Detection of Atrial Electrical Instability in Hypertensive Patients

A Oręziak[1], M Niemczyk[1], E Piątkowska-Janko[2], G Opolski[1]

[1] Chair and Department of Cardiology, Medical University of Warsaw, Poland
[2] Nuclear and Medical Electronics Division, Institute of Radioelectronics, Warsaw University of Technology, Poland

Abstract

The aim of our study was to evaluate the influence of the left ventricular hypertrophy (LVH) on the atrial electrical instability in hypertensive patients (pts). 76 hypertensive pts without symptomatic coronary disease, systolic dysfunction, electrolyte disturbances or antiarrhythmic therapy were included in our study, and were divided into two groups: with LVH (group I) – 55 pts and without LVH (group II) – 21 pts. There were additionally measured: ECHO, the 12-lead ECG. From SAECG ECG were calculated the filtered P-wave duration (hfP), the root mean square voltages for last 30msec (RMS_{30}); RMS voltages for the last time quarter of P-wave divided by RMS voltages of full time of P-wave (RMS4/RMS); envelope of the first 10 ms of P-wave vector magnitude (Penv10).

Conclusion: Signal averaged P-wave ECG may be a useful tool to evaluation of atrial electrical instability in hypertensive pts with LVH.

1. Introduction

Left ventricular hypertrophy (LVH) occurs in approximately 15-30% patients (pts) with primary hypertension. LVH has been found as an independent risk factor for ventricular arrhythmias and sudden cardiac death. Relationship between LVH and atrial arrhythmias has not been fully understood. Epidemiologic studies disclose that hypertension is the main reason of atrial fibrillation.

This study demonstrates presence of the arrhythmogenic markers in pts with hypertension and LVH. The aim of our study was to evaluate atrial electrical instability in hypertensive pts with LVH.

2. Patients and methods

We studied 76 hypertensive pts (26 women and 50 men). All pts were on sinus rhythm and had primary hypertension. We excluded pts with secondary hypertension to avoid potential influence of the cause of hypertension on proarrhythmia markers (i.e QT interval prolongation induced by hypocalemia in primary hyperaldosteronism). Hypertensive pts with symptomatic coronary disease, post infarction and with heart failure were also excluded. None of them had bundle branch block, arrhythmia and valvular heart disease. Serum electrolytes were within normal limits in all pts. None of them had antiarrhythmic therapy. No antihypertensive therapy was permitted during 7 days before the study - the most of pts had a new diagnosed hypertension. All study subjects provided writing informed consent. The local Ethics Committee approved protocol of the study.

Pts were studied using standard 12-lead ECG, 24-hour Holter monitoring, M-mode and two-dimensional echocardiography and signal averaged electrocardiogram (SAECG). All of the tests were done in 48-hour period.

2.1. Echocardiography

Examinations were performed using a Hewlett-Packard Sonos 2500 recorder with 3,5 MHz transducer. In each pt 2-dimmentional and M-mode tracing were obtained.

The left ventricular end-systolic diameter (LVESD), the left ventricular end-diastolic diameter (LVEDD), the posterior wall thickness at end-diastole (PWT) and the intraventricular septal thickness at end-diastole were obtained according to the recommendations American Society of Echocardiography [1].

Left ventricular mass (LVM) was calculated using the Penn formula [2]:

$$LVM = 1{,}04 * [(IVS+LVEDD+PWT)^3 - LVEDD^3] - 13{,}6$$

Left ventricular mass index (LVMI) was defined as relation LVM and the body surface area (SA) in m^2 and was calculated using the Du Bois formula as follows:

$$SA = 0{,}007184 * H^{0.725} * W^{0{,}425}$$

where H is height in cm and W is weight in kg. LVH was confirmed when LVMI > 111 g/m^2 in men and LVMI > 106 g/m^2 in women [3]. All pts had also measured internal diameters of left atrium in short and long axis and areas of left and right atrium (LAsax, LAlax, LAar, RAar).

2.2. Signal averaged electrocardiogram

The SAECGs were recorded the orthogonal X, Y and Z leads (in accordance to the Frank's standard) by the computer equipment.

The model of P-wave signal characteristic for the each pt were built by the first ten beats of the heart – even next beat was compared with the model. If the correlation between the model and next signal had been higher than 0,85, the signal would have been recorded. The signal of each lead was amplified. Minimum 250 beats were averaged and filtered at FIR with Kaiser window 45-150Hz. The noise level < 0,7 μV was acceptable. The signal were combined into vector magnitude $(X^2+Y^2+Z^2)^{0,5}$ after amplification, averaging and filtering.

