Left and right ventricular hypertrophy at autopsy of hypertensive individuals

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Summary

Objective: To measure the right and left ventricular thickness in deceased individuals with a history of hypertension submitted to clinical autopsies. **Methods:** We selected 90 cases from the Death Verification Service of the city of Recife, state of Pernambuco, Brazil, of both sexes, with a history of essential arterial hypertension related to heart wall thickness, in addition to correlation with autopsy findings and other clinical reports. **Results:** There was a significant association between the presence of left ventricular hypertrophy (LVH) and right ventricular hypertrophy (RVH) and between severe hypertensive cardiomyopathy and RVH. There was a predominance of RVH and LVH in men aged 60-79 years and a higher prevalence in the Brazilian mulatto and Black ethnic groups and in those with adequate nutritional status or overweight and obese individuals. **Conclusion:** It was observed that the presence of RVH was related to LVH, suggesting that there are similar pathogenic factors involved in the development of bilateral hypertrophy. The RVH seems to be associated with more severe heart disease and may, based on other studies, be considered as a new prognostic factor in the evaluation of hypertensive patients.

Keywords: Right ventricular hypertrophy; left ventricular hypertrophy; autopsy; hypertension.

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Introduction

Systemic arterial hypertension (SAH) is one of the most common diseases in our country, and population studies have shown a prevalence of 22.3% to 43.9% in Brazil. Assessment of SAH becomes relevant due to potential complications, such as cerebrovascular and coronary disease, in addition to being responsible for medical and hospital costs and substantial socioeconomic burden to society[1].

SAH is one of the causes of left cardiac hypertrophy, and the myocardium is the main a□ected target organ in hypertensive individuals[2]. The increase in peripheral vascular resistance is the determining agent for this adaptation; however, in addition to hemodynamic changes, other factors such as neurohormonal activity, cytokines, oxidative stress, ischemia or the expression of genetic factors are involved in the development of cardiac remodeling, whose main goal is to maintain systolic function[3]. Left ventricular hypertrophy (LVH) has been shown to be associated with higher cardiovascular risk. Collagen deposition and myocardial fibrosis, arrhythmias, diastolic dysfunction, and changes in coronary □ow may be responsible for increased morbidity and mortality related to this morphological change[4-7].

In addition to the e□ects of hypertension on LV, some studies have shown that the disease also a□ects the right ventricle (RV)[8-10]. Many of these evaluations were carried out through echocardiographic analysis, particularly the study of cardiac mass and volume. In one of these studies, Nunez et al. found that the right ventricular wall thickness was about two times higher in hypertensive patients with left ventricular hypertrophy compared to normotensives without hypertrophy[9]. The observed RV remodeling also leads to diastolic dysfunction in both cardiac chambers, with decreased ejection fraction and ventricular relaxation time to the right. Thus, the observed functional interdependence would occur because the LV and RV share the interventricular septum and form a single structural unit[11-13].

Methods

The research objectives were focused on the analysis of cardiac ventricular hypertrophy at clinical autopsy of hypertensive individuals, in order to help clarify its determinant factors. Initially, we measured the cardiac wall thickness in deceased individuals who had essential hypertension, and the results were subsequently correlated. When present, left and/or right hypertrophy was classified in grades and related to other clinical reports and autopsy findings.

This was a descriptive, cross-sectional study of 90 hearts of cadavers from the Death Verification Service of Recife – PE (SVO-PE), which were removed for routine study and had complete necroscopic examination. The selected age group was 30 years and older, with no upper age limit, and both sexes were evaluated.

The inclusion criterion was a history of essential hypertension, using data obtained from relatives and/or medical-hospital records at the referrals to SVO-PE. Exclusion criteria were defined as findings at autopsy or clinical report of kidney disease; tumors or vascular or endocrine disorders that caused secondary hypertension; ischemic heart disease; tumors or infectious valve disease that could interfere with the thickness or volume of the heart chambers (e.g., acute myocardial infarction and dilated cardiomyopathy); idiopathic or acquired pulmonary hypertension; chronic obstructive pulmonary disease (COPD); recurrent pulmonary thromboembolism and autoimmune diseases that could interfere with right heart thickness[14].

