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ORIGINAL ARTICLE

Home-measured blood pressure is more strongly associated with electrocardiographic left ventricular hypertrophy than is clinic blood pressure: the Finn-HOME study

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Electrocardiographic evidence of left ventricular hypertrophy (ECG-LVH) has a grave prognostic significance in hypertensive patients. The purpose of our study was to assess whether ECG-LVH is more strongly associated with home-measured blood pressure (BP) than with clinic BP, and whether the correlation between home BP and ECG-LVH increases with the number of home measurements performed. We studied a representative sample of the general adult population (1989 subjects 45–74 years of age) in Finland. Subjects included in the study underwent a clinical interview, electrocardiography and measurement of clinic BP (mean of two clinic measurements) and home BP (mean of 14 duplicate home measurements performed during 1 week). Home BP correlated significantly better than clinic BP with the Sokolow–Lyon voltage (home/clinic systolic: $r=0.23/0.22$, $P=0.60$; diastolic: $r=0.17/0.12$, $P=0.009$), Cornell voltage (systolic: $r=0.30/0.25$, $P=0.004$; diastolic: $r=0.21/0.12$, $P<0.001$) and Cornell product (systolic: $r=0.30/0.24$, $P=0.001$; diastolic $r=0.22/0.14$, $P<0.001$) criteria of ECG-LVH. The correlation between home BP and ECG-LVH increased slightly with the number of home measurements, but even the mean of the initial two home BP measurements correlated equally well (systolic BP), or better (diastolic BP) with ECG-LVH than did clinic BP. In conclusion, home BP measurement allows us to obtain a large number of measurements that have a strong association with ECG-LVH. Our data support the application of home BP measurement in clinical practice.
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Introduction

Self-measurement of blood pressure (BP) is becoming increasingly popular. Compared with clinic BP, home BP provides better reproducibility,[1] the absence of the white-coat effect, and the lack of digit preference and observer bias. Most importantly, recent studies have shown that home BP has a stronger predictive power for future cardiovascular mortality and morbidity than clinic BP.[2–4]

Left ventricular hypertrophy (LVH) is an independent risk factor for increased cardiovascular mortality.[5] LVH is linearly related to the level of BP, and can eventually lead to congestive heart failure, of which approximately half is caused by hypertension.[6] Some studies have already reported that home BP is more strongly associated with the degree of LVH as determined by echocardiography.[7,8] However, echocardiography is not widely available and despite of a poor sensitivity, electrocardiograms are often the only method available for assessing LVH in the hypertensive patient, especially in typical primary care settings. Current data also support the use of Cornell product and Sokolow–Lyon voltage criteria to identify patients with hypertension who are most likely to benefit from aggressive antihypertensive therapy and suggest that serial evaluation of these criteria during treatment can be used to monitor risk.[9]

The treating physician should use a BP measurement method that provides a good image of the patient's true BP level, and also has a strong correlation with electrocardiographic evidence of

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LVH (ECG-LVH) to facilitate risk assessment and the adjustment of antihypertensive therapy. Several studies have already reported that the association between BP and ECG-LVH is stronger when BP is measured with ambulatory monitoring than with clinic measurements.[7,10,11] As far as we know, only one study with merely 38 selected hypertensive subjects has compared the relations of clinic and home BP with ECG-LVH.[7]

The purpose of our study was to assess in an unselected nationwide population whether ECG-LVH is more strongly associated with home-measured BP than with clinic BP at the population level, and whether the correlation between home BP and ECG-LVH increases with the number of home measurements performed.

Methods

Subjects

The study sample for the Finnish home BP monitoring study (Finn-HOME study) was drawn from the participants of a multidisciplinary epidemiological survey, the Health 2000 study, which was carried out in Finland from fall 2000 to spring 2001. The study population was a stratified two-stage cluster sample of 8028 subjects drawn from the population register to represent Finnish adults aged 30 years or over. The stratification and sampling procedures have been described previously in detail.[12]

Of the subjects aged 45–74 years ($n=4388$), 84% ($n=3672$) agreed to participate in the interview and attended the health examination. In the present study, analyses were restricted to subjects who had, in addition to the Health 2000 survey, participated in the home BP measurement substudy (Finn-HOME study) for persons aged 45–74 years ($n=2120$). Home measurement of BP was not performed on all subjects willing to participate due to the limited number of home monitors (approximately 800). Thus, study subjects willing to participate in the home BP measurement substudy were practically randomly selected on the basis of monitor availability. Subjects who had not performed $\geqslant 14$ valid home measurements of BP ($n=56$), had missing clinic BP values ($n=16$), ECG measurements ($n=34$), or intraventricular conduction abnormalities ($n=47$) were excluded from the study. After removing subjects with one or more exclusion factors, the final study population consisted of 1989 subjects of 45–74 years of age.