By the averaged signal the computer program calculated:

- duration of atrial signal (hfP) (msec) – onset was defined as the first deflection from baseline exceeding a value approximately more than twice the noise amplitude, offset was defined as the return of the atrial signal to a level less than approximately twice noise amplitude above baseline or the onset of the QRS, whichever was earlier. hfP>120 msec was confirmed as prolonged.
- the root-mean-square voltages in μV (RMS) for the last 30 msec of the P wave (RMS_{30})
- RMS for the last time quarter of P-wave divided by RMS voltages of full time of P-wave (RMS_4/RMS);
- envelope of the first 10 ms of P-wave vector magnitude (Penv10).

2.3. Electrocardiogram

On the base of the standard 12-lead ECG (CardioPerfect 3,4: 50mm/s, 10mm/mV) were measured;

-duration P (Pd) – was defined as the time interval between the earliest onset of the P wave in any of the I, II, and III leads and latest offset in any of the I, II, and III leads in msec.

-dispersion P (ΔP) – was defined as difference between the longest and the shortest P wave (msec).

-PQ interval (PQ) – PQ was measured from the beginning P wave to the beginning the Q wave (msec).

2.4. A 24-hour Holter monitoring

24-hour ambulatory ECG monitoring was undertaken with an Oxford Medilog recorder during normal activities of each pt.

Atrial abnormal activity was counted by the analyses of the recorded ECG signals and was divided on:

-supraventriculare extrasystoles (SV_{Es}) - number of SV_{Es} per 24 hour

-paroxysmal atrial tachycardias (number of occurrence)

-nonsustained supraventriculare tachycardias (SVT) - number of occurrence and time of duration.

2.5. Statistical methods

Results were presented as mean ± SD, or as a number of pts (percentage). Means were compared using Student's t test. A p value < 0,05 was considered significant.

3. Results

3.1. Patients characteristics and echocardiography results

Pts were divided into two groups:

I – LVH(+) - 55 pts with primary hypertension and LVH by ECHO – (21 women, 34 men), mean age 45,6 ± 8,3 years;

II – LVH(-) - 21 pts with primary hypertension and without LVH – (8 women, 13 men), mean age 44,2 ± 9,7 years.

The characteristic of the pts studied are summarised in table below.

Table 1. Clinical features comparison – pts with and without LVH.

	Group I with LVH n=55	Group II without LVH n=21	p
Age (years) ± SD	45,6 ± 8,3	45,6 ± 8,3	NS
SA (m^2) ± SD	2,00 ± 0,17	1,964 ± 0,16	NS
LAsax (cm) ± SD	4,0 ± 0,4	3,6 ± 0,4	< 0,001
LAlax (cm) ± SD	5,43 ± 0,55	5,28 ± 0,56	NS
LAar (cm^2) ± SD	21,7 ± 6,9	16,9 ± 1,4	< 0,001
RAar (cm^2) ± SD	17,4 ± 4,3	16,8 ± 2,4	NS
LVEED (cm) ± SD	5,2 ± 0,4	4,7 ± 0,4	< 0,001
IVS (cm) ± SD	1,2 ± 0,1	1,0 ± 0,2	< 0,001
PWT (cm) ± SD	1,1 ± 0,1	0,9 ± 0,1	< 0,001
LVMI (g/m^2) ± SD	135,9 ± 25,4	94,6 ± 11,0	< 0,001

SA – body surface area, LAsax – left atrial internal diameter-short axis, LAlax – left atrial internal diameter-long axis, RAar – internal area of right atrium, LAar – internal area of left atrium, LVEED – left ventricular end diastolic diameter, IVS – thickness of the intraventricular septum, PWT – left ventricle posterior wall thickness, LVMI – left ventricle mass index

There were no significance differences in SA, time duration and mean value of hypertension and serum concentration of K^+ among the groups.

We observed the significance increase of the LAsax

(4,0 vs 3,6 cm; p< 0,001) and LAar (21,7 vs 16,9 cm^2; p< 0,001) in the I group. The other atrial ECHO parameters were similar in both groups.

3.2. Signal averaged electrocardiogram

17 pts (31%) with and only 2 pts (9,5%) without LVH had prolonged hfP in SAECG. The mean of hfP were in I group 127,78 ± 8,4 msec and in II group 108,05 ± 9,16 msec (p< 0,05). RMS_{30} was significant lower in hypertensive pts with LVH and Penv10 was increased in this group.

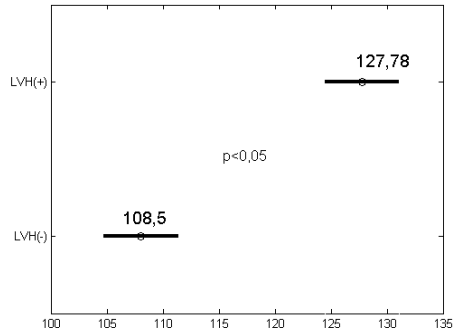

Figure 1. Significant difference between hfP in group I and group II.

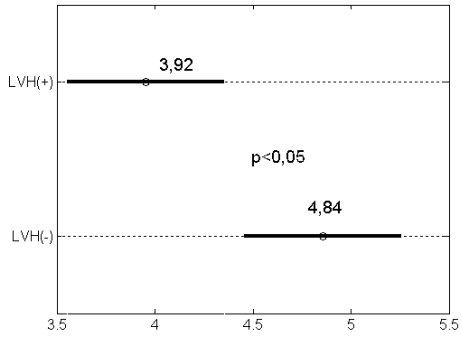

Figure 2. Significant difference between RMS_{30} in group I and group II.

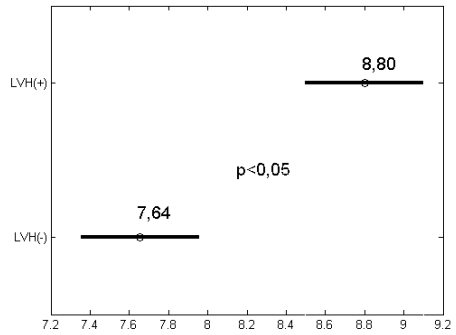

Figure 3. Significant difference between Penv10 in group I and group II.

3.3. Electrocardiogram and 24-hour Holter monitoring

Pd was increased (115 ± 3 vs 107 ± 3 msec; p< 0,001) and PQ was significance longer (171 ± 21 vs 151 ± 18 msec; p< 0,001) in group I. There were noted no difference in ΔP.

43 pts (78%) in I group had isolated SV_{Es} (116-mean of SV_{Es} in this group), SVT was recorded in 7 pts (13%) - total ten episodes, mean time of SVT was 450 msec (per one of 7 persons). In II group 16 pts (76%) had isolated SV_{Es} (56 – mean of SV_{Es} in this group), only one person (4,8 %) had 190 msec epizode of SVT.

4. Discussion

It has been postulated that the P-wave signal averaged might show atrial areas of delayed electrical conduction. Slow atrial conduction may cause increase of signal averaged P-wave duration, which is found as a risk factor of an occurrence of atrial fibrillation (AFi) and other atrial arrhythmias. Electrical atrial potential can be modified by several factors: myocardial changes with intra-atrial conduction abnormalities, left atrial hypertension, left atrial distension and chronicity of disease [4]. The hfP wave duration can be also affected by changes in autonomic tone [5]. LVH in hypertension influence the structure of left atrial myocardium. Diastolic filling of left ventricular was impaired in pts with hypertensive heart disease and left atrial enlargement might be attributed to the impairment of blood flow from left atrium to left ventricle due to the increased LV stiffness [6]. Another authors postulate that left atrial size correlates with LV wall thickness in pts with essential hypertension and normal left ventricular systolic function [7]. Gottdiener et al. has shown that there was no relation between LV mass and LA size in normal weight hypertesive men, but was a significant positive relation in obese pts - obesity was the strongest independent predictor of increased LA size [8]. Increased LA diameter occurred more frequent in group with LVH in our study. It seems that LA as a thin-wall structure is susceptible to increased pressure. Left atrial dilatation also leads to alteration in electrophysiologic properties of the atrium such as shortening of refractoriness and prolongation of conduction time. Atrial depolarisation is influenced by atrial pathology such as fibrosis, changes in atrial elasticity and conduction pathways. Left atrial dilatation plays a significant role in the pathophysiology of the increased incidence of atrial arrhythmias [9]. Some authors postulated that LA diameter increased after the onset of Afi [10].

In our study the hfP was prolonged in group with LVH, which was influenced by LA dilatation. Some authors reported that terminal segments of hfP had a poor predictive factor of AF, but Fukunami et al. found

significantly reduced RMS voltage in the terminal 10 and 20 ms of hfP in pts with Afi [11]. In our study no pt in both groups had atrial fibrillation. More episodes of supraventricular arrhythmias were recorded in Holter monitoring in pts with LVH (6 pts had nonsustained supraventricular tachycardia). In standard 12-lead ECG we observed prolongation of atrial depolarisation in pts with LVH. In one of previous study P-wave duration on the standard limb leads lengthened as LA size increased. Duration of P-wave was considered as the most sensitive for LA enlargement [12]. Matsuda et al. evaluated pts with LA functional abnormalities in the setting of increased LV mass [13], other studies showed possibility association of blood pressure with LA size and LVH in hypertension.

5. Conclusion

LVH in hypertensive pts influence the increase of ECHO parameters of left atrium and may be the cause of supraventricular arrhytmias.

Dilatation of LA is probably the reason for the atrial electrical instability of the heart, which could be measured by signal-averaged P-wave ECG.

Signal averaged P-wave ECG may be a useful tool to evaluation of atrial electrical instability in hypertensive pts with LVH.

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Address for correspondence.
Artur Oręziak.
Chair and Department of Cardiology
Medical University of Warsaw
1a Banacha str, 02-097 Warsaw, Poland
e-mail: artur.oreziak@amwaw.edu.pl