The heart was removed from the mediastinal bed after resection of the great vessels and placed in anatomical position. The highest-volume section was determined, generally coincident with the midpoint between the apex of the heart and the left atrioventricular groove.

At this point, the heart was carefully sectioned through a cross-section, exposing the LV, septum, and RV. The thickest heart slice was selected and used for measurements. A transparent millimeter ruler was used to measure the thickness of the anterior, lateral, and posterior LV (ALV, LLV, and PLV, respectively) and RV (ARV, LRV, and PRV). Endocardial trabeculations, papillary muscle, and epicardial fat were excluded from the measures because they are considered data confounders.

The values were recorded in adequate file cards, as well as data identifying the cadaver (age, race, sex, nutritional status), clinical reports, and other autopsy findings. LV thickness was considered normal up to 1.5 cm[15] and RV thickness up to 0.5 cm[8,15]. Thus, we defined LV hypertrophy (LVH) when the thickness of any of the LV walls reached values above 1.5 cm and RV hypertrophy (RVH) when any RV wall was thicker than 0.5 cm.

All deceased individuals with LV hypertrophy (LV wall thickness□> 1.5 cm) were diagnosed with hypertensive hypertrophic cardiomyopathy (HHC) at the autopsy, which is the terminology used when completing death certificates. The 90 cases were separated into groups according to presence or absence of HHC and its contribution to death as follows:

Group A – Individuals who died due to HHC, with signs of congestive heart failure (polyvisceral congestion, acute pulmonary edema);

Group B – Individuals who had HHC associated with the immediate cause of death, but not as its primary cause;

Group C – Individuals in whom HHC was a minor finding at autopsy;

Group D – Absence of HHC (LV wall thickness □ 1.5 cm).

For data analysis, the absolute and relative distributions were obtained, as well as measurements of central distribution and dispersion. We used the statistical tests of chi-square and Fisher's exact, and the error rate of 5.0% was considered.

The program used for the calculations was the Statistical Package for Social Sciences (SPSS), release 15. The study was approved by the Research Ethics Committee of UFPE (registration No. 119/10, on 06/04/10).

Results

In these 90 cases, the age ranged from 39 to 104 years, with mean of 67.5, median of 70.0, and standard deviation of 15.3 years. The age group of 70-79 years old was the most represented, with 27.8% of individuals. A predominance of female cases (59.9%), mulattoes (60%), and adequate nutritional status (34.1%) was observed.

There was a predominance of male among individuals with RVH and LVH in proportional values. In women, RVH was observed in 37.7% and LVH in 64.2% of cases. In men, these values were 59.5% and 81.1%, respectively. Women were also assessed according to age group (pre- and post-menopausal) and presence of ventricular hypertrophy. Up to 49 years (n = 6), the percentage of women with LVH and/or RVH was 33.4% (n = 2). In women aged 50 years and older (n = 47), this value was 72.3% (n = 34).

The deceased diagnosed with HHC represented by Group A accounted for 20% of the total. Group B was the most prevalent, with 38.9%. The presence of HHC among all groups (A + B + C) was 71.1%. The deceased who did not have HHC (Group D) accounted for 28.9% of all cases.

In the deceased with RVH, a higher prevalence of posterior wall involvement was observed (PRV) corresponding to 30% of the total. In descending order, the lateral wall (LRV) was observed in 24.4% and the anterior (ARV) in 17.8%. In cases with LVH, the lateral wall was the most a□ected (LLV) in 55.6%, followed by anterior (ALV) with 52.2% and posterior (PLV) with 46.7%. It is noteworthy that each organ evaluated could display more than one LV or RV a□ected wall.

Hearts exhibiting LV thickness > 1.5 cm and RV > 0.5 cm accounted for 44.4% of all cases; the lowest percentage found was observed with LV thicknesses □ 1.5 cm and RV > 0.5 cm (2.2%). The di□erence revealed a significant association (p <□0.001) between the presence of LVH and RVH. The same percentage of 26.7% was found for deceased individuals with LV□> 1.5 and RV □ 0.5 cm and for those with LV □ 1.5 cm and RV □ 0.5 cm (Table 1).