The study protocol of the Health 2000 survey was approved by the Epidemiology Ethics Committee of the Helsinki and Uusimaa hospital region, and all participants gave signed informed consent.

Flow of the study

At an initial health interview at the subject's home, basic background and socio-demographic characteristics, information about health, illnesses and use of medication were gathered by centrally trained interviewers. A total of 2120 subjects from 45 to 74 years of age who were willing to participate in the home measurement substudy received home BP monitors for measuring BP at home during the week following the health interview.

A physical examination was performed on each subject 1–6 weeks after the health interview at a local health centre by centrally trained doctors and nurses. Each subject's height, weight, body circumference and clinic BP were measured, and an ECG was recorded. Details of the methodology of the project have been published elsewhere.[13]

BP measurements

Clinic BP was measured by a nurse with a conventional, calibrated, mercury sphygmomanometer from the sitting person's right arm after a 10-min rest. The last 5 min of rest were spent in the measuring room with the cuff around the right upper arm. BP was measured using a pressure cuff of appropriate size and methods that were in accordance with current guidelines.[14] Systolic BP and diastolic BP were defined according to Korotkoff sounds I and V. Means of two measurements performed at a 2-min interval were used to determine clinic BP. Clinic heart rate was determined before BP measurement by palpation of the radial pulse over 30 s.

Home BP was self-measured with a validated, automatic oscillometric device (Omron model HEM-722C, Omron Corporation, Tokyo, Japan)[15] according to the current guidelines.[16] Subjects received written instructions and individual guidance on how to measure BP correctly. Preparations for self-measurement of BP were the same as for clinic BP. Seated BP was measured twice, approximately at a 2-min interval every morning between 0600 and 0900 and every evening between 0600 and 0900 on seven consecutive days. Home BP was determined as the mean of 14 duplicate measurements (28 measurements). The mean number of home BP measurements was 27.0 ± 2.8.

ECG measurements

Standard resting 12-lead ECGs were digitally recorded by using a Marquette MAC 5000 device and stored as digital data on a Marquette MUSE CV 5B system (Marquette Hellige, Milwaukee, WI, USA). All ECGs were overread, and the computerized diagnoses and measurements corrected if needed, by a single physician experienced with electrocardiography (HK) before being stored into the database. QRS duration was measured to the nearest 4 ms and the QRS amplitudes to the nearest 0.5 mm. ECG-LVH was assessed with three commonly used ECG criteria: (1) the Sokolow–Lyon voltage ($S_{V1}+R_{V5/6}$),[17] 2) the Cornell voltage ($R_{aVL}+S_{V3}$, plus

6 mm for women),[18,19] and (3) the Cornell product (Cornell voltage × QRS duration)[20] as indicators of ECG-LVH. Threshold values of 35 mm, 26 mm and 2440 mm × ms were used to identify LVH using the Sokolow–Lyon, Cornell voltage and Cornell product criteria, respectively.

Statistical analyses

Data are reported as mean ± standard deviation. The difference between home BP and clinic BP was compared by paired *t*-test. Pearson's correlation was used to calculate correlation coefficients for BP parameters and LVH-ECG. Testing equality of two correlations was carried out in a LISREL model (LISREL, version 8.54; SSI Inc., Chicago, IL, USA) by using the χ^2 difference test for correlation matrices with and without the equality constraint.[21] Database management and other statistical analyses were performed with SAS software (version 9.1; SAS Institute, Cary, NC, USA). *P*-value <0.05 was considered statistically significant.

Results

Subject characteristics

Subject characteristics are reported in Table 1. Characteristicts of the the Finn-HOME study population were very similar to the Finnish 45–74-year-old general population, as previously reported.[12] Clinic systolic (137.3 ± 20.1 mm Hg, range 89.0–240.5) and diastolic BP (83.7 ± 10.6 mm Hg, range 35.0–124.0) were significantly higher ($P<0.001$ for all) than home systolic (129.6 ± 18.7 mm Hg, range 87.1–208.4) and diastolic BP (80.3 ± 9.4 mm Hg, range 57.4–114.7).

