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ORIGINAL ARTICLE

The determinants of left ventricular hypertrophy defined by Sokolow–Lyon criteria in untreated hypertensive patients

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Left ventricular hypertrophy (LVH) measured by electrocardiography (ECG LVH) in hypertensive patients has been shown to be associated with an increased risk of cardiovascular sequelae. Analysis of the determinants predisposing to ECG LVH may be helpful in the prevention of LVH. The Department of Health and Social Security Hypertension Care Computer Project studied 2994 hypertensive patients in whom an electrocardiogram was recorded while not on treatment. LVH was determined as the voltage sum SV1+RV5 or RV6 $\geqslant$ 35 mm using Sokolow–Lyon voltage criteria. The relations were determined between the presence of LVH or voltage sum and different variables. Untreated systolic (SBP) and diastolic (DBP) blood pressure and pulse pressure were positively related to the increasing ECG voltage, while body mass index (BMI) and serum cholesterol were inversely related. Blood glucose and age did not correlate significantly. Patients with the presence of ECG LVH were more often men, black people, smokers and users of alcohol. In multiple logistic regression analyses, SBP, DBP, male gender and black race were positively, whereas BMI was negatively related to the presence of LVH. The positive relation of smoking and negative relation of serum cholesterol concentration to the presence of ECG LVH were apparent in men but not in women. This study confirms the adverse association between ECG LVH and SBP and DBP, male gender, black race and decreased BMI. It also addresses the less well-known associations of blood glucose, cholesterol, smoking and alcohol consumption.
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Introduction

Left ventricular hypertrophy (LVH) in hypertensive patients has been shown to be associated with an increased risk of cardiovascular sequelae, irrespective of whether it is determined by electrocardiogram (ECG LVH) or echocardiography (ECHO-LVH).[1] The ECG shows a high specificity in diagnosing anatomical LVH revealed by necropsy or ECHO, whereas its sensitivity is relatively low.[2] Owing to availability and low cost, the ECG has traditionally been the principal method recommended to recognise this target organ damage in the heart.[3]

The Sokolow–Lyon ECG voltage criteria[4] are commonly used and very easy to employ in the assessment of LVH. Regression of LVH by Sokolow–Lyon criteria in subjects with normal or controlled hypertension has been shown to result in a reduction of comorbidity in the Heart Outcomes Prevention Evaluation (HOPE) Study.[5] The recent Losartan Intervention for Endpoint Reduction in Hypertension (LIFE) Study was designed to investigate whether patients with essential hypertension and ECG LVH defined by Cornell product or Sokolow–Lyon criteria differ in their cardiovascular benefits from different antihypertensive therapies aimed at reducing LV mass. The results of the LIFE Study revealed that the risk of cardivascular morbidity and death was lower, and reversing LVH more effective, in patients with ECG LVH prescribed the angiotensin receptor blocking agent losartan, than in those assigned the β-blocker atenolol. The benefit seemed

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to be independent of the blood pressure reduction.[6] Thus, LVH is not only a major risk factor, but its reduction is also an important goal in the treatment of cardiovascular diseases.

The aim of this paper was to analyse the determinants of LVH ECG by Sokolow–Lyon criteria. These results may help the understanding of the development of LVH and suggest strategies in prevention.

Material and methods

During 1971–1986, the Department of Health and Social Security Hypertension Care Computer Project (DHCCP) studied 10 186 patients referred with hypertension. In 3924 patients, an ECG was recorded while not on treatment. In all, 95% of patients entered the system on presention to one of the hospital clinics (Hammersmith Hospital, London; King's College Hospital, London; John Radcliffe Hospital, Oxford; City Hospital, Birmingham; Aberdeen Royal Infirmary). The remaining 5% were recruited from the general practices involved in the study (Kentish Town, London; Harlow; Oxford; Norwich).

