Relationship between left ventricular hypertrophy and somatotype of high performance athletes using structural equations modeling

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Summary

Introduction: Sports induce morphological and functional adaptations in the human heart that directly relate to the type, duration and intensity of training and the years of practice. These changes are present in different ways in the electrocardiogram. A high QRS voltage is the most significant finding. Its correlation with left ventricular hypertrophy is low. In this study, the aim was to determine if a relationship exists between electrocardiographic alterations of left ventricular hypertrophy and somatotype in high performance athletes.

Methods: A retrospective, cross-sectional, quantitative, multiple correlation, observational and analytical study of a database of 180 resting electrocardiograms and anthropometric evaluations of men´s soccer, women´s soccer, swimming, cycling, basketball, and tennis athletes was performed. A database containing somatotype and Sokolow-Lyon electrocardiographic voltage criteria was created.

Results: The study group was composed of 83.3% men and 16.7% women. Age ranged from 10 to 51 years with a mean of 19.73 ± 5.8. Weight ranged from 35.90 to 122.3 kg with a mean of 66.98 ± 12.67 and height ranged from 143 to 213 cm with a mean of 174.11 ± 10.29 cm. Endomorphy for the entire group ranged from 1.0 to 5.7 with a mean of 2.5 ± 0.9. Mesomorphy ranged from 1.6 to 7.1 with a mean of 4.2 ± 0.95. Ectomorphy ranged from 1.1 to 5.8 with a mean of 2.9 ± 0.96. The structural equation model had a normal multivariable distribution of 3.161, reaching a Pearson of .26 for mesomorphy with a goodness of fit and a variance of 0% for mesomorphy and left ventricular hypertrophy.

Conclusion: Based on the findings, we can say that somatotype does not predict left ventricular hypertrophy in high performance athletes.

Key words:
Endomorphy. Ectomorphy. Mesomorphy. Structural equation model. Cardiac sudden death.

Relación entre la hipertrofia ventricular izquierda y el somatotipo en atletas de alto rendimiento utilizando modelamiento de ecuaciones estructurales

Resumen

Introducción: Los deportes inducen adaptaciones morfológicas y funcionales en el corazón humano directamente relacionadas con el tipo, duración e intensidad del entrenamiento y los años de práctica. Estos cambios se manifiestan de diversas formas en el electrocardiograma. Un alto voltaje del QRS es el hallazgo más significativo. Su correlación con la hipertrofia ventricular izquierda es baja. En este estudio, el objetivo era determinar si existe una relación entre las alteraciones electrocardiográficas de hipertrofia ventricular izquierda y el somatotipo en deportistas de alto rendimiento.

Métodos: Se efectuó un estudio transversal, cuantitativo, observacional, analítico retrospectivo de correlación múltiple de una base de datos de 180 electrocardiogramas en reposo y antropometría de atletas de soccer varonil, soccer femenil, natación, basquetbol, ciclismo y tenis. Se creó una base de datos con el somatotipo y los criterios de voltaje electrocardiográfico de Sokolow-Lyon.

Resultados: El grupo de estudio estaba compuesto por 83,3% varones y 16,7% mujeres. El rango de edad fue de 10 a 51 años con una media de 19,73 ± 5,8. El peso varió de 35,90 a 122,3 kg con una media de 66,98 ± 12,67 y la estatura varió de 143 a 213 cm con una media de 174,11 ± 10,29 cm. Endomorfia para todo el grupo osciló entre 1,0 y 5,7 con una media de 2,55 ± 0,9. Mesomorfia varió de 1,6 a 7,1 con una media de 4,2 ± 0,95. Ectomorfia varió de 1,1 a 5,8 con una media de 2,9 ± 0,96. El modelo de ecuaciones estructurales tenía una distribución multivariable normal de 3.161, alcanzando un Pearson de 0,26 para mesomorfia con una bondad de ajuste y una varianza de 0% para mesomorfia e hipertrofia ventricular izquierda.

Conclusiones: En base a los hallazgos podemos decir que el somatotipo no predice hipertrofia ventricular izquierda en atletas de alto rendimiento.

Palabras clave:
Endomorfia. Ectomorfia. Mesomorfia. Modelo de ecuaciones estructurales. Muerte súbita cardiaca.

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Introduction

Sports activity produces a series of morphological and functional adaptations in the human heart directly related to the type, duration, and intensity of the training and years of sports practice. These changes are seen in different ways in an electrocardiogram. A high QRS voltage is the most significant finding in male athletes. Its correlation with the presence of left ventricular hypertrophy is low[1].

The athlete's heart has intrigued physicians and scientists for over a century. Initial investigations date back to the late 1800s and early 1900s where an enlarged heart and bradyarrhythmias were documented in individuals with maximum oxygen consumption (VO_2) above normal, with no concomitant signs of cardiovascular disease[2].

In Europe, in 1899, the Swedish physician Henschen, using only his physical examination skills of auscultation and percussion, showed increased cardiac dimensions in elite Nordic skiers[3]. Similar findings were made by Darling[4] of Harvard University in college rowers. Later, White[5] described sinus bradycardia at rest in long distance runners and other athletes.

Since then, numerous studies using new methods have confirmed that the athlete's heart has manifestations of chronic adaptations to endurance training. The concentric growth observed is the result of an increase in the size of the heart chambers and the thicknesses of their walls. These changes were called physiological cardiac remodeling by Kindermann[6], Baggish and Wood[7] which refers to cardiac remodeling as a complex process influenced by multiple factors such as the athlete's age, gender, ethnicity, genetics, type of sport and body size[8,9].

Even though these changes are observed mainly in adult athletes, adolescent athletes who practice endurance sports also present greater left ventricular growth than non-athletes of the same age as demonstrated by Sharma[10].

Hypertrophic cardiomyopathy is a common cause of sudden death in apparently healthy athletes, and this condition is often a differential diagnosis with adaptive heart changes in athletes[11]. The importance of its early detection is one of the objectives of ergometric measurements, even if we do not yet have definitive diagnostic tests or gold standards as mentioned by Weinstock[12]. One of the most valid and reliable methods is the M-mode echocardiogram (ECHO) as reported by Devereaux *et al.*[13], despite considering that magnetic resonance imaging (MRI) has more than twice the accuracy over ECHO and being a more precise and reliable method for measuring LVH[14]. Even though these methods are more sensitive and precise, their high cost and limited availability provide an obstacle for routine use. Although an ECHO is less precise than MRI, an electrocardiogram (ECG) can serve as a less expensive, practical, and widely available alternative for LVH screening.

The ECG has the potential to accurately distinguish between physiological and pathological hypertrophy, since ECG abnormalities in hypertrophic cardiomyopathy slightly overlap with ECG voltage changes. In patients with hypertrophic cardiomyopathy (HCM), pathological hypertrophy of the left ventricle (LV) is associated with additional criteria, not only left atrial voltage and dilation, but also left axis deviation, and T wave, ST segment and Q wave alterations[15].

Kinanthropometry is a discipline that studies the size, shape and composition of the human body. Physical activity, nutrition, growth and race, among other variables, can alter an individual´s body composition[16]. For this, different measurements of size and proportions of the body are performed to determine body composition[17]. The Heath-Carter method is most frequently used to determine somatotype[18].

Hense *et al.*[19] demonstrated the influence of body composition on the size of the adult heart. The MESA (Multi-Ethnic Study of Atherosclerosis) study demonstrated a relationship between LV mass and end-diastolic volume with an increased body mass index, waist-to-hip ratio, waist circumference, and fat percentage; however, the ejection fraction showed no significant association with obesity measures[20].

Guerra *et al.*[21] studied 380 patients with essential hypertension, obesity and/or overweight and metabolic syndrome who suffered a problem with adequate control of their blood pressure. This study found that hypertensive patients with metabolic syndrome had a higher BMI and also a higher mean arterial and systolic pressure as well as greater thickness of the septum and of the interventricular wall together with an ejection fraction smaller than that of those without metabolic syndrome.

It is necessary to know the electrocardiographic manifestations of the athlete´s heart and determine if there is a direct relationship between the somatotype, the type of sport performed, and the electrocardiographic alterations of left ventricular hypertrophy. Therefore, the objective of this investigation was to determine if there is a relationship between electrocardiographic alterations related to left ventricular hypertrophy (LVH) and somatotype in high-performance athletes of various sports.

Material and method

This was a retrospective, cross-sectional, quantitative, multiple correlation, observational and analytical study of a database of resting electrocardiograms and anthropometrics evaluated in the Department of Sports Medicine and Physical Rehabilitation of men´s soccer, women´s soccer, swimming, cycling, basketball, and tennis athletes. Individuals with a history of previous heart disease and with electrocardiograms and anthropometric measures that were not legible were excluded from the evaluation. Athlete records that did not have anthropometry and/or an electrocardiogram were eliminated.

The measuring instrument for the dependent variable, left ventricular hypertrophy, was the electrocardiographic voltage criteria of the Sokolow-Lyon index for left ventricular hypertrophy[22]. These consist of the sum of the S wave in V1 and the R wave in V5 or V6 ≥ 3.5 mV (35 mm) and/or an R wave in aVL ≥ 1.1 mV (11 mm).

To measure the independent variable, somatotype, measures of weight, height, skinfolds, girth and breadth were obtained using the restricted profile of anthropometric measures in accordance with the recommendations of the International Society for the Advancement of Kinanthropometry (ISAK)[23].

Sample size

Structural models need large samples with more parameters to estimate those that work with larger samples (24). Some authors, such as Hu, Bentler, & Kano[25] and Schreiber, Nora, Stage, Barlow, and King[26]

propose as a basic rule the choice of 10 observations per indicator as a lower limit of sample sizes. Jackson[27] suggests a relation N:q, 20 to 1 where N equals 20 and q is the number of parameters in the model. However, it is important to mention that no rule can be applied to all cases and adequate sample size depends on many factors, including the psychometric properties of the variables, the strength of the relationships between variables considered model size, and the distribution characteristics of the variables. In the present research, we used the criteria proposed by Jackson. In this case there are 8: Endo, Meso, Ecto and SV1, SV2, RV5, RV5, RAVL, multiplied by 20 equals 160 participants.

For the statistical analysis, SPSS version 21 for Windows (IBM Corp., Armonk, NY) was used. For structural equation modeling, Amos 21 was used to verify the relationships between observed and unobserved (latent) variables and test the hypothesis and confirm relationships. Diverse statistical tests were performed as part of a multivariate analysis: determination of Mardia´s coefficient[28], which seeks multivariate normality, considered one of the most common assumptions of the distribution of normality in multivariate analysis, analysis of the Mahalanobis distance[29], multiple regression analysis, chi square analysis, and goodness of fit. The final sample consisted of 180 individuals.

Once the constructs were evaluated, SEM was used to quantify and test the validity of hypothetical assertions, possible interrelationships between constructs, and the relationship with evaluation measures (Figure 1).

Results

The study group was composed of 83.3% men and 16.7% women, with a mean Age 19.73± 5.8, weight ranged from 35.90 to 122.3 kg with a mean of 66.98 ± 12.67 and height ranged from 143 to 213 cm with a mean of 174.11 ± 10.29 cm.

Endomorphy of the entire group ranged from 1.0 to 5.7 with a mean of 2.55 ± 0.9. Mesomorphy ranged from 1.6 to 7.1 with a mean of 4.2 ± 0.95. Ectomorphy ranged from 1.1 to 5.8 with a mean of 2.9 ± 0.96. (Table 1).

Regarding left ventricular hypertrophy factors, S in V1 ranged from 0.0 to 25.0 mm with a mean of 9.3 ± 4.9; S in V2, ranged from 0.0 to 38.0 mm with a mean of 16.7 ± 7.3; R in V5 ranged from 1.5 to 32 mm with a mean of 16.1 ± 6.2; R in V6 ranged from 1.5 to 30 mm,

Figure 1. Proposed model to estimate LVH represented by SV1, SV2, RV5, RV6, RaVL, (endogenous variable) and Somatotype represented by endomorphy, mesomorphy and ectomorphy (exogenous variable), e (error).

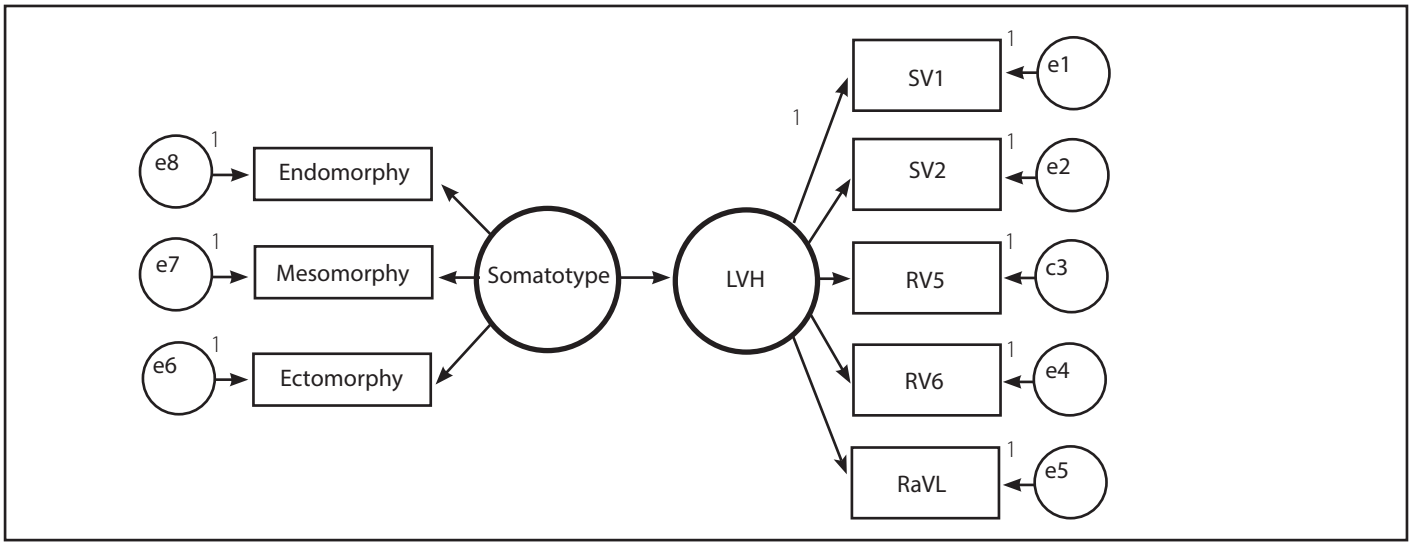


Table 1. Demographic characteristics of the athletes.

	Gender	Age, years	Body weight, kg	Height, cm	Endomorphy	Mesomorphy	Ectomorphy
Men	Mean	19.71	69.1645	176.257	2.416	4.387	2.986
N = 150	SD	5.912	12.30439	9.5152	.8557	.9036	.9525
Women	Mean	19.80	56.0667	163.397	3.207	3.530	2.830
N = 30	SD	5.242	8.14758	6.8107	.8741	.8272	1.0232
Total	Mean	19.73	66.9816	174.114	2.548	4.244	2.960
N = 180	SD	5.792	12.67829	10.2947	.9058	.9450	.9634

SD: standard deviation.

Table 2. Somatotype by sport.

	Sport	Endomorphy	Mesomorphy	Ectomorphy
Soccer	Mean	2.424	4.427	2.898
N = 126	SD	.8109	.8292	.8650
Cycling	Mean	3.720	4.200	2.220
N = 5	SD	1.0756	1.1000	.3834
Swimming	Mean	2.837	3.653	3.808
N = 19	SD	1.2868	1.0611	1.2640
Women's Soccer	Mean	3.106	3.522	2.600
N = 18	SD	.5846	.7256	.8007
Tennis	Mean	2.200	2.733	3.933
N = 3	SD	.8000	.9815	.7638
Basketball	Mean	2.022	4.911	2.844
N = 9	SD	.7345	.9239	1.0620
Total	Mean	2.548	4.244	2.960
N = 180	SD	.9058	.9450	.9634

SD: standard deviation.

Table 3. Presence of left ventricular hypertrophy (LVH) by sport.

Sport	Frequency	Percent	Cumulative Percent
Soccer	61	74.4	74.4
Swimming	14	17.1	91.5
Women's Soccer	4	4.9	96.3
Basketball	2	2.4	98.8
Cycling	1	1.2	100.0
Total	82	100.0	

Table 4. Presence of left ventricular hypertrophy (LVH) in athletes by age.

Age, years	Frequency	Percent with LVH	Cumulative Percent
15	12	14.6	14.6
18	12	14.6	29.3
17	11	13.4	42.7
19	9	11.0	53.7
13	5	6.1	59.8
16	5	6.1	65.9
24	5	6.1	72.0
14	4	4.9	76.8
20	3	3.7	80.5
30	3	3.7	84.1
12	2	2.4	86.6
26	2	2.4	89.0
27	2	2.4	91.5
10	1	1.2	92.7
11	1	1.2	93.9
21	1	1.2	95.1
25	1	1.2	96.3
28	1	1.2	97.6
33	1	1.2	98.8
51	1	1.2	100.0
Total	82	100.0	

Table 5. Regression Weights: (Group number 1 - Default model).

			Estimate	S.E.	C.R.	P	Label
LVH	<---	Meso	.263	.135	1.940	.052	par_3
SV1	<---	LVH	1.000				
RV5	<---	LVH	5.626	2.610	2.155	.031	par_1
RV6	<---	LVH	4.755	2.011	2.365	.018	par_2

SE: standard error; CR: critical ratio; P: bilateral asymptotic significance.

with a mean of 13.2 ± 4.7; and R in aVL ranged from 0.0 to 5.0 mm with a mean of 0.8 ± 0.98.

The sports and mean somatotypes of the sample for a total of 180 individuals are shown in Table 2. Of these, 82 (46.6%) had voltage criteria for LVH. The distribution of LVH by sport and age are shown in Table 3 and Table 4 respectively.

In Table 5, in the column critical ratio, it is seen that the loads of the indicators are significant. After evaluating several models, both the endogenous variable LVH and the exogenous variable somatotype, through a confirmatory factor analysis for LVH with the 5 factors shown in Figure 1, R in aVL and S in V2 were eliminated and a correlation between two residues was introduced to improve fit. In addition, the model had to be restructured because somatotype was not coherent as a construct in this study (Figure 2). Of all the models, it was this one that presented the best fit to the data and with this the maximum likelihood estimation (MLE) was performed.

Regarding standardized regression weights, mesomorphy positively impacts LVH with a regression of 0.259. S in V1 in relation to LVH has a weighted regression of 0.197 with a positive correlation and low weight. R in V5 in relation to LVH has a weighted regression of 0.868. R in V6 in relation to LVH has a weighted regression of 0.961 with a positive correlation and high weight.

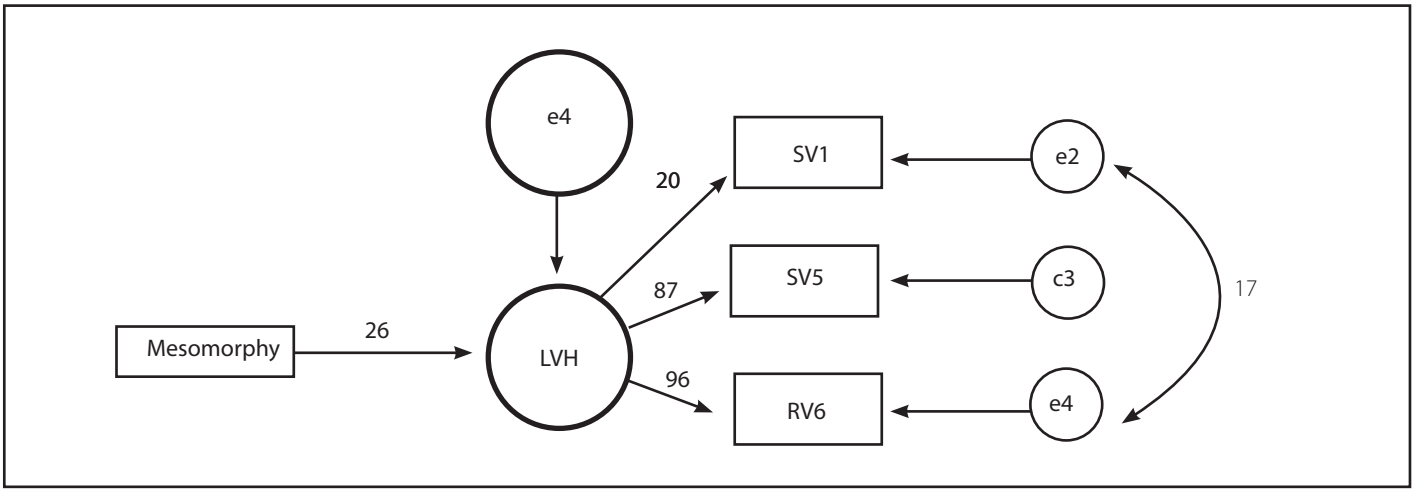
Discussion

The somatotype and its components, endomorphy, mesomorphy and ectomorphy, were not congruent as a construct in this study. Mesomorphy was the component that presented a direct and proportional relationship with LVH, without this being a statistically significant factor in the prediction of LVH in the study participants. In fact, somatotype was the most problematic factor, since it not only showed low loadings which caused a loss of endomorphy and ectomorphy; therefore, we could not speak strictly of somatotype.

The dominant component of this group was mesomorphy since all participants were athletes. The somatotype category for the female athletes was mesomorph-endomorph, and for male athletes it was balanced mesomorph. In relation with the sport 73.6% of the swimmers, 48% of the men´s soccer players, 22% of the women´s soccer players, 22% of the basketball players and 20% of the cyclists had LVH; 50% of LVH cases were younger than 18 years old.

The structural model reported a normal multivariate distribution of 3.161. As for the structural relationship between the exogenous and endogenous variables, the model reached a Pearson of 0.26 for meso-

Figure 2. Standardized regression weights: (Group number 1–Default model). The coefficient above each path is AMOS's maximum likelihood estimate of the effect size.



morphy. This is interpreted as a positive correlation of low weight with good goodness of fit and a proportion of explained variance of 0% for the relationship between mesomorphy and LVH indicating that no linear combination of the independent variables is a better predictor than the fixed mean of the dependent variable. The components that explained or diagnosed LVH were R in V5, 75.4%, and R in V6, 92.3%, while S in V1 only explained 6.9%.

As mentioned by Baggish and Wood and Escudero and Pinilla[7,30], cardiac hypertrophy is a combination of genetic, physiological and environmental factors. The underlying molecular mechanisms that induce physiological or pathological responses are not yet fully elucidated.

Unlike patients with hypertension in which a high correlation with body composition, BMI, and waist-hip ratio is observed, athletes do not have this relationship[19,21].

Conclusion

Mesomorphy was the dominant component and the one related to LVH. Based on the findings in this study we can say that somatotype has no utility in predicting left ventricular hypertrophy in athletes. Since 50% of LVH cases were under 18 years of age, we are obliged to continue with the ECG for the detection and subsequent study of these athletes to prevent possible complications in the long or medium term.

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