Table 1 Characteristics of subjects included in the study

Characteristic	*Study population* (n = *1989*)
Age (years)	56.2 (8.4)
Male (%)	46.2
Body mass index (kg/m^2)	27.4 (4.5)
Obesity (%)	24.3
Diabetes (%)	6.4
Smokers (%)	19.8
Treated for hypertension (%)	22.6
Office systolic BP (mm Hg)	137.3 (20.1)
Diastolic BP (mm Hg)	83.7 (10.6)
Home systolic BP (mm Hg)	129.6 (18.7)
Diastolic BP (mm Hg)	80.3 (9.4)
Sokolow–Lyon voltage (mm)	25.2 (7.4)
Cornell voltage (mm)	18.8 (5.7)
Cornell product (mm × ms)	1707 (592)

Abbreviations: BP, blood pressure; s.d., standard deviation.
Values expressed as mean (s.d.). Obesity was defined as a body mass index ⩾30 kg/m^2. Diabetes was defined as a serum fasting glucose ⩾7.0 mmol/l, and/or the use of antidiabetic medication.

Association between ECG-LVH and BP measured at home and in the clinic

The correlation coefficients for home or clinic BP and ECG-LVH are presented in Table 2. Both home and clinic BP were significantly associated with ECG-LVH ($P<0.001$ for all correlations). Home BP, however, correlated significantly better with ECG-LVH than did clinic BP, except for the association between systolic BP and the Sokolow–Lyon voltage (Table 2). Even the mean of the initial two home BP measurements correlated equally well (systolic BP), or better (diastolic BP) with ECG-LVH than did clinic BP (Table 2). ECG-LVH was mainly more closely associated with BP in women and in subjects with no antihypertensive medication (Table 3). The Cornell voltage and Cornell product were more closely associated with BP than the Sokolow–Lyon voltage, especially for home BP. Home systolic BP was significantly higher in the evening than in the morning (131.2 ± 18.8/80.3 ± 9.4 mm Hg vs 127.9 ± 19.5/80.3 ± 9.9 mm Hg, $P<0.001$ for systolic and $P=0.89$ for diastolic), but both morning and evening home BPs correlated equally well with ECG-LVH ($P>0.05$ for all comparisons; data not shown).

The risk ratios of ECG-LVH were calculated to assess whether home hypertension (home BP ⩾135/85 mm Hg) poses a higher risk than clinic hypertension (clinic BP ⩾140/90 mm Hg).[16] Subjects on antihypertensive medication ($n=450$) were excluded from this analysis to avoid any confounding effects. An equal share of home and clinic hypertensives met the criteria for ECG-LVH as reported in Table 4. No difference was seen in the risk for the presence of ECG-LVH between clinic and home hypertensives. The prevalence and risk of ECG-LVH were slightly, but not significantly higher in subjects who had elevated home and clinic BP.

Number of home BP measurements and association between home BP and ECG-LVH

To investigate further how the number of home BP measurements affects the association between home BP and ECG-LVH, the relationship between the number of days of home BP measurement and the correlation between home BP and ECG-LVH was evaluated (Figure 1). The correlation coefficients increased only slightly with the number of measurements, especially for diastolic BP. Exclusion of measurements performed during day 1 from mean home BP did not result in a higher correlation coefficients, and the best possible correlation between systolic and diastolic BP and ECG-LVH was achieved by using the mean of all measurements as home BP.

Discussion

In this study with a representative sample of a nationwide adult population aged 45–74 years, we

Table 2 Correlation coefficients for home/office BP and ECG parameters

ECG parameter		*Systolic BP*			*Diastolic BP*		
		Clinic	*Home week*	*Home 2*	*Clinic*	*Home week*	*Home 2*
Sokolow–Lyon	Pearson's *r*	0.22	0.23	0.21	0.12	0.17	0.16
	P-value	—	0.60	0.17	—	0.009	0.05
Cornell voltage	Pearson's *r*	0.25	0.30	0.28	0.12	0.21	0.20
	P-value	—	0.004	0.14	—	<0.001	<0.001
Cornell product	Pearson's *r*	0.24	0.30	0.27	0.14	0.22	0.21
	P-value	—	0.001	0.10	—	<0.001	<0.001

Abbreviation: BP, blood pressure.
All correlations were statistically significant ($P<0.001$). *P*-value for the difference between clinic and home correlation; home week, mean of all home BP measurements included in the analysis; home 2, mean of the two initial home measurements included in the analysis.