Table 2 shows the comparison between groups diagnosed with HHC and RV measurements. It was observed that the highest prevalence of RV thickness > 0.5 cm was found in individuals in Group A (88.9%). This prevalence was lower in Group B (54.3%) and Group C (45.5%). When there was no HHC, the percentage was 7.7%. The observed association between Group A and RVH was statistically significant.

There was a higher prevalence of LVH in deceased individuals aged 60-69 and 70-79 years (86.7% and 76%) of Black or mixed ethnicity (85.7% and 75.9%) and in

Table 1 – Comparison between right and left ventricular wall thickness

□	Evaluation of left ventricle				□		□
Right ventricle	LV > 1.5 cm		LV □ 1.5 cm		Total group		p-value
□	n	%	n	%	n	%	□
RV > 0.5 cm	40	44.4	2	2.2	42	46.7	p[1] < 0.001*
RV ≤ 0.5 cm	24	26.7	24	26.7	48	53.3	
Total	64	71.1	26	28.8	90	100.0	□

* Significant difference at the level of 5.0%; [1]using Pearson's chi-square test.

Table 2 – Right ventricular wall thickness in cadavers, according to the diagnosis of hypertensive hypertrophic cardiomyopathy*

□	Evaluation of right ventricle						
Diagnosis of HHC *	RV > 0.5 cm		RV □ 0.5 cm		Total group		p-value
□	n	%	n	%	n	%	□
Group A	16	88.9	2	11.1	18	100.0	p[1] < 0.001*
Group B	19	54.3	16	45.7	35	100.0	
Group C	5	45.5	6	54.5	11	100.0	
Group D	2	7.7	24	92.3	26	100.0	
Total	42	46.7	48	53.3	90	100.0	□

HHC, hypertensive hypertrophic cardiomyopathy (LV > 1.5 cm); Group A, died due to HHC; Group B, associated HHC; however, not a direct cause of death; Group C, HHC as minor finding at autopsy; Group D, absence of HHC (ALV, LLV or PLV ≤ 1.5 cm); * significant difference at the level of 5.0%; [1] using Pearson's chi-square test.

males (81.1%). In Caucasians, the percentage found was 62.5%. Regarding nutritional status, obese, overweight and normal weight individuals had similar percentages (77.8%, 75%, and 76.7%). In underweight individuals, however, the finding was 43.7%.

For RVH, the highest prevalence was found in the age group of 60-69 years (60%) and in males (59.5%). Among the Black, mixed, and Caucasian ethnicities, we observed percentages of 57.1%, 46.3% and 45.8%, respectively. As for the nutritional status, the highest percentages were seen among those with normal weight, overweight, and obese (53.3%, 50%, 55.3%). In underweight individuals, the prevalence was 25%. Although these percentages of LVH and RVH have been shown to be higher among some variables of the study, considering the margin of error (5%), no significant association was demonstrated.

The values found in the LV and RV after measuring the thickness of the walls varied as follows:

- ALV: 1.0-2.1 cm;
- PLV: 0.9-2.0 cm;
- LLV: 1.0-2.4 cm;
- ARV: 0.2-0.7 cm;
- PRV: 0.2-0.7 cm;
- LRV: 0.1-0.7 cm.

The measurements observed in the LV walls (ALV, PLV and LLV) were divided into grades:

- Normal: LV thickness up to 1.5 cm;
- Grade 1: LV thickness from 1.6 to 1.8 cm;
- Grade 2: LV thickness from 1.9 to 2.1 cm;
- Grade 3: LV thickness from 2.2 to 2.4 cm.

Once the LV values were graded and compared to measurements of the RV wall (ARV, PRV and LRV) and the presence or absence of hypertrophy (Table 3), we observed that, as the left ventricular wall became thicker, the proportion of cases with RV thickness > 0.5 cm increased. This association was statistically significant between the degree of hypertrophy of the ALV in relation to the ARV, PRV, and LRV. It was also observed in LLV compared to PRV. In the remaining walls of LV and RV shown in the Table 3, this association was not confirmed for the margin of error used (5%).