The standard 12-lead resting ECGs were performed by experienced observers and evaluated by trained clerical workers. ECG LVH was determined by Sokolow–Lyon voltage criteria[4] and was diagnosed when the sum SV1+RV5 or RV6 was equal to or more than 35 mm. Blood pressure and biochemical measurements were not formally standardised, although the latter were done in laboratories linked to the United Kingdom quality assurance scheme. Blood was not necessarily taken in the fasting state. Most observers used standard mercury sphygmomanometers and phase V Korokoff as the diastolic blood pressure (DBP). Body mass index (BMI) was calculated as weight (kg)/height squared (m^2). Untreated pulse pressure (PP) was the difference between untreated systolic blood pressure (SBP) and untreated DBP. Smokers were those who had ever smoked. Alcohol use was determined by self-report (yes or no). Subjects younger than 18 years or with both SBP <140 mmHg and DBP <90 mmHg were excluded from the study ($n=926$). Also excluded were three subjects whose information on gender was missing. Thus, 2994 subjects were included, in whom complete data on age, sex, DBP and the presence or absence of LVH were recorded. Of these, the information on SBP was available in 2993 subjects, that on race, BMI, smoking, blood glucose, serum cholesterol and alcohol use in 2856, 2606, 2581, 1790, 1766 and 1691 subjects, respectively.

Database management was performed using the Statistical Analysis System (The SAS Institute Inc., Cary, NC, USA). The differences in mean values and standard errors among subjects with or without ECG LVH were compared using Student's *t*-test. Comparisons between proportions were performed using the χ^2 test for independent groups. The relation between increasing ECG voltage and continuous variables such as SBP, DBP, PP, age, gender, BMI, serum cholesterol and blood glucose concentration was determined using Pearson's correlation coefficient. Multiple logistic regression analysis was employed to determine the independent effect of variables on the presence or absence of LVH in 2498 subjects, where information on untreated SBP, sex, race and BMI was complete. SBP and DBP were introduced separately into the model. The variables in model A (Tables 2 and 3) were selected from correlated variables associated with the presence of LVH known for the vast majority of patients. Additionally, analyses were performed introducing blood glucose or serum cholesterol concentrations, smoking, the use of alcohol or age into the model. We analysed the data in all subjects and in men and women separately.

Results

The baseline characteristics in men and women, with and without ECG LVH, are presented in Table 1. The prevalence of LVH defined using the Sokolow–Lyon criteria was 19% (14% for women, 23% for men) in this untreated hypertensive population. Patients with LVH had higher SBP, PP but lower average BMI and they were more often black people. Men with LVH were more often smokers and had a lower serum cholesterol concentration. In men and women combined, an increase in ECG voltage per 1 mm was correlated with SBP ($R=0.26$, $P<0.0001$), DBP ($R=0.21$, $P=<0.0001$) and PP ($R=0.19$, $P<0.0001$), BMI ($R=-0.20$, $P<0.0001$) and serum cholesterol ($R=-0.11$, $P<0.0001$) were inversely related, while age ($R=0.02$, $P=0.1851$) and blood glucose ($R=-0.04$, $P=0.1338$) did not correlate significantly with an increase in ECG voltage. Compared with subjects without ECG LVH, alcohol consumption (76.2 *vs* 70.2%, $P=0.0285$), male gender (63.4 *vs* 48.6%, $P<0.0001$), black race (20.3 *vs* 8.3%, $P<0.001$) and smoking (39.1 *vs* 28.9%, $P<0.0001$) were more common in subjects with the presence of LVH. When analysed for men and women separately, the correlation remained very similar for continuous variables.

In the multiple logistic regression model, male gender, black race and SBP were positively related to the presence of LVH, whereas BMI was negatively related (Table 2, Model A). After introducing smoking, serum cholesterol, alcohol consumption, blood glucose concentration or age (Table 2, Models B–F) into the model, these relations remained virtually unchanged. The inverse effects of serum cholesterol and blood glucose concentration were statistically significant, whereas the effects of smoking and alcohol consumption were not. Age was weakly and inversely correlated with the

Table 1 Baseline characteristics for previously untreated hypertensive women and men according to absence or presence of LVH. A total of 205 women and 355 men were categorised as ECG LVH present and 1251 and 1183, respectively, as ECG LVH absent

	Women				*Men*			
	Number of subjects with information	*LVH absent*	*LVH present*	P-*value*	*Number of subjects with information*	*LVH absent*	*LVH present*	P-*value*
Age (years) ± s.e.	1456	49.1 ± 0.40	50.3 ± 1.12	0.2606	1538	48.1 ± 0.36	48.7 ± 0.69	0.3947
SBP (mmHg) ± s.e.	1456	173 ± 0.78	192 ± 2.37	<0.0001	1537	168 ± 0.74	183 ± 1.49	<0.0001
DBP (mmHg) ± s.e.	1456	104 ± 0.39	112 ± 1.24	<0.0001	1538	104 ± 0.40	112 ± 0.90	<0.0001
PP (mmHg) ± s.e.	1456	69 ± 0.62	80 ± 1.82	<0.0001	1537	64 ± 0.58	71 ± 1.12	<0.0001
Race Black (%).	1387	10.1	23.6	<0.0001	1469	6.4	18.4	<0.0001
BMI (kg/m^2) ± s.e.	1268	27.0 ± 0.16	25.3 ± 0.33	<0.0001	1338	27.3 ± 0.12	25.8 ± 0.21	<0.0001
Smokers (%)	1253	24.3	27.3	0.3976	1328	33.8	45.5	0.0002
Alcohol use (%)	794	56.7	65.0	0.0956	897	83.7	82.0	0.5749
B-gluc (mmol/l) ± s.e.	879	5.2 ± 0.05	5.0 ± 0.09	0.2360	911	5.4 ± 0.06	5.2 ± 0.12	0.1102
S-chol (mmol/l) ± s.e.	839	6.3 ± 0.05	6.2 ± 0.14	0.7322	927	6.1 ± 0.04	5.8 ± 0.07	0.0002

LVH, Left Ventricular hypertrophy; s.e., standard error; SBP, untreated systolic blood pressure; DBP, untreated diastolic blood pressure; PP, untreated pulse pressure; BMI, body mass index; B-gluc, blood glucose concentration; S-chol, serum cholesterol concentration.

Table 2 Multiple logistic regression

		Slope	*s.e.*	P
Model A:	*N*=2498			
Sex (m/f)		0.8652	0.116	<0.0001
Race (bl/nb)		1.2622	0.155	<0.0001
BMI (kg/m^2)		−0.1282	0.015	<0.0001
SBP (mmHg)		0.0223	0.002	<0.0001
Model B:	*N*=2138			
Smoker (yes/no)		0.2321	0.128	0.0687
Model C:	*N*=1467			
S-chol (mmol/l)		−0.1527	0.063	0.0148
Model D:	*N*=1497			
Alcohol (y/n)		0.1778	0.171	0.2985
Model E:	*N*=1422			
B-gluc (mmol/l)		−0.1248	0.052	0.0156
Model F:	*N*=2498			
Age (years)		−0.0119	0.005	0.0117

m, male=1; f, female=0; bl, black people=1; nb, nonblack people=0. Regression coefficients (slopes) of baseline variables determining the presence of LVH among hypertensive subjects. Model A employs sex, race, body mass index (BMI) and systolic blood pressure (SBP). In Model B, smoking was added to Model A. In Model C, serum cholesterol was added instead of smoking. The other models added the use of alcohol (Model D), blood glucose concentration (B-gluc) (Model E) or age (Model F) instead of smoking or cholesterol.

presence of LVH (Table 2, Model F) When DBP was added into the analyses instead of SBP, the relations were very similar. However, age and blood glucose concentration were no longer significantly related to the presence of ECG LVH (data not shown).

The clinical variables predicted LVH similarly in both women and men. However, the positive relation of smoking with the presence of ECG LVH and inverse relation of serum cholesterol concentration and age were found only in men (Table 3). In men also, the association between age and the presence of ECG LVH was dependent on DBP (data not shown).