Table 3 Group differences in correlations between home/clinic BP and ECG-LVH

Characteristic	*Sokolow–Lyon*				*Cornell voltage*				*Cornell product*			
	Systolic		*Diastolic*		*Systolic*		*Diastolic*		*Systolic*		*Diastolic*	
	Clinic	*Home*	*Clinic*	*Home*	*Clinic*	*Home*	*Clinic*	*Home*	*Clinic*	*Home*	*Clinic*	*Home*
Gender												
Male ($n=919$)	0.20	0.14**	0.06	0.06**	0.23*	0.28**	0.16	0.20**	0.22	0.26	0.14**	0.17**
Female ($n=1070$)	0.23	0.26	0.10	0.20	0.32	0.43	0.21	0.38	0.28	0.18	0.38	0.33
AH treatment												
Yes ($n=450$)	0.17	0.13*	0.06	0.04**	0.15	0.27	0.00*	0.10*	0.14	0.27	0.01**	0.11*
No ($n=1539$)	0.24	0.26	0.13	0.20	0.24	0.26	0.13	0.20	0.23	0.26	0.15	0.21

All values are reported as Pearson's correlations. Asterisk indicates $P<0.05$, double asterisk indicates $P<0.01$ in difference between groups. AH treatment, subjects on antihypertensive medication.

Table 4 Proportion of unmedicated subjects ($n=1539$) with ECG diagnosis

ECG criteria	*Home BP (%)*		*Clinic BP (%)*		*Home and clinic BP (%)*		*Risk ratios (95 % Cl)*		
	NT (n = 1005)	*HT (n = 534)*	*NT (n = 895)*	*HT (n = 644)*	*NT (n = 1127)*	*HT (n = 412)*	*Home HT*	*Clinic HT*	*Home and clinic HT*
Cornell product ⩾2440 mm ms	6.4	14.0	5.7	14.1	6.7	16.0	2.3 (1.7–3.1)	2.5 (1.8–3.4)	2.6 (1.9–3.7)
Cornell voltage ⩾26 mm	5.2	15.0	4.9	13.7	5.8	16.3	2.9 (2.1–4.0)	2.8 (2.0–3.9)	3.2 (2.2–4.6)
Sokolow–Lyon ⩾35 mm	6.9	16.7	7.2	14.6	7.7	17.2	2.4 (1.8–3.3)	2.0 (1.5–2.8)	2.5 (1.8–3.5)

Abbreviations: BP, blood pressure; H, home; HT, hypertensives; NT, normotensives; ECG-LVH, electrocardiographic evidence of left ventricular hypertrophy.
Values reported as % of subjects with ECG diagnosis for LVH. Home hypertension was defined as a home BP ⩾135/85 mm Hg and clinic hypertension as a clinic BP ⩾140/90 mm Hg. Risk ratios are reported as clinic or home hypertensives' risk for ECG-LVH.

have shown that home BP measured during 1 week has a stronger association with ECG-LVH than does clinic BP. The correlation between home BP and ECG-LVH increases only slightly with the number of home measurements, and even the mean of the initial two home BP measurements correlates equally well (systolic BP), or better (diastolic BP) with ECG-LVH than does clinic BP. Home and clinic hypertensives have a similar risk for ECG-LVH when using currently recommended diagnostic thresholds for home and clinic BP. BP was mainly more closely associated with ECG-LVH in women, and in subjects without antihypertensive medication.

