]. Echocardiography allows quantitation of LVM and provides values reasonably near to those found at necropsy [11,12]. ECG allowed detection of LVH in no more than 38 per cent of hypertensive patients with echocardiographic LVH[13]. Some findings have suggested that echocardiographic LVH could be an independent predictor of cardiovascular morbidity[14] and mortality[15].

Race and sex may affect the relationships between LVM and cardiovascular disease in adults. Hypertensive black people develop more LVH than do hypertensive caucasians at similar levels of blood pressure, which may be partially responsible for the greater morbidity and mortality in hypertensive black people[16-19]. Therefore, the data from different races may not allow generalizability of the results. They need to be examined in a separate population.

Men have greater LVM than women, even when corrections are made for body size[6,16,17] and physical activity[17]. Recently, the concept of a multiple risk factor syndrome was introduced; this is usually defined as a metabolic disorder manifested by obesity, diabetes mellitus, hypertension, LVH, etc., in connection with hyperinsulinemia [20]. Screening for a high-risk population, characterised by this syndrome, is an effective strategy for the prevention of coronary artery disease. Increased LVM, which includes wall thickness and internal dimensions in its calculation, is also associated with hypertension.

The purpose of this investigation was to find the prevalence of and risk factors associated with LVH in a general population of Thai elderly men and women. Before this, however, no prospective data has been available for a precise assessment of prevalence of the seriousness of this finding as it occurs in the general Thai population.

METHOD

Study subjects:

Of the 445 registered elderly men and women around Sakaeo Crown Prince Hospital, 341 (71%) were found by survey. All elderly men and women aged 60 years and over were visited by a home health care team and referred to the echocardiographic unit at Sakaeo Crown Prince Hospital.

The study group consisted of 157 (46%) subjects who were referred from home health care and came to our echocardiographic unit where clinical evaluation, repeated blood pressure had been measured and clinical information had been accomplished. The population was unselected. Furthermore, only subjects who had technically satisfactory M mode echocardiogram were included.

Echocardiographic method

A protocol of the echocardiographic examination in brief, 30 minutes was allotted during an extensive clinical visit to obtain systematic M-mode and screening with two-dimensional (2D), spectral, and colour Doppler studies in each subject with a Toshiba SSA-380A Power vision cardiac ultrasound machine (Toshiba, Japan) and according to a standardised protocol. The studies were measured on line and recorded on VHS videotapes.

Echocardiographic Measurements

These were made from M-mode echocardiograms with 2D guide. Each subject was studied in the supine and left lateral decubitus position. Measurements were made according to the recommendations of the American Society of Echocardiography[21] (ASE). The internal dimension of the left ventricle at end-diastole was measured at the onset of the QRS complex, and the end-systolic dimen-

sion was measured at the nadir of septal motion (i.e., when the ventricular septum was farthest from the anterior chest wall). Both measurements made with the ultrasound beam passing at or just below the tip of the mitral valve leaflets only on high-quality tracings on which the right and left sides of the interventricular septum and the endocardial and epicardial surfaces of the posterior LV wall were recorded continuously throughout the cardiac cycle. Particular attention was paid to identifying the correct level for LV measurements from standard sweeps. Three measurements were made and averaged. ASE-cube LVM correlated to necropsy LVM ($r=0.9$, $p<0.001$), but systematically overestimated by a mean of 25 per cent; the overestimation could be corrected by the equation; LVM = 0.80 (ASE-cube LVM)+ 0.6 g, whereas, ASE-cube LVM = $1.04*((IVSD+LVIDD+PWD)^3 - LVIDD^3)$; IVSD is interventricular septal thickness at end-diastole, LVIDD is left ventricular internal dimension at end diastole, and PWD is posterior wall thickness at end diastole. Each of these structures had to be measurable for the study to be considered adequate. This formula has been anatomically validated, and values obtained using this formula were similar to those obtained with the Penn cube method[12].

In this report we selected the criteria widely used in cohort studies[22]. In the Framingham Heart Study, the means value ± 2 SD were 131 and 100 g/m^2 for men and women, respectively, for left ventricular mass indexed by body surface. The corresponding value was 143 and 102 g/m, respectively, for LVM/height, and 259 and 166 g, respectively, for an unindexed mass. The partition values reported by de Simone et al for[23] 97.5 percentile of LVM/body surface, LVM/height and LVM/height$^{2.7}$ were 117 g/m^2, 126 g/m and 50 g/m$^{2.7}$ for men and 104 g/m^2, 105g/m, and 47 g/m$^{2.7}$ for women, respectively, the nongender-specific upper limit of 51 g/m$^{2.7}$. In addition, the partition value of 125 g/m^2 for both men and women has been used for mass index by BSA in several studies.