Discussion

In all subjects, SBP, DBP, male gender and black race were independently and positively related to the presence of ECG LVH, while BMI and serum cholesterol were negatively related. In both sexes, black race, SBP or DBP were positively and BMI was negatively and statistically significantly related to the presence of ECG LVH. However, the positive relation of smoking and the negative relations of age or serum cholesterol concentration were only independently associated with the presence of LVH in men.

The main strength of our study is the high number of hypertensive subjects who were not treated at baseline and who had information on the presence or absence of ECH LVH as defined by the commonly employed Sokolow–Lyon criteria. Since most patients referred with hypertension between 1971 and 1986 presented with elevated DBP, this study clearly was biased towards diastolic hypertension. Blood pressure measured only once was employed in the analyses. However, patients were referred with hypertension to the study. Thus, the single blood pressure measurement might not greatly overstimate the patient's usual blood pressure level, as these subjects will have been accustomed to the blood pressure readings. Inclusion of individuals who have smoked little or stopped for many years ago may have underestimated the strength between ECG LVH and smoking.

The Sokolow–Lyon voltage criteria were used to define ECG LVH in the European Working Party on High Blood Pressure in the Elderly (EWPHE) Trial.[7] In the LIFE Study,[8] LVH was diagnosed from a standard ECG based on Cornell criteria. To increase the sensitivity of the test, ECG LVH by Sokolow–Lyon criteria, after increasing voltage to 38 mm,

Table 3 Multiple logistic regression

	Women			*Men*		
	Slope	*s.e.*	P	*Slope*	*s.e.*	p
Model A		*N*=1218			*N*=1280	
Race (bl/nb)	1.3158	0.233	<0.0001	1.2099	0.208	<0.0001
BMI (kg/m^2)	−0.1223	0.218	<0.0001	−0.1358	0.021	<0.0001
SBP (mmHg)	0.0235	0.003	<0.0001	0.0214	0.003	<0.0001
Model B		*N*=1045			*N*=1093	
Smoker (yes/no)	−0.1081	0.226	0.6329	0.3999	0.158	0.0113
Model C		*N*=700			*N*=767	
S-chol (mmol/l)	−0.0936	0.088	0.2864	−0.2163	0.089	0.0155
Model D		*N*=743			*N*=756	
B-gluc (mmol/l)	−0.1459	0.095	0.1241	−0.1201	0.062	0.0530
Model E		*N*=664			*N*=758	
Alcohol (yes/no)	0.2768	0.244	0.2564	0.0750	0.241	0.7551
Model F		*N*=1218			*N*=1280	
Age (years)	−0.0098	0.007	0.1856	−0.0137	0.006	0.0269

m, male people=1; f, female people=0; bl, black people=1; nb, nonblack people=0.
Regression coefficients (slopes) of baseline variables on the presence of left ventricular hypertrophy among hypertensive women and men. Model A employs sex, race, body mass index (BMI) and systolic blood pressure (SBP). In Model B, smoking was added to Model A. In Model C, serum cholesterol was added instead of smoking. The other models added the use of alcohol (Model D), blood glucose concentration (B-gluc) (Model E) or age (Model F) instead of smoking or cholesterol.

additionally was accepted. Unlike in our study, all 9194 hypertensive patients of the LIFE Study had LVH as criteria for entry. At baseline, only 23.1% of patients had ECG LVH by the modified Sokolow–Lyon criteria.[8] An increasing SBP and PP[8] but not DBP[7] predicted ECG LVH, while an increasing age[7,8] was inversely and very weakly related to the presence of ECG LVH by Sokolow–Lyon criteria.

In black persons, the prevalence of ECG LVH defined by Sokolow–Lyon criteria has been found to be particularly high in comparison with the LVH prevalence among white people.[9,10] Sokolow–Lyon criteria may overestimate the prevalence of anatomical LVH among black or African people by yielding a high number of false-positive diagnoses of ECG LVH.[9–13]

Only little is known about the relation of alcohol use, smoking and serum cholesterol level with ECG LVH by Sokolow–Lyon criteria. In agreement with our observations, patients with LVH by Sokolow–Lyon criteria in the LIFE Study were more likely to be current smokers and have a lower serum cholesterol level than patients without LVH.[8] In the present paper, the positive association between smoking and the negative association between serum cholesterol concentration and the presence of LVH were statistically significant in men, but not in women. It should be noted that the prevalence of smoking was lower in women than in men. Alcohol use has been shown to be associated with the presence of ECG LVH defined by Minnesota code 3.1 or 3.3[14] or by Perugia score.[15] Information on use of alcohol, serum cholesterol or glucose concentrations in the present study was available only in about half of the 2498 patients; therefore, selection bias cannot be excluded. However, when we analysed subjects in whom information on alcohol use, serum cholesterol and glucose concentration was missing, the relation of other variables (sex, race, BMI and SBP) with the presence of LVH remained virtually the same (data not shown). The correlation coefficients for the above variables were not particularly strong and the underlying inducing mechanisms for LVH are not clear.

Autopsy and echocardiographic studies favour our findings of an increased absolute left ventricular mass in men,[16] although this is complicated by the fact the LVH is increased by age and body weight more in women.[17–19] LVH may be more common among middle-aged women than among men.[20–22]

Obesity has been shown to be associated independently with increased anatomical LVH among hypertensive subjects[23–25] and in a general population.[17] However, in our analysis as well as in the LIFE Study,[8] an increase in BMI was significantly and negatively related to the presence of LVH by Sokolow–Lyon criteria. The sensitivity of ECG LVH by Sokolow–Lyon criteria to recognise ECHO LVH among obese subjects, especially among obese women, has been confirmed to be low.[26,27] This decreased sensitivity in obese subjects may be because of accumulation of the subcutaneous adipose tissue of the chest wall,[26–28] although, in women, the effect of breast tissue appears to have a surprisingly small effect on ECG voltages.[29] Non-insulin-dependent diabetes mellitus has been observed to be positively associated with ECHO LVH,[30,31] as well as with ECG LVH-determined Cornell voltage-duration product.[8] Obesity is also a well-known risk factor of type II diabetes mellitus.[32]

This may explain the fact that blood glucose in our study and the presence of diabetes in the LIFE Study[8] tended to be inversely related to the presence of ECG LVH determined using Sokolow–Lyon voltage criteria. It must be admitted that our increased detection of LVH with decreasing BMI may be an artefact of measurement, as obesity of the chest wall will influence readings of QRS voltages in precordial leads. We plan to estimate survival in both obese and nonobese hypertensive subjects, with the Sokolow–Lyon voltage criteria for LVH.

ECHO LVH has been found to correlate with postmorterm left ventricular weight, whereas ECG LVH correlated poorly.[1,2,33,34] Several different ECG criteria have been evaluated to enhance the sensitivity of criteria while maintaining high specificity, but nevertheless, ECG has found to be a poor screening test for ECHO LVH.[9,11–13,20,26,35] The ECG LVH may not depend solely on left ventricular mass and there is a poor correlation between the ECG and ECHO indicators of LVH, suggesting that anatomical ECHO and electrical ECG versions might reflect different pathogeneses. ECG provides information not available from ultrasound measurements such as repolarization changes, which may provide additional information on myocardial perfusion and left ventricular stress.[1] The two techniques should be regarded as complementary.

The determinants of ECG LVH among hypertensive patients have been found to differ in several ways depending on the criteria used.[2,8] In the LIFE Study,[8] unlike by Sokolow–Lyon criteria, LVH by Cornell voltage-duration product criteria was associated with advancing age, female gender, white race and increasing BMI. The Sokolow–Lyon criteria are easy to apply and widely used, but more information is needed to estimate their prognostic value.

Conclusions

This study confirms the positive association between LVH and SBP and DBP, male gender, black race and decreased BMI. It also addresses the less well-known associations of blood glucose, cholesterol, smoking and alcohol consumption. It remains to be demonstrated whether the commonly used ECG LVH by Sokolow–Lyon criteria is a predictor of cardioivascular disease mortality in hypertensive subjects.

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