BP was mainly more closely associated with ECG-LVH in women than in men. Previous studies have noted that there are gender differences in LVH and its determinants. In a study with 851 unselected subjects, only women showed significant correlations between BP and left ventricular mass determined by echocardiography.[22] Although BP

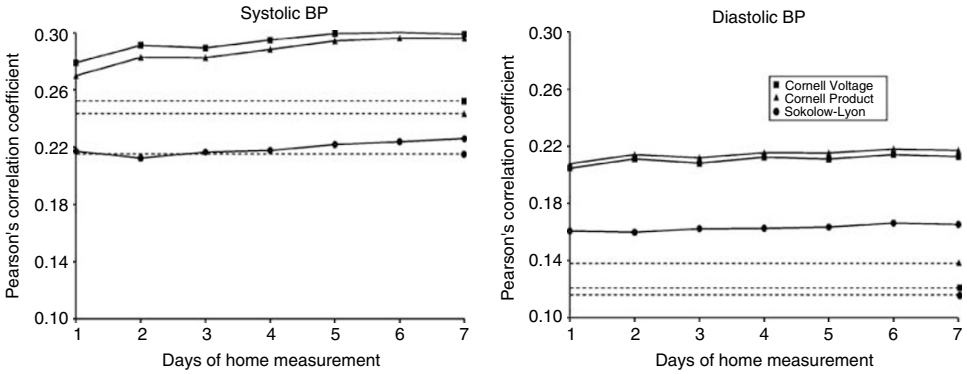

Figure 1 The relationship between the number of days of home BP measurement and the correlation between home BP and ECG-LVH. Dashed lines indicate correlation between clinic BP and ECG-LVH.

overload appears to be a major trigger of LVH, obesity, high sodium intake and male gender are independent determinants of anatomic LVH and may reduce the independent association between BP and LVH especially in men.[23,24] Previous studies have also shown that obesity decreases the likelihood of ECG-LVH by Sokolow–Lyon criteria.[25,26] Our finding of low correlations between BP and ECG-LVH by Sokolow–Lyon voltages particularly in men further confirms that other ECG criteria, such as the Cornell voltage or product, should be used in assessing ECG-LVH. Antihypertensive medication also has a confounding effect on the relationship between BP and ECG-LVH, demonstrated by the lower correlations of the medicated subjects.

Home hypertensives had a similar risk for ECG-LVH than did clinic hypertensives. The currently recommended 5 mm Hg lower BP thresholds for home BP measurement therefore seem to be appropriate, at least in terms of ECG-LVH prevalence and risk.[27,28] The prevalence and risk of ECG-LVH were slightly, but not significantly higher in subjects who had elevated home and clinic BP. This finding is in line with the results of the PAMELA study, as the overall ability to predict death increased by the combination of clinic and home values.[4]

In our study, the association between ECG-LVH and home BP increased slightly with the number of home measurements. It has also been previously demonstrated in the Japanese Ohasama study that the predictive value for stroke risk increases progressively with the number of home measurements.[29] The current European guidelines for home BP measurement recommend 7 days of home measurement, which appears to be sufficient, as no additional increase in the correlation between home BP and ECG-LVH was achieved after day 6 in our study.[27]

The greater number of readings, which can be obtained in a practical way with home BP measurement, contributes to a better diagnostic accuracy compared with clinic BP measurement. However, because of better reproducibility,[30] the absence of the white-coat effect, and the lack of digit preference and observer bias, the benefits of home BP measurement are not only limited to the greater number of measurements. In the Ohasama study, the two initial home BPs were more closely associated with cardiovascular risk than the two initial clinic BPs.[29] In addition, in the PAMELA study only two home readings were obtained, and these were stronger predictors of cardiovascular risk than six clinic measurements.[4] As demonstrated by these studies and our study, home BP is strongly associated with cardiovascular outcomes and target-organ damage, even with a low number of home measurements.

There are some limitations in our study. First, clinic BP, although very meticulously assessed, was measured on day 1 only and duplicate home BP readings were performed two times daily for 7 days. Therefore, we cannot exclude the possibility that taking clinic BP values over multiple days could have increased the association between clinic BP and ECG-LVH. However, even the mean of the initial two home BP measurements correlated equally well (systolic BP), or better (diastolic BP) with ECG-LVH than did clinic BP. Furthermore, home BP measurement always produces a higher number of BP readings than clinic measurement in reality. Second, because of the cross-sectional nature of our study, no cause–effect relationships can be drawn from our findings. Third, clinic BP was measured with a mercury sphygmomanometer and home BP with an automated oscillometric device, but this situation reflects reality in Finland, where clinic BP is still mainly measured with a mercury sphygmomanometer. Nevertheless, repeating this study with the same measurement method and number of measurements for home and clinic BP is necessary to validate our findings and to provide more conclusive results.