Clinical features and groups:
Hypertension

Stage 2 hypertension was present at the baseline examination if the seated blood pressure (average of three readings) exceeded 159 mm Hg systolic or exceeded 99 mm Hg diastolic. Stage 1 hypertension was present if the average of the three-pressure readings was 140 to 159 mm Hg systolic and 90 to 99 mm Hg diastolic. A normotensive subject was SBP <140 and DBP <90 mm Hg[24].

Adiposity[25].

Body mass index (BMI) was selected as a measure of adiposity determined as BW (kg) divided by height squared (m^2). "Normal" weight was defined by BMI 20-27 kg/m^2; "overweight" as 27 to 30 kg/m^2; and "obesity" as > 30 kg/m^2. Underweight was defined BMI < 20 kg/m^2.

Why M mode?

As echocardiographic techniques have advanced from M mode to two-dimensional and, more recently, three-dimensional technique[26,27], it might be argued that this type of M mode research is rapidly becoming irrelevant. We believe that this is not so. Quantitative M mode echocardiography remains widely used in most echocardiography laboratories. Furthermore, M mode echocardiography still plays an important role in cardiovascular research. For example, M mode technique figured prominently in the method and tables of three recent articles on clinical hypertension and cardiac structure[26,28,29]. M mode technique was also used in a recent report from the Cardiovascular Health Study[30] and widely cited studies of physiologic hypertrophy in athletes[31,32]. Furthermore, M mode measurements have been found to be powerful predictors of cardiovascular prognosis[22]. Even if we used an echomachine specialised for cardiac examination it can apply to a general-purpose ultrasound machine, which is now ubiquitously used in general hospitals in Thailand without the need of Colour Doppler examination.

Statistical method.

All values for quantitative measures are expressed as mean value ± SD. Chi-square statistics were used to compare LVH prevalence and the unpaired Student's *t* test was used to test the mean difference between the subjects grouped when the variables had a normal distribution and Mann-Whitney U test was used for variables that had skewed distribution.

Univariate analysis. Pearson' correlation coefficients were obtained for relation between LVM and other variables.

Multivariate analysis. Clinical and demographic variables that exhibited significant univa-

riate correlation to LVM were evaluated by stepwise multiple regression with LVM. Variables assessed in this analysis were BW, BMI, SBP, PP, height, DBP and gender. The purpose of this analysis was to elucidate the relative importance of the different variables as predictors and potentially as determinants of left ventricular muscle mass[33].

RESULTS

Characteristic of participants.

M-mode echocardiographic studies of adequate quality to permit calculation of LVM were obtained in 125 (80%) of 157 participants in whom studies were attempted.

The clinical characteristics of the study subjects (mean±SD) are summarised in Table 1. The average age of the subjects was 67±7 yr; BW 55±11 kg; height 155±7 cm; PP 59±14 mmHg; male 53 (42%); BSA 1.5±0.2 m^2; BMI 23±4 kg/m^2; underweight 36 (29%); overweight 19 (15%);

Separate comparison in each blood pressure subgroup revealed that Stage 2 hypertensive subjects were younger, heavier, taller and had greater PP than normotensive and stage 1-hypertensive subjects. Stage 1 hypertensive subjects were heavier, had greater PP and were more overweight than normotensive subjects.

Echocardiographic findings:

The average IVSD was 11±2 mm; LVIDD 46±6 mm; LVPW 11±2 mm; LVM 176±49 g; LVM/BSA 115±29 g/m^2; LVM/Ht 113±31 g/m; LVM/$Ht^{2.7}$ 54±15 $g/m^{2.7}$.

No difference in LVIDD existed in any blood pressure group. Septal and posterior wall thickness were greater in stage1 and stage 2 hypertensive subjects than normotensive subjects but there was no difference between stage1 and stage 2 hypertensive subjects. Separate comparison of men and women in each blood pressure group revealed similar patterns of findings in the women subgroup but men showed no difference in any blood pressure group.

Prevalence of left ventricular hypertrophy. (Table 2)

Prevalence of LVH depended on the different types of indexation. LVH defined by 1) unindex LVM ≥259 g in men and ≥166 g in women was 35 (28%). Defined by LVM/BSA 2) LVM/

Table 1. Clinical and demographic findings in normotensive and hypertensive subjects.

	Normotensive	Stage 1 hypertension	Stage 2 hypertension	Total
Number (%)	72(58) †,‡	42(34) ‡,§	11(9) †,§	125(100)
Age (yr)	67±7†	67±6§	62±2 †, §	67±7
BW (kg)	52±9†,‡	56±12 ‡,§	65±13 † ,§	55±11
Height (cm)	156±8†	153±7§	161±7 †, §	155±7
Pulse pressure	50±9†, ‡	68±9 ‡, §	77±10 †, §	59±14
Male (%)	29(40)	17(41)	7(64)	53(42)
Body surface area	1.5±0.1†	1.5±0.2§	1.7±0.2†, §	1.5±0.2
BMI (kg/m^2)	21±3†	24±5	25±4 †	23±4
Underweight (%)	25(35) †	11(26)	0(0) †	36(29)
Overweight (%)	3(4) ‡	13(31) ‡	3(27)	19(15)
Echocardiographic data				
IVSD (mm)	10±2†, ‡	11±2 ‡	12±1 †	11±2
LVIDD (mm)	46±6	45±6	48±7	46±6
LVPWD (mm)	10±2†, ‡	11±2 ‡	12±2 †	11±2
LVM (g)	168±47†	179±50	216±50 †	176±49
LVMI/BSA (g/m^2)	112±28	117±30	129±29	115±29
LVMI/height (g/m)	108±29	117±33	134±30	113±31
LVMI/$Ht^{2.7}$ ($g/m^{2.7}$)	51±14	57±17	60±14	54±15