Discussion

In this evaluation, it was observed that 71.1% of the cases had LVH and received the diagnosis of HHC (Groups A + B + C); this percentage is consistent with the maximum rates reported in the literature, in which up to 75% of hypertensive patients had left ventricular hypertrophy, depending on the pressure levels and therapeutic regimens[16,17]. Among all the deceased individuals that showed LVH, 44.4% also exhibited RVH; this was one of the findings with statistical significance observed in this study. Significant association was also observed by Gottdiener et al. who found RVH in up to 80% of patients who had LVH[10].

Among the a□ected walls, there was a predominance of hypertrophy in PRV and LLV. The data di□er from those observed by other authors, among them Cicala et al.[18] who showed prevalence of hypertrophy in ARV and PLV.

The mechanisms of right hypertrophy have not been fully elucidated, but several factors have been proposed: 1) There would be transmission of tension experienced by the LV to the RV due to the existence of myocardial fibers common to both structures, as they share the interventricular septum and are limited by the pericardial sac; 2) Neurohumoral factors would mediate ventricular hypertrophy in response to pressure overload in one of them; 3) As a result of increased volume in the LV, diastolic pressure would increase in the RV; 4) Abnormal emptying of the hypertrophic LV, re□ecting the reduced compliance in the disease, would be a determinant of the increased volume imposed on the RV, leading to RVH [8-10,12,18-20].

The presence of LVH and/or RVH was higher in postmenopausal women, when compared to premenopausal women, with this finding being in agreement with those reported in the literature. Cardiac hypertrophy is more prevalent in menopausal patients, a consequence of the decrease in female sex hormones, which are heart protective[3]. As the number of premenopausal women in this study was reduced (n = 6), despite the suggestion, there is insu□ cient evidence to assess this finding with statistical significance.

There was a higher prevalence of LVH in certain variables (Black or mixed ethnicity, male, and aged 60-79 years), although not statistically significant. For RVH, the highest values were observed in the deceased aged 60-69 years and in males. The literature confirms in part the findings obtained in this work. There is a higher prevalence of hypertension and LVH among men compared to the same prevalence in premenopausal women. Left hypertrophy is approximately two-fold more prevalent in Black patients than in Caucasians, especially when one takes into account the foreign series[3]. It has also been observed that it is more frequently diagnosed in elderly patients[1].

The miscegenation in Brazil makes ethnicity a criterion of lesser validity for assessing the risk of developing heart disease[21]. Although the highest prevalence has been observed in Blacks and Brazilian mulattoes with LVH, the percentage di□erence in comparison to Caucasians was small (85.7%, 75.9%, and 62.5%, respectively). For RVH, this di□erence was even lower (57.1%, 46.3%, and 45.8%, respectively). In an autopsy study, Lessa et al.[22] also observed that there was no predominance of LVH in Black patients evaluated.

Table 3 – Right ventricle wall thickness (ARV, PRV and LRV) according to the degree of LV wall thickness (ALV, LLV, PLV)

	ALV								
RV wall	Normal§		Grade 1§		Grade 2§		Total		p-value
Thickness (cm)	n	%	n	%	n	%	n	%	
ARV									
> 0.5	1	1.5	9	10.0	6	6.7	16	17.8	$p^1 < 0.001$*
≤ 0.5	42	46.7	25	27.8	7	7.8	74	82.2	
PRV									
> 0.5	3	3.3	17	18.9	7	7.8	27	30.0	$p^1 < 0.001$*
≤ 0.5	40	44.4	17	18.9	6	6.7	63	70.0	
LRV									
> 0.5	3	3.3	12	13.3	7	7.8	22	24.4	$p^1 < 0.001$*
≤ 0.5	40	44.4	22	24.4	6	6.7	68	75.6	
Total	43	47.8	34	37.8	13	14.3	90	100.0	