In conclusion, home BP measurement allows us to obtain a large number of measurements that have a strong association with ECG-LVH. Hypertension is mainly treated by primary care physicians who have to focus on the probability of LVH, but often have neither the facilities nor the budget to perform

ambulatory BP monitoring or an echocardiogram on their patients. Physicians should be aware that the use of home BP measurement could enhance the prediction of LVH and facilitate patient selection for more sophisticated tests. One week of home measurement, as recommended by the current guidelines, seems to be sufficient for the optimal assessment of an individual's BP level. Our data support the application of home BP measurements in clinical practice.

What is known about topic

- ECG-LVH has a grave prognostic significance in hypertensive patients
- No studies have compared the association between home BP and ECG-LVH in an unselected nationwide population

What this study adds

- Home BP is more strongly associated with ECG-LVH than is clinic BP
- The association between ECG-LVH and home BP increases slightly with the number of measurements
- One week of home measurement, as recommended by the current guidelines, seems to be sufficient for the optimal assessment of an individual's BP level

Abbreviations: BP, blood pressure; ECG-LVH, electrocardiographic evidence of left ventricular hypertrophy.

References

1 Stergiou GS, Baibas NM, Gantzarou AP, Skeva II, Kalkana CB, Roussias LG *et al.* Reproducibility of home, ambulatory, and clinic blood pressure: implications for the design of trials for the assessment of antihypertensive drug efficacy. *Am J Hypertens* 2002; **15**: 101–104.
2 Ohkubo T, Imai Y, Tsuji I, Nagai K, Kato J, Kikuchi N *et al.* Home blood pressure measurement has a stronger predictive power for mortality than does screening blood pressure measurement: a population-based observation in Ohasama, Japan. *J Hypertens* 1998; **16**: 971–975.
3 Bobrie G, Chatellier G, Genes N, Clerson P, Vaur L, Vaisse B *et al.* Cardiovascular prognosis of 'masked hypertension' detected by blood pressure self-measurement in elderly treated hypertensive patients. *JAMA* 2004; **291**: 1342–1349.
4 Sega R, Facchetti R, Bombelli M, Cesana G, Corrao G, Grassi G *et al.* Prognostic value of ambulatory and home blood pressures compared with office blood pressure in the general population. Follow-up results from the Pressioni Arteriose Monitorate e Loro Associazioni (PAMELA) study. *Circulation* 2005; **111**: 1777–1783.
5 Kannel WB. Prevalence and natural history of electrocardiographic left ventricular hypertrophy. *Am J Med* 1983; **75**: 4–11.
6 Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from hypertension to congestive heart failure. *JAMA* 1996; **275**: 1557–1562.
7 Mule G, Caimi G, Cottone S, Nardi E, Andronico G, Piazza G *et al.* Value of home blood pressures as predictor of target organ damage in mild arterial hypertension. *J Cardiovasc Risk* 2002; **9**: 123–129.
8 Tsunoda S, Kawano Y, Horio T, Okuda N, Takishita S. Relationship between home blood pressure and longitudinal changes in target organ damage in treated hypertensive patients. *Hypertens Res* 2002; **25**: 167–173.
9 Okin PM, Devereux RB, Jern S, Kjeldsen SE, Julius S, Nieminen MS *et al.* Regression of electrocardiographic left ventricular hypertrophy during antihypertensive treatment and the prediction of major cardiovascular events. *JAMA* 2004; **292**: 2343–2349.
10 Sokolow M, Werdegar D, Kain HK, Hinman AT. Relationship between level of blood pressure measured casually and by portable recorders and severity of complications in essential hypertension. *Circulation* 1966; **34**: 279–298.
11 Parati G, Pomidossi G, Albini F, Malaspina D, Mancia G. Relationship of 24-h blood pressure mean and variability to severity of target-organ damage in hypertension. *J Hypertens* 1987; **5**: 93–98.
12 Niiranen TJ, Jula AM, Kantola IM, Reunanen A. Prevalence and determinants of isolated clinic hypertension in the Finnish population: the Finn-HOME study. *J Hypertens* 2006; **24**: 463–470.
13 Aromaa A, Koskinen S. Health and Functional Capacity in Finland. *Baseline Results of the Health 2000 Health Examination Survey*. National Public Health Institute: Helsinki, 2004. Available at: http://www.ktl.fi/terveys2000/julkaisut/baseline.pdf.
14 Rose G, Blackburn H, Gillum R, Prineas R. *Cardiovascular Survey Methods*, 2nd edn. World Health Organization: Geneva, 1982.
15 Bortolotto LA, Henry O, Hanon O, Sikias P, Mourad JJ, Girerd X. Validation of two devices for self-measurement of blood pressure by elderly patients according to the revised British Hypertension Society protocol: the Omron HEM-722C and HEM-735C. *Blood Press Monit* 1999; **4**: 21–25.
16 O'Brien E, Asmar R, Beilin L, Imai Y, Mallion JM, Mancia G *et al.* European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. *J Hypertens* 2003; **21**: 821–848.
17 Sokolow M, Lyon T. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *Am Heart J* 1949; **37**: 161–186.
18 Okin PM, Devereux RB, Jern S, Kjeldsen SE, Julius S, Dahlof B. Baseline characteristics in relation to electrocardiographic left ventricular hypertrophy in hypertensive patients: the Losartan intervention for endpoint reduction (LIFE) in hypertension study. The Life Study Investigators. *Hypertension* 2000; **36**: 766–773.
19 Schillaci G, Verdecchia P, Borgioni C, Ciucci A, Guerrieri M, Zampi I *et al.* Improved electrocardiographic diagnosis of left ventricular hypertrophy. *Am J Cardiol* 1994; **74**: 714–719.
20 Molloy TJ, Okin PM, Devereux RB, Kligfield P. Electrocardiographic detection of left ventricular hypertrophy by the simple QRS voltage-duration product. *J Am Coll Cardiol* 1992; **20**: 1180–1186.
21 Jöreskog KG, Sörbom D. *LISREL 8: User's Reference Guide*. Scientific Software International: Chicago, 1996.

22 Marcus R, Krause L, Weder AB, Dominguez-Meja A, Schork NJ, Julius S. Sex-specific determinants of increased left ventricular mass in the Tecumseh Blood Pressure Study. *Circulation* 1994; **90**: 928–936.
23 Jula AM, Karanko HM. Effects on left ventricular hypertrophy of long-term nonpharmacological treatment with sodium restriction in mild-to-moderate essential hypertension. *Circulation* 1994; **89**: 1023–1031.
24 Kupari M, Koskinen P, Virolainen J. Correlates of left ventricular mass in a population sample aged 36 to 37 years. Focus on lifestyle and salt intake. *Circulation* 1994; **89**: 1041–1050.
25 Antikainen R, Grodzicki T, Palmer AJ, Beevers DG, Coles EC, Webster J *et al.* The determinants of left ventricular hypertrophy defined by Sokolow–Lyon criteria in untreated hypertensive patients. *J Hum Hypertens* 2003; **17**: 159–164.
26 Okin PM, Jern S, Devereux RB, Kjeldsen SE, Dahlof B. Effect of obesity on electrocardiographic left ventricular hypertrophy in hypertensive patients : the losartan intervention for endpoint (LIFE) reduction in hypertension study. *Hypertension* 2000; **35**: 13–18.
27 O'Brien E, Asmar R, Beilin L, Imai Y, Mancia G, Mengden T *et al.* Practice guidelines of the European Society of Hypertension for clinic, ambulatory and self blood pressure measurement. *J Hypertens* 2005; **23**: 697–701.
28 Pickering T. Recommendations for the use of home (self) and ambulatory blood pressure monitoring. American Society of Hypertension Ad Hoc Panel. *Am J Hypertens* 1996; **9**: 1–11.
29 Ohkubo T, Asayama K, Kikuya M, Metoki H, Hoshi H, Hashimoto J *et al.* How many times should blood pressure be measured at home for better prediction of stroke risk? Ten-year follow-up results from the Ohasama study. *J Hypertens* 2004; **22**: 1099–1104.
30 Stergiou GS, Baibas NM, Gantzarou AP, Skeva II, Kalkana CB, Roussias LG *et al.* Reproducibility of home, ambulatory, and clinic blood pressure: implications for the design of trials for the assessment of antihypertensive drug efficacy. *Am J Hypertens* 2002; **15**: 101–104.