†: $p<0.05$ Normotensive *vs* Stage 2 hypertension.
‡: $p<0.05$ Normotensive *vs* Stage 1 hypertension.
§: $p<0.05$ Stage 1 hypertension *vs* Stage 2 hypertension.

Table 2. Prevalence of LVH defined by various left ventricular mass indexation and partition values.

	Normotensive	%	Stage 1 hypertension	%	Stage 2 hypertension	%	Total	%
Number (%)	72	100 †,‡	42	100 ‡,§	11	100 †,§	125	100
Defined by unindexes LVM								
1. ≥259 g in men, 166 g in women	16	22 †	13	31	6	55 †	35	28
Defined by LVM/BSA								
2. ≥131 g/m^2 in men ≥100 g/m^2 in women	32	44	24	57	7	64	63	50
3. ≥117 g/m^2 in men ≥104 g/m^2 in women	36	50	24	57	8	73	68	54
4 ≥125 g/m^2 in both men and women	23	32	14	33	6	55	43	34
Defined by LVM/height								
5. ≥143 g/m in men, ≥102 g/m in women	25	35	17	41	7	64	49	39
6. ≥126 g/m in men, ≥105 g/m in women	27	38 †	17	41	8	73 †	52	42
Defined by LVM/height$^{2.7}$								
7. ≥51 g/m$^{2.7}$ in both men and women	30	42 †	23	55	9	82 †	62	50
8. ≥50 g/m$^{2.7}$ in men ≥47 g/m$^{2.7}$ in women	39	54	29	70	9	82	77	62

†: p<0.05 Normotensive *vs* Stage 2 hypertension.
‡: p<0.05 Normotensive *vs* Stage 1 hypertension.
§: p<0.05 Stage 1 hypertension *vs* Stage 2 hypertension.

BSA ≥131 g/m^2 in men and ≥100 g in women was 63 (50%) 3) LVM/BSA ≥117 g/m^2 in men and ≥104 g/m^2 in women was 68 (54%) 4) LVM/BSA ≥125 g/m^2 in both men and women 43 (34%). Defined by LVM/height 5) LVM/height ≥143 g/m in men and ≥102 g/m in women 49 (39%) 6) LVM/height ≥126 g/m in men and ≥105 g/m in women 52 (42%). Defined by LVM/height$^{2.7}$ 7) ≥51 g/m$^{2.7}$ in both men and women 62(50%) 8) LVM/height$^{2.7}$ ≥50 g/m$^{2.7}$in men and ≥47 g/m$^{2.7}$ in women 77 (62%).

Separate comparison in each blood pressure group revealed a similar pattern in all indexation. Stage 2 hypertension showed higher prevalence than stage 1 and stage 1 higher prevalence than the normotensive group but attained statistical significance only in indexation for height (LVM/height ≥126 g/m in men and ≥105 g/m in women), height$^{2.7}$(LVM/height$^{2.7}$ ≥51 g/m$^{2.7}$ in both men and women) and in unindex LVM (LVM ≥259 g in men and ≥166 g in women) for stage 2 *vs* normotensive (Table 2).

Univariate correlates of left ventricular mass (Table 3); All studied variables except age exhibited statistical significance in univariate correlation with LVM in the total population of 125 subjects.

Table 3. Univariate correlates of left ventricular mass.

	Normotensive (n=72)	Stage 1 HT (n=42)	Stage 2 HT (n=11)	Total 125
SBP	*	ns	ns	**
DBP	*	ns	ns	*
Pulse pressure	ns	ns	ns	**
Age	ns	ns	ns	ns
Height	*	ns	ns	**
BW	**	**	ns	**
BMI	**	**	ns	**
Gender	*	ns	ns	*

ns: not significant
* Correlation is significant at the 0.05 level (2-tailed)
** Correlation is significant at the 0.01 level (2-tailed)
Value assigned to gender; male = 1, female = 2

Among 72 normotensive subjects BW, BMI, gender, DBP, height, SBP exhibited the strongest relations with LVM, but age and PP were not related. Separate analysis of normotensive men and women revealed that only BW and BMI were significant correlates of LVM in each group and age was also correlated in men but not in the women subgroup.