	LLV										
RV wall	Normal§		Grade 1§		Grade 2§		Grade 3§		Total		p-value
Thickness (cm)	n	%	n	%	n	%	n	%	n	%	
ARV											
> 0.5	5	5.6	5	5.6	4	4.4	2	2.2	16	17.8	$p^2 = 0.023$*
≤ 0.5	35	38.9	30	33.3	9	10.0	–	–	74	82.2	
PRV											
> 0.5	3	3.3	18	20.0	4	4.4	2	2.2	27	30.0	$p^2 < 0.001$*
≤ 0.5	37	41.1	17	18.9	9	10.0	–	–	63	70.0	
LRV											
> 0.5	3	3.3	13	14.4	5	5.6	1	1.1	22	24.4	$p^2 = 0.003$*
≤ 0.5	37	41.1	22	24.4	8	8.9	1	1.1	68	75.6	
Total	40	44.4	35	38.9	13	14.4	2	2.2	90	100.0	

	PLV								
RV wall	Normal§		Grade 1§		Grade 2§		Total		p-value
Thickness (cm)	n	%	n	%	n	%	n	%	
ARV									
> 0.5	8	8.9	6	6.7	2	2.2	16	17.8	$p^2 = 0.251$
≤ 0.5	40	44.4	32	35.6	2	2.2	74	82.2	
PRV									
> 0.5	7	7.8	18	20.0	2	2.2	27	30.0	$p^2 = 0.002$
≤ 0.5	41	45.6	20	22.2	2	2.2	63	70.0	
LRV									
> 0.5	9	10.0	11	12.2	2	2.2	22	24.4	$p^2 = 0.208$
≤ 0.5	39	43.3	27	30.0	2	2.2	68	75.6	
Total	48	53.3	38	42.2	4	4.4	90	100.0	

RV. right ventricle; ARV. anterior right ventricle; PRV. posterior right ventricle; LRV. lateral right ventricle; ALV. anterior left ventricle; LLV. lateral left ventricle; PLV. posterior left ventricle. [1] Pearson's chi-square test; [2] Fisher exact test; § degrees of LV wall thickness: normal – LV up to 1.5 cm. grade 1 – LV from 1.6 to 1.8 cm. grade 2 – LV from 1.9 to 2.1 cm. grade 3 – LV from 2.2 to 2.4 cm.

Obese individuals are certainly more susceptible to developing hypertension and HHC[1,3]. Regarding the nutritional status, both cases with LVH and RVH showed to be predominant in deceased individuals with normal weight, overweight or obese, with no statistically significant difference between groups and, similarly, when compared to underweight individuals. Therefore, we could not state such association in this study.

The literature often addresses the influence of anthropometric data and body mass index on cardiac mass, particularly in heart weight assessments. Several authors consider the physical constitution of the patients (weight and height) when interpreting their results[23-25]. Rodrigues stressed in 2006 that all assessments of cardiac mass should be adjusted to the values found in autopsies for the population to be studied. They vary with age, sex, ethnicity, body size, and composition[26]. Salton et al.[27], in a study using magnetic resonance in a cardiovascular disease-free population, observed that the constitution did not influence only the ventricular mass, but also the volume and linear dimensions of the heart. Thus, although this is not the assessment of cardiac mass and weight, anthropometric data that were not obtained in this study should be considered in further studies.

Deceased individuals categorized as Group A in the diagnosis of HHC (considered as having heart disease of poor prognosis, as it was related to congestive heart failure) had RVH in 88.9% of cases. The observed association was significant in statistical tests. Based on these results, it is suggested that the presence of RVH is related to more severe hypertensive disease. The value of this association may indicate the existence of a new prognostic factor for the categorization of hypertensive patients.

Regarding the degree of LV wall hypertrophy and its association with the presence of RV wall hypertrophy, it was observed that the greater the thickness of LV, the greater the proportion of cases with RV thickness > 0.5 cm. These findings are corroborated by the literature in which some authors have observed the same linear association between LV wall thickness and presence of RVH[9,10,18]. Despite the higher percentages of RVH according to the increased LV walls thickness, there was no statistically significant association for all ventricular walls considered in the study. This is probably due to the small number of cases evaluated.

Study conclusions and limitations

Based on the findings of this study, we conclude that RVH is associated with a more severe hypertensive disease; therefore, it should be taken into account in the routine assessments of hypertensive patients. It may be a new prognostic factor, which requires the evaluation of a larger sample to be better studied and recognized as such. Another point of significance is related to the observed association between the presence of LVH and RVH; this finding corroborates the theories that postulate common pathogenic factors related to the development of bilateral hypertrophic disease.

Due to the lack of more reliable data sources, such as medical records, the information reported by family members were used. These data are routinely used to complete additional information in death certificates (one example is the reporting on lifestyle and preexisting conditions, such as smoking, alcohol consumption, diabetes mellitus). Regarding physical type or nutritional status, the categorization of the deceased individuals was obtained from reports of autopsies performed by pathologists, corresponding to subjective evaluations. As this is a study of linear dimensions (thickness) of the cardiac walls, we decided not to assess the weight of the ventricles due to the natural difficulty in separating the interventricular septum between left and right myocardium. Knowing these limitations, it is expected that further work on the subject will be able to address them.

References

1. Sociedade Brasileira de Cardiologia. Sociedade Brasileira de Hipertensão. Sociedade Brasileira de Nefrologia. VI Diretrizes Brasileiras de Hipertensão. Arq Bras Cardiol. 2010;95 (Supl.1):1-51. [cited 2010 Feb 12]. Available from: http://publicacoes.cardiol.br/consenso/2010/Diretriz_hipertensao_associados.pdf.
2. Barrios V, Escobar C, Calderón A, Barrios S, Navarro-Cid J, Ferrer E, et al. Gender diόerences in the diagnosis and treatment of left ventricular hypertrophy detected by diόerent electrocardiographic criteria. Findings from the SARA study. Heart Vessels. 2010;25:51-6.
3. Batlouni M. Hipertrofia cardíaca. Fatores determinantes e mecanismos moleculares. Arq Bras Cardiol. 1995;65:533-9.
4. Frimm CC. Editorial: mecanismos de hipertrofia cardíaca na hipertensão arterial. [cited 2010 Jan 10]. Available from: http://www.jbn.org.br/audiencia_pdf. asp?aid2=1017&nomeArquivo=1702-02.doc.
5. Chahal NS, Lim TK, Jain P, Chambers JC, Kooner JS, Senior R. New insights into the relationship of left ventricular geometry and left ventricular mass with cardiac function: a population study of hypertensive subjects. Eur Heart J. 2009;19:1-7. [cited 2010 jan. 10]. Available from: http://eurheartj.oxfordjournals.org/content/early/2009/11/19/eurheartj.ehp490.abstract.
6. Maciel BC. A hipertrofia cardíaca na hipertensão arterial sistêmica: mecanismo compensatório e desencadeante de insuficiência cardíaca. Rev Bras Hipertens. 2001;8:409-13.
7. Chakko S, Marchena E, Kessler KM, Materson BJ, Myerburg RJ. Right ventricular diastolic function in systemic hypertension. Am J Cardiol. 1990;65:1117-20.
8. Silva RP, Amodeo C, Ramires JAF. O ventrículo direito e a hiper-tensão arterial: aspectos ecocardiográficos. Arq Bras Cardiol. 2002;79:313-8.
9. Nunez BD, Messerli FH, Amodeo C, Garavaglia GE, Scmieder RE, Frolich ED. Biventricular cardiac hypertrophy in essential hypertension. Am Heart J. 1987;813-8.
10. Gottdiener JS, Gay JA, Maron BJ, Fletcher RD. Increased right ventricular wall thickness in left ventricular pressure overload: echocardiographic determination of hypertrophic response of the "nonstressed" ventricle. J Am Coll Cardiol. 1985;6:550-5.
11. Pereira R, Ramires JA, Amodeo C, Pontes S, Assef J. Análise ecodopplercardiográfica das alterações morfológicas e funcionais causadas pela hipertensão arterial no ventrículo direito. [cited 2010 Jan 10]. Available from: http://www.siicsalud.com/dato/ experto.php/6922.
12. Akkoç A, Uçaman B, Kaymak H, Temamogullari AV, Iltümür K, Karadede A, et al. Right and left ventricular diastolic filling parameters in essential hypertension. Asian Cardiovasc Thorac Ann. 1999;7:214-20.
13. Klima U, Guerrero JL, Vlahakes GJ. Contribution of the interventricular septum to maximal right ventricular function. Eur J Cardiothorac Surg. 1998;14:250-5.
14. Kumar V, Abbas AK, Fausto N. Robbins & Cotran. Patologia: bases patológicas das doenças. 7ª ed. Rio de Janeiro: Elsevier; 2005. p.589;781.
15. Baker RD. Técnicas de necropsia. Tradução de Orestes Pereyra M. 2ª ed. Rio de Janeiro: Editorial Interamericana; 1992.
16. Santello JL. Hipertensão arterial e hipertrofia ventricular. Arq Bras Cardiol. 1992;59:63-8.