Among 42 subjects with stage 1 hypertension, BW and BMI were most closely correlated with LVM; no correlation was observed with SBP, DBP, PP, height, age and gender. Subdivision of stage 1 hypertensive subjects by gender revealed that LVM was predicted only by BW and BMI in women and by PP, DBP, BW, age and BMI among men.

Among 11 stage 2 hypertensive group, no variable exhibited significant relation with LVM. Separate analysis in women and men with stage 2 hypertensive subjects revealed only PP (r =0.974, p=0.026) to be the only statistically independent predictors of LVM in women, whereas, no variable contributed to the predictive equation among men.

The univariate analyses in the present study revealed strong, statistically independent associations between echocardiographically determined LVM and BW, BMI, SBP, PP, height, DBP in a large population of normotensive and hypertensive elderly. Increasing blood pressure subgroups showed less strong and less consistent relations with increasing LVM.

In multivariate analysis (Table 4). In all the 125 subjects, BW and PP independently predicted LVM in descending order of statistical significance. BMI, height, SBP, DBP and gender failed to contribute significantly to the predictive model. Among the entire group of 72 normotensive subjects only BW contributed independently to prediction of LVM. Separate analysis of normotensive women and men revealed BW to be the strongest predictor of LVM in women, whereas, BMI was the strongest in men. Among the entire group of 42 subjects with stage 1 hypertension, BW and PP were the strongest statistically independent predictors of LVM. Subdivision of stage 1 hypertensive subjects by gender revealed that LVM was predicted only by BMI among women and by PP and BW among men.

Table 4. Multivariate prediction of left ventricular mass.

	Normotensive (n=72)	Stage 1 HT (n=42)	Stage 2 HT (n=11)	Total 125
SBP	ns	ns	ns	ns
DBP	ns	ns	ns	ns
Pulse pressure	ns	*	ns	*
Age	ns	ns	ns	ns
Height	ns	ns	ns	ns
BW	**	**	ns	**
BMI	ns	ns	ns	ns
Gender	ns	ns	ns	ns

ns: not significant
* Correlation is significant at the 0.05 level (2-tailed)
** Correlation is significant at the 0.01 level (2-tailed)
Value assigned to gender; male = 1, female = 2

DISCUSSION

Indexation of left ventricular mass to body size[22]. Since the 19th century, many aspects of human metabolism and organ growth have been "normalised" by BSA. This indexation has subsequently been applied to cardiovascular measurements, including ventricular mass. Height was chosen as a measure of body size because it is obesity independent[34], and the linear growth determined by height was the major factor influencing LVM in children and young adults. Residual relations of LVM/BSA to BSA and of LVM/height to height were markedly reduced by normalisation of LVM for height$^{2.7}$. However, a weak inverse association between LVM/height$^{2.7}$ and height has been demonstrated, which may indicate possible overadjustment. In fact, intragroup variability of LVM was essentially the same regardless of the indexes used, except for height, where it was somewhat greater[41]. The present study shows that different methods of indexation had a minimal effect on the observed association between LVM and blood pressure except defined by LVM/height$^{2.7}$ where it was somewhat greater.

Predictive values of different methods of indexation: de Simone et al[35] reported that during follow-up, LVM indexed for BSA or for height consistently demonstrated that a higher proportion of subjects with LVH (23% to 29%) had a subsequent cardiovascular event than those without LVH (9% to 12%). Of all indexes examined, identification of the proportion of patients who had a morbid event with or without baseline LVH was slightly better with height-based than BSA indexes. Among gender-specific criteria, a higher odds ratio was obtained using LVM/height$^{2.7}$ (odds ratio 3.26) than LVM/BSA, LVM/height or LVM/height.$^{2.13}$ The latter three were virtually identical in performance to LVM/BSA (all odds ratio 2.50 to 2.66). Liao Y et al[22] reported high correlation among various body size indexes, LVH, defined by different indexes for LVM similarly confers an increased risk

of mortality in patients with or without coronary artery disease. No increased mortality was found for hypertrophy detected by height-based indexes in the absence of hypertrophy based on criteria indexed for BSA. These patients were usually overweight and had a lower degree of hypertrophy. Conversely, the only method of indexing that rendered statistically nonsignificant the predictive value of LVM/BW[35,36].

Comparison with previous studies: Echocardiographic LVH is a common finding, occurring in 28 per cent to 50 per cent by Framingham criteria of the present study. These rates are likely to overestimate the prevalence of this condition in the general population because the prevalence of echocardiographic LVH increases dramatically with advancing age from 6 per cent in persons less than 30 years of age to 43 per cent in those over 69. For every 10-year increment of age, the risk for LVH increases 15 per cent in men and 67 per cent in women. In elderly women the prevalence of LVH approaches 50 per cent[37]. These findings in a Thai elderly population confirm and modify conclusions concerning factors correlated with a potentially determined LVM that have been derived from previous studies in a selected clinical as well as a volunteer population[33,37]. Cross-sectional studies by several investigators have revealed significant univariate relations between LVM and BW or BSA, SBP and DBP measured either clinically or by ambulatory recorder, increasing age, male gender and estimated dietary sodium consumption[38]. Support for the possibility that several of these variables may be not merely correlates but actual determinants of LVM has been provided by studies in which changes in LVM in the expected direction were induced by weight gain, blood pressure reduction by restriction of dietary sodium or weight loss and treatment by antihypertensive medication, with a moderate correlation being observed between the mean changes in blood pressure and LVM in group data from published trials[39].

In most previous studies, LVM has been more closely correlated with SBP than DBP but did not include pulse pressure in their analyses[33,36]. Although our univariate analyses tend to confirm these previous observations, our multivariate analysis suggest that several of the aforementioned variables only appear to be associated with LVM because of covariabilitly with actual determinants of LVM, or exhibit associations with LVM that are so weak and inconsistent as to be practically negligible despite statistical significance. Although PP positive correlation observation is new, it is not entirely surprising in the light of previous data.

Studies of conduit vessel function have demonstrated that as the proximal elastic conduit vessels (aorta, carotids) dilate and stiffen with advancing age, pulse wave velocity and characteristic impedance increase and arterial compliance decreases. Pulse wave velocity and characteristic impedance increase more than double from the teens to the 50's in humans. In contrast, steady flow load (total peripheral resistance) increases only by about one third. Stiffening of conduit vessels is accelerated in the presence of hypertension, even in populations in which the prevalence of arteriosclerosis is low. Stiffening of the vessel wall leads to acceleration of pulse wave velocity and increased characteristic impedance. Because characteristic impedance dictates the PP response to an instantaneous change in flow in the proximal aorta, the early peak in central aortic pressure is enhanced even if peak flow is unchanged. Accelerated pulse wave velocity causes this enhanced forward pressure wave to travel more rapidly down the conduit vessels to reflecting sites that are created by branch points and by the abrupt impedance change that occurs at the level of the resistant vessels. Because peripheral resistance is increased in hypertension, a greater percentage of this already enhanced pressure wave is reflected back toward the heart. The reflected wave returns to the proximal aorta prematurely, in mid to late systole rather than in diastole, and augments PP and LV load while simultaneously diminishing diastolic pressure. This shifting of the reflected wave from diastole to systole has no effect on mean arterial pressure yet results in the highly unfavourable combination of an increase in myocardial oxygen demand and a decrease in coronary perfusion pressure. Increased PP increases pulsatile stress and promotes fracture of the elastic elements and dilation of the aorta. Dilation further increases stress and leads to a vicious cycle that culminates in vessel failure at points of structural weakness. This vicious cycle, leading to progressively increasing pulsatile load, likely has implications for the progressive increase in LVM that occurs in the setting of a stable increase in mean arterial pressure in long-standing hypertension[40].

As previous studies have shown, SBP is a better predictor of cardiovascular risk than DBP in

hypertensive patients aged ≥55 years. This is probably so because of the increasing importance of aortic stiffness with hypertension and aging, leading to disproportionate increases in SBP and the resulting effects on target organs. As increased aortic stiffness not only increases SBP, but also decreases DBP, PP would appear to be even better than SBP as an index of aortic stiffness. However, there has been only limited study of the role of PP in cardiovascular morbidity and mortality independent of MBP or DBP[41]. The relation among MBP, PP and cardiovascular risk was investigated in 18,336 men and 9,351 women, aged 40 to 69 years, living in Paris[42] in this study, PP alone was shown to be independently related to the degree of cardiac hypertrophy. Furthermore, in a 10-year survival analysis, PP was an independent predictor of cardiac mortality in women aged ≥55 years. The prognosis value of pretreatment PP was also evaluated in New York City as a predictor of MI in a union-sponsored hypertension control program[43] in which 2,207 patients with blood pressure >160/>95 mm Hg were grouped by tertile of PP. During an average follow-up interval of 5 years, 132 cardiovascular events (50 MIs, 23 strokes) were observed. Wide PP was identified as a predictor of MI. This finding was confirmed in a larger group of 5,730 participants[44] in whom a wide pretreatment PP (>63 mm Hg) was associated with subsequent cardiovascular complication. Wide PP with low DBP was also used to identify a subgroup of patients at greater risk of MI from either too large or too small a treatment induced decrease in DBP (J curve).

Taken together, these findings indicate that the brachial PP is a significant predictor of cardiovascular risk, especially LVH and MI. Both epidemiological and hemodynamic findings support the belief that increased arterial stiffness, the major contributor of disproportionate SBP over DBP, should be considered in future evaluations of cardiac and other target organ complications[41]. The present study supports this hypothesis.

Effect of indexing on detection of left ventricular hypertrophy. The use of one normalisation instead of another is likely to affect the results when subjects of different body size are compared. Normalisation of LVM for BSA yielded a significant difference between none obese and both mildly obese and moderately obese patients (both $p<0.001$), with 23 per cent mean increase in ventricular mass index in those with mild obesity. Preliminary analysis in hypertensive patients has shown that use of the allometric (or growth) signal for height detects a higher prevalence of LVH in overweight patients than does conventional indexing by BSA[45]. The present study does not support these findings. In the overweight subgroup (Table 5), by Framingham criteria, we found no difference in prevalence between normalisation of LVM for BSA or height. But conversely, LVM/BSA detects a higher prevalence of LVH in underweight and normal weight subjects although it did not attain statistical significant. Among the indexes studied, indexing for BSA ($\geq 117 g/m^2$ in M and $\geq 104 g/m^2$ in F) provide methods of normalising LVM for body size that reduces LVH variability in underweight, normal weight and overweight subgroups as well as sexes. (Table 5-6) Indexing of LVM for height and height$^{2.7}$ are less successful in reducing variability for prevalence of LVH among underweight, normal weight and overweight.

Clinical implications. The mechanisms by which LVH is associated with incidence of coronary heart disease are speculative. Increased LVM may promote coronary disease in a causal manner or may merely serve as a potent marker of risk. It represents a cardiac end-organ manifestation of hypertension and obesity, yet it predicts risk independent of these traditional risk factors. Increased LVM results in increased myocardial oxygen demand which, when coronary blood supply is fixed or compromised, may hasten the development of ischemia, infarction, and sudden cardiac death. It is also possible that increased LVM serves only as a marker for coronary heart disease, because LVH and coronary heart disease appear to share common risk factors. The limitations in predicting coronary disease incidence according to levels of traditional risk factors may reflect several interacting phenomena. In this aging cohort, secular trends in risk factors levels or in their relationship to coronary heart disease may have occurred, for example, the powerful relationship of blood pressure level to coronary heart diseases may no longer pertain in an era in which most hypertensive persons receive antihypertensive drug therapy, which in most instances resulted in hypertension control. This theory is suggested by the lack of association of SBP with coronary disease incidence in men, and only marginally significant association in women[46].

Table 5. Influence of BMI on the distribution of LVM normalised for body size.

	Under-weight	Normal weight	Over-weight	Total
Number	36	70	19	125
1. LVM ≥259 g in M ≥166 g in F	3(8) †, ‡	20(29) ‡,§	12(63) †,§	35(28)
Defined by LVM/BSA				
2) ≥131 g/m^2 in M ≥100 g/m^2 in F	13(36) †	37(53)	13(68) †	63(50)
3) ≥117 g/m^2 in M ≥104 g/m^2 in F	19(53)	37(53)	12(63)	68(54)
4) ≥125 in both M and F	9(25)	28(40)	6(32)	43(34)
Defined by LVM/height				
5) ≥143 g/m in M ≥102 g/m in F	7(19) †, ‡	29(41) ‡, §	13(68) †, §	49(39)
6) ≥126 g/m in M ≥105 g/m in F	5(14) †, ‡	34(49) ‡	13(68) †	52(42)
Defined by LVM/height$^{2.7}$				
7) ≥51 g/m$^{2.7}$ in M and F	10(28) †, ‡	38(54) ‡	14(74) †	62(50)
8) ≥50 g/m$^{2.7}$ in M ≥47 g/m$^{2.7}$ in F	16(44) †, ‡	45(64) ‡	16(84) †	77(62)

†: p<0.05 underweight *vs* overweight
‡: p<0.05 underweight *vs* normal weight
§: p<0.05 normal weight *vs* overweight.

Table 6. Influence of sex on the distribution of LVM normalised for body size.

	Male	Female	Total
Number (%)	53(100%)*	72(100%)*	125(100%)
Defined by unindexes LVM			
1. ≥259 g in men, 166 g in women	4(8) **	31(43)**	35(28)
Defined by LVM/BSA			
2. ≥131 g/m^2 in men ≥100 g/m^2 in women	16(30) **	47(65)**	63(50)
3. ≥117 g/m^2 in men ≥104 g/m^2 in women	27(51)	41(57)	68(54)
4. ≥125 g/m^2 in both men and women	21(40)	22(31)	43(34)
Defined by LVM/height			
5. ≥143 g/m in men, ≥102 g/m in women	9(17) **	40(56)**	49(39)
6. ≥126 g/m in men, ≥105 g/m in women	16(30)	36(50)	52(42)
Defined by LVM/height$^{2.7}$			
7. ≥51 g/m$^{2.7}$ in both men and women	25(47)	37(51)	62(50)
8. ≥50 g/m$^{2.7}$ in men ≥47 g/m$^{2.7}$ in women	27(51) *	50(69)*	77(62)

**p<0.01
* p<0.05

Abbreviations and Acronyms
BMI = Body mass index. BSA = Body surface area. BW = Body weight. SBP = Systolic blood pressure. DBP = Diastolic blood pressure. IVSD = interventricular septal thickness at end-diastole. LVM = left ventricular mass. LVIDD = left ventricular internal dimension at end diastole. MBP = mean blood pressure. PP = pulse pressure. PWD = posterior wall thickness at end diastole.
BSA (body surface area)= 0.007184 x BW$^{0.425}$ x Height$^{0.725}$

As previous Framingham report documented an association between ECG-LVH and risk for coronary heart disease, it was an infrequent finding, occurring in less than 3.2 per cent of this population[8]. The overall sensitivity of echocardiographic LVH for coronary events is far greater than that of ECG-LVH (46% compared with 3%) in men; 39 per cent compared with 9 per cent in women and occurs without associated loss of predictive value of a positive test compared to the ECG-LVH in men (17% for ECG-LVH compared to 17% for echocardiogram LVH). However, a decrease in predictive value of a positive test for the echocardiogram compared to ECG was noted in women (5% compared with 25%, reflecting the low overall incidence of coronary disease (approximately 1% per year and the high prevalence of echocardiographic LVH (35%) in women of this largely healthy elderly cohort)[46].

LVH denotes a serious prognosis in the course of hypertension or coronary heart disease. When LVH appears it should be regarded as a grave prognosis sign rather than an innocent compensatory phenomenon, because ECG-LVH may indicate myocardial ischaemia as well as hypertrophy. ECG-LVH merits preventive management against coronary disease, its major and most lethal squela. This is justified because the outlook for ECG- LVH closely resembles that of ECG evidence of myocardial infarction. In individuals with an adverse coronary risk profile, and ECG repolarization abnormality often signifies silent ischaemic myocardial involvement. Despite the lack of symptoms, this common ECG abnormality is associated with a high rate of development of overt manifestations of coronary heart disease. The serious prognosis of this silent ischaemia demands an active preventive regimen, which includes aspirin and beta-blockers. Risk factor modification and supervised exercise should be recommended[47]. The direct beneficial influence in LVH of the reduction of BW in overweight individuals has been demonstrated in a randomized controlled trial of young, overweight hypertensive patients[48]. LVM was reduced independently of changes in blood pressure. Thus, weight reduction can be recommended in overweight hypertensive patients as a means not only of lowering the blood pressure but also for the prevention or correction of LVH.

There are many unresolved issues concerning the pathogenesis and treatment of LVH. Antihypertensive therapy and weight control have been shown to at least partly reverse hypertrophy. It is not clear, however, whether the remaining myocardium becomes functionally improved and whether some drugs are more efficacious for this purpose than others. More data are also needed on whether reverse ECG-LVH and anatomical hypertrophy by antihypertensive therapy or weight reduction substantially improves the patient's prognosis. The seriousness of LVH requires, however, that all information is used in a rational, effective manner.

Study limitation. However, it is important to note several limitations. Clinically important mitral and aortic regurgitation usually results in LV dilatation. We did not systematic examination with Doppler to determine the presence and severity of coincidental mitral regurgitation (MR) and aortic regurgitation (AR). However, we detected AR 15 (12%), MR 4 (3.2%), MS 1 (0.8%), others 3 (2.4%) subjects during examination by color Doppler and Spectral screening. All subjects were included in each subgroup for analysis. Those who take antihypertensive drug(s) were included in each blood pressure group according to blood pressure recorded during the study.

SUMMARY

The presence of LVH adds substantially to the risk of cardiovascular disease in particular, at any level of other predisposing risk factors. The prevalence of LVH in general Thai elderly (28-62%) is very common in echocardiographically findings. BW and PP are major risk factors. However, it is clinically significant although not closely examined in this country. In the United States, it was found that from epidemiological observations that the risk of coronary heart disease in individuals with ECG-LVH varies over more than a 10-fold range depending on the number of coexisting risk factors. LVH defined by different indexes for LVM similarly confers an increased risk of mortality in patients with or without coronary heart disease (odds ratio 2.50 to 2.66). This offers the possibility for reducing the risk factors for coronary heart disease. Thai elderly are different from caucasians and black people, they are more underweight and less overweight. Even in overweight LVM/BSA can detect the prevalence of LVH equal to index for height and

detect LVH more in underweight and normal weight. Indexing for BSA (LVM/BSA $\geq$117 g/m^2 in M, $\geq$104 g/m^2 in F) reduces the prevalence of LVH variability in underweight, normal weight and overweight subgroups as well as sexes.

Clearly, we have more to learn about the role of LVH in the Thai population. However, enough is known to assign a high priority to detection, prevention and correction of LVH in preventive cardiology.

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ความชุกและปัจจัยเสี่ยงที่ทำให้เกิดหัวใจเว็นตริเกิ้ลซ้ายโตในผู้สูงอายุไทย: สำรวจด้วยการตรวจภาพคลื่นเสียงสะท้อนจากหัวใจ

สมรัก จันทรา, พ.บ., บรรหาร ภู่ทอง, พ.บ*.*

บทนำ: ยังไม่มีการสำรวจหาความชุกของการเกิดหัวใจเว็นตริเกิ้ลซ้ายโต (left ventricular hypertrophy) (LVH) และปัจจัยเสี่ยงที่ทำให้เกิดในคนไทยทั่วๆไป ปัจจัยเสี่ยงที่ทำให้เกิด LVH อาจเกิดจาก เชื้อชาติ, อ้วน, อายุ, เพศ และความดันโลหิตสูง การศึกษาในเชื้อชาติหนึ่งไม่สามารถนำมาใช้ในอีกชาติหนึ่งได้ ต้องการการศึกษาโดยเฉพาะในแต่ละเชื้อชาติ

วัตถุประสงค์: เพื่อหาความชุกและปัจจัยเสี่ยงที่ทำให้ เกิด LVH สำรวจด้วยการตรวจภาพคลื่นเสียงสะท้อนจากหัวใจ ในผู้สูงอายุไทยชายและหญิง

วิธีการศึกษา: ผู้สูงอายุ 157 คนในเขตรับผิดชอบของโรงพยาบาลสมเด็จพระยุพราชสระแก้วได้รับการตรวจภาพคลื่นเสียงสะท้อนจากหัวใจ คำนวณหามวลของเว็นตริเกิ้ลซ้าย โดยใช้สูตร LVM = 0.8x{1.04x[(IVSD+LVID+PWD)^3-LVID^3]}+0.6 g วิเคราะห์ความสัมพันธ์ของตัวแปรต่างๆโดย univariate และ multivariate analysis

ผลการศึกษา: จากผู้สูงอายุ 157 คน สามารถตรวจด้วยคลื่นสะท้อนเสียงหัวใจได้คุณภาพเพียงพอ 125 คน (80%) ความชุกของ LVH ขึ้นอยู่กับค่าจำกัดความต่างๆดังนี้ 1) unindexes LVM (≥259 กรัม ในชาย (M), 166 กรัม ในหญิง (F) คือ 35 (28%); 2) จำกัดความโดย LVM/BSA (≥131 กรัม/เมตร^2 ใน M, ≥100กรัม/เมตร^2 ใน F) 63 (50%); 3) (≥117 กรัม/เมตร^2 ใน M, ≥104 กรัม/เมตร^2 ใน F) 68 (54%), 4) (≥125 กรัม/เมตร^2 ใน M และ F) 43 (34%); 5) จำกัดความโดย LVM/ความสูง (≥143 กรัม/เมตร ใน M และ ≥102 กรัม/เมตร ใน F) 49 (39%); 6) (≥126 กรัม/เมตร ใน M และ ≥105 กรัม/เมตร ใน F) 52 (42%); 7) จำกัดความโดย LVM/height$^{2.7}$ (≥51 กรัม/เมตร$^{2.7}$ ใน M และ F) 62(50%); 8) (≥50 กรัม/เมตร$^{2.7}$ ใน M, ≥47 กรัม/เมตร$^{2.7}$ ใน F) 77 (62%). ในประชากรทั้งหมด ตัวแปรอิสระที่มีอิทธิพลต่อ LVH เรียงลำดับตามนัยยะทางสถิติคือ BW, BMI, SBP, pulse pressure, ($p < 0.01$), ขณะที่ DBP และเพศมีนัยยะสำคัญน้อยกว่าและอายุไม่มีความสัมพันธ์ การวิเคราะห์ความถดถอยเชิงซ้อนพบว่า BW, Pulse pressure เท่านั้นที่มีความสัมพันธ์ทางสถิติกับ LVM

สรุป: LVH เป็นปัจจัยเสี่ยงที่ตรวจพบได้บ่อยในผู้สูงอายุไทยมีความชุก 28-62% ตามการใช้ดัชนีต่างๆ ปัจจัยเสี่ยงที่สัมพันธ์กับการเกิด LVH คือน้ำหนักตัวและ pulse pressure การวิจัยครั้งนี้สนับสนุนการควบคุมน้ำหนักและ pulse pressure ในการป้องกันและควบคุมการเกิด LVH การใช้ดัชนี LVM/BSA (≥117 กรัม/เมตร^2 ใน M, ≥104 กรัม/เมตร^2 ใน F) สามารถลดความผันแปรของ LVH ในการจำแนกตาม BMI และเพศ

คำสำคัญ : LVH, LVMI, Echocardiogram, Thai Elderly

สมรัก จันทรา, บรรหาร ภู่ทอง
จดหมายเหตุทางแพทย์ ฯ 2543; 83: 1082-1094

* โรงพยาบาลสมเด็จพระยุพราชสระแก้ว, กระทรวงสาธารณสุข, สระแก้ว 27000