17. Tenenbaum A, Fisman EZ, Schwammenthal E, Adler Y, Benderly M, Motro M, et al. Increased prevalence of left ventricular hypertrophy in hypertensive women with type 2 diabetes mellitus. Cardiovasc Diabetol. 2003;2:14.
18. Cicala S, Galderisi M, Caso P, Petrocelli A, D'Errico A, Divitiis O, et al. Right ventricular diastolic dysfunction in arterial systemic hypertension: analysis by pulsed tissue doppler. Eur J Echocardiography. 2002;3:135-42.
19. Myslinski W, Mosiewicz J, Ryczak E, Barud W, Biãan A, Palusinski R, et al. Right ventricular function in systemic hypertension. J Hum Hypertens. 1998;12:149-55.
20. Nogueira JB, Mourão L, Morais C, Freitas A, Silva D, Soares-Costa JTS, et al. Hipertensão arterial e coração direito. Estudo hemodinâmico e ecocardiográfico. Rev Port Cardiol. 1991;10:837-43.
21. Latado AL, Lopes MB, Passos LCS, Lopes AA. Existe evidência para tratar insuficiência cardíaca baseada na raça ou etnia?. Rev Assoc Med Bras. 2009;55:110-6.
22. Lessa I, Fonseca ACTG, Santos AG. Raça e hipertrofia do ventrículo esquerdo na ausência de hipertensão e de doenças cardiopulmonares. Arq Bras Cardiol. 1994;62:413-6.
23. Rodrigues SL, Pimentel EB, Mill JG. Peso dos ventrículos cardíacos determinado em necropsia de indivíduos saudáveis mortos por causas externas. Arq Bras Cardiol. 2007;89:279-84. [cited 2010 Jan 10]. Available from: http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0066-782X2007001700001.
24. Mancia G, Carugo R, Grassi G, Lanzarotti A, Schiavina R, Cesana G, et al. Prevalence of left ventricular hypertrophy in hypertensive patients without and with blood pressure control: data from the PAMELA population. Hypertension. 2002;39:744-9.
25. Cardoso E, Martins IS, Fornari L, Monachini MC, Mansur AP, Caramelli B. Alterações eletrocardiográficas e sua relação com os fatores de risco para doença isquêmica do coração em população da área metropolitana de São Paulo. Rev Assoc Med Bras. 2002;48:231-6.
26. Rodrigues SL. Hipertrofia do ventrículo esquerdo: correlação entre critérios eletrocardiográficos e ecocardiográficos em estudo de base populacional. [PhD thesis]. Vitória: Centro de Ciências Humanas da Universidade Federal do Espírito Santo; 2006.
27. Salton CML, Chuang CJ, O´Donnell MJ, Kupka MG, Larson KV, Kissinger RR, et al. Gender diã erences and normal left ventricular anatomy in an adult population free of hypertension: a cardiovascular magnetic ressonance study of the Framingham Heart Study Oã spring Cohort. J Am Coll Cardiol. 2002;39: