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THE INFLUENCE OF DIETARY SODIUM MODULATION ON ELECTROCARDIOGRAPHIC VOLTAGE CRITERIA FOR LEFT VENTRICULAR HYPERTROPHY IN NORMOTENSIVE INDIVIDUALS

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Abstract

Objective—Dietary sodium intake and left ventricular hypertrophy (LVH) on electrocardiogram (ECG) are both independent determinants of cardiovascular risk. Prior studies demonstrated that acute dietary sodium modulation significantly altered LVH-specific ECG voltage in hypertensives, thus modifying cardiovascular risk prediction; but whether this phenomenon exists in normotensive individuals is not known. We evaluated the influence of dietary sodium intake on ECG voltage and ECG criteria for LVH in normotensives.

Methods—Retrospective evaluation of ECG's of healthy normotensives (n=39) who were prospectively randomized to a dietary study protocol of 1 week of high sodium (HS) diet (>200 mmol Na/day) and 1 week of low sodium (LS) diet (<10 mmol Na/day) was conducted. ECG voltage amplitudes and biochemical assessments were performed at the end of each dietary intervention.

Results—As expected, blood pressure declined and measures of circulating renin-angiotensin-aldosterone system activity rose significantly with LS diet. No significant changes in specific LVH voltage criteria, or overall precordial or limb lead ECG voltage amplitudes, were detected between diets.

Conclusion—Although acute dietary sodium modulation has been shown to significantly alter LVH-specific ECG voltage and the detection of LVH in hypertensives, dietary sodium intake did not influence ECG voltage in normotensives. Healthy normotensive individuals may exhibit adaptive measures that dampen ECG voltage fluctuations in response to dietary sodium modulation. More specific cardiac imaging studies may provide additional insight into this observation and the influence of dietary sodium in cardiac health.

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Keywords

Left ventricular hypertrophy; dietary sodium; electrocardiogram; renin angiotensin aldosterone system

INTRODUCTION

Electrocardiogram (ECG) determined left ventricular hypertrophy (LVH) is a known independent predictor of cardiovascular morbidity and mortality [1–4]. Prior investigations have observed that higher ECG voltage is associated with worsening cardiovascular risk [5], whereas regression of LVH voltage criteria on ECG improves risk [6,7]. These studies reaffirmed that while the ECG is not the most sensitive tool in detecting LVH [8,9], its high specificity is still strongly associated with cardiovascular risk profiles.

High dietary sodium intake is a risk factor for developing high blood pressure and an independent predictor of left ventricular mass [10–15]. In contrast, *chronic* dietary sodium restriction has been shown to improve blood pressure and reduce cardiovascular outcomes [11,12]. We previously observed that *acute* dietary sodium restriction significantly reduced ECG voltage amplitude and the prevalence of ECG-voltage defined LVH in a cohort of hypertensive individuals [16]. This unique observation demonstrated marked changes in ECG voltage with only one week of dietary sodium modulation; while prolonged dietary sodium modulation may result in structural myocardial adaptation [15], we speculated that acute dietary sodium restriction influenced ECG-determined LVH voltages by altering intravascular and cardiac chamber volumes [16,17]. Since the detection of LVH on ECG portends higher cardiovascular risk, these prior findings suggested that assessment of dietary sodium intake in hypertensives may be an important consideration when interpreting the ECG. However, whether this phenomenon of acute dietary sodium intake influences LVH specific ECG voltages in normotensives is unknown.

To extend our prior evaluation in a hypertensive cohort [16], we investigated whether randomization to acute dietary sodium modulation altered ECG voltage amplitude, and specifically LVH voltage criteria on ECG, in a population of normotensive individuals. Differential ECG responses to acute dietary sodium intake between hypertensive and normotensive individuals might identify unique myocardial physiologic behaviors with potential clinical implications in the interpretation of screening ECG's. Herein, we report the retrospective analysis of 39 paired ECG assessments in normotensives who completed an acute dietary modulation study protocol that meticulously controlled dietary sodium, potassium, and calcium intake, as well as body posture.

MATERIALS AND METHODS

Study Population

Healthy, normotensive individuals (n=39) recruited from a general population participated in a randomized acute dietary modulation research protocol (described below) identical to the conditions of our previously reported study protocol in hypertensives [16]. The original study was designed to evaluate the effect of acute dietary modulation on the renin-angiotensin system. Eligible subjects had a seated blood pressure of <130/80 on three consecutive measurements, no reported history of hypertension, hyperlipidemia, diabetes, cardiac disease or use of antihypertensive or cholesterol lowering medications, and no first-degree relative with hypertension, diabetes, or cardiovascular disease. A normal screening physical exam and laboratory assessment of serum creatinine, electrolytes, complete blood count, thyroid and liver function, and urinalysis were also required prior to participation.

Each subject provided informed consent prior to enrollment and approval was obtained from the Human Research Committee of Brigham and Women's Hospital.

Study Protocol

Eligible subjects participated in a 2-week crossover dietary sodium protocol. Subjects were randomized to one week of high dietary sodium (HS) intake, >200 mmol/24 h, or one week of low dietary sodium (LS) intake, <10 mmol/24 h, and then crossed over to the other diet for one week. Study diets also included fixed amounts of potassium (40 mmol per day) and calcium (1000 mg per day). Diets were prepared by the metabolic kitchen of the Brigham and Women's Hospital Clinical Research Center (CRC). External sodium balance and compliance on each diet were confirmed with a 24-h urine collection for sodium at the end of each study week. HS balance required a 24-h urinary sodium excretion >180 mmol/24 h and LS balance <30 mmol/24 h.

At the end of each diet week, subjects were admitted to the CRC at Brigham and Women's Hospital for 1 night and 1 day and underwent blood pressure measurement, ECG assessment, and biochemical analyses. Supine blood pressure was obtained in the morning following 10 hours of overnight supine rest using the average of five readings from an indirect recording sphygmomanometer (Dynamap automated device, Critikon, Tampa, Florida). 12-lead ECGs were obtained in the supine position at the end of each dietary period, upon admission to the CRC. Although each subject also had a screening ECG assessment upon enrollment into the study, screening ECG's were not considered in this analysis to avoid variability of salt intake on uncontrolled diets [18].

ECG Analysis

ECG voltage was measured in each lead to the nearest 0.05 mV. Per normal convention, precordial leads were defined as leads V1-V6, and limb leads were defined as leads I, II, III, aVL, aVR, and aVF. Comparison of LVH voltage criteria on ECG were made using the following standard criteria: (i) $SV1 + RV5$ (Sokolow-Lyon [V5]) or $RV6$ (Sokolow-Lyon [V6]), (ii) $SV3 + RaVL$ (Cornell voltage), or (iii) $RaVL$ (AVL). These LVH-specific voltage criteria were chosen based on their established use in the literature [6,8,9,19,20] and because their inclusion of precordial and limb leads minimized the role of lead placement variability in ECG interpretation. We also measured a "time voltage" criteria, the Cornell-product ($SV3 + RaVL + 0.8$ mV in women) $\times$ QRS duration, as another objective assessment for electrocardiographic LVH [21]. ECGs were independently assessed by two individuals, one of whom was blinded to diet status. The correlation of ECG voltage interpretation between the two reviewers was very high; the overall Pearson correlation for all ECG leads between the two reviewers was $r=0.99$, and the correlation for each LVH voltage criterion on ECG was $r=0.63$ for Cornell, $r=0.98$ for Sokolow-Lyon-V5, $r=0.99$ for Sokolow-Lyon-V6, and $r=0.98$ for AVL.

Laboratory analyses

Blood samples were collected on ice and centrifuged for 20 min. Samples were stored at -20°C without preservatives until assayed. Plasma renin activity (Diasorin Inc, Stillwater, MN), serum aldosterone concentration (Siemens, Los Angeles, CA), sodium, potassium and creatinine were measured on each diet, on the morning of admission to the CRC, following overnight supine rest as previously described [16].

Sample Size Analysis

We estimated the approximate sample size required to observe a statistically significant disparity in ECG voltage using a paired comparison of means design. Our prior data in

hypertensive individuals showed that acute dietary sodium modulation most prominently affected AVL [16]: a mean voltage difference in AVL of 0.12 mV was seen with a standard deviation of the difference in the response of matched pairs of 0.21. Thus, a minimal sample size of 34 paired ECG's (HS and LS) would be required to see this difference with a power of 0.9 and a type I error of 0.05. Since hypertensives displayed less prominent ECG voltage changes using other LVH-voltage criteria [16], and normotensives may be expected to display less prominent voltage responses with dietary sodium intake, we acknowledged that this approximation could underestimate the true sample size needed to reject the null hypothesis: acute dietary sodium modulation does affect LVH voltage criteria on ECG among normotensives.

Statistical analysis

Primary analyses were conducted to evaluate the difference in total ECG voltage, and LVH specific voltage criteria on ECG, for subjects undergoing controlled dietary sodium manipulation. Data are presented as means $\pm$ standard error of means for normally distributed variables. Non-normally distributed variables (plasma renin activity and serum aldosterone) are presented as medians with interquartile ranges. Paired t-tests were used to compare mean values within subjects (HS vs. LS), student's t-tests were used to compare means between independent populations, and Wilcoxon signed ranks test was used to compare non-normally distributed variables. The level for significance for all statistical tests was set at $\alpha=0.05$ and reported as two-tailed p-values. Data analyses were performed using SPSS (Statistical Package for Social Sciences) version 15 software (Chicago, IL).

RESULTS

Study population

The normotensive study population was young and lean, and was mostly white and male, as summarized in Table 1. As anticipated, mean blood pressure was significantly lower on LS balance, while plasma renin activity and aldosterone were both significantly higher on LS balance, when compared to HS (Table 1).

General ECG Voltage Features

The general voltage characteristics of ECG's were evaluated. When comparing paired ECG's of subjects maintained in HS and LS balance, there was no appreciable difference in total precordial or limb lead R-wave or S-wave voltages (Table 2).

LVH Voltage Criteria on ECG

Voltage from ECG's on HS and LS diet were analyzed for LVH specific voltage criteria and compared in a paired manner; we observed no significant differences in established ECG voltage criteria for LVH with acute dietary sodium modulation (Table 3). Additionally, dietary sodium modulation did not influence the time-voltage Cornell product (HS: 1182.3 ± 83.1 vs. LS: 1223.7 ± 93.1 mV ms, $P=0.55$). As expected for a normotensive population, the overall prevalence of absolute LVH on ECG was very low. In HS balance only two individuals demonstrated LVH by AVL criteria, whereas in LS balance three individuals exhibited LVH on ECG by Sokolow-Lyon-V5 criteria; however, there was no overlap between these subjects. We further examined whether there were differences between subjects who displayed higher LVH voltage criteria on HS diet, versus those who had higher LVH voltage criteria on LS diet; we observed no significant disparity in demographic features, blood pressure, or renin-angiotensin-aldosterone system activity between these groups (Table 4).

DISCUSSION

High dietary sodium intake and the detection of LVH on ECG are both predictors of cardiovascular risk [1–4,10–15]; the regression of electrocardiographic LVH and dietary sodium restriction are both associated with improved cardiovascular risk profiles [6,7,11,12]. We previously observed that the *acute* modulation of dietary sodium and renin-angiotensin system activity resulted in marked changes in ECG voltage and the prevalence of LVH on ECG in individuals with hypertension [16]; following one week of dietary sodium restriction, 60% of hypertensives who had detectable LVH on ECG no longer exhibited electrocardiographic LVH. In this study, we employed the same study protocol to retrospectively evaluate whether acute dietary sodium modulation influenced ECG voltage and LVH specific voltage criteria in a population of normotensives. In contrast to our prior observations in hypertension, dietary sodium had no detectable effect on ECG voltage in normotensives. This observation suggests that normotensives and hypertensives differ in their electrophysiological response to acute dietary sodium changes; normotensive individuals may have physiologic adaptive factors that regulate the interplay between dietary sodium intake and myocardial responsiveness that are diminished or lacking in hypertensives.

Our current findings extend those of other studies and shed new insights on the interaction between dietary salt intake and ECG voltage. Despite its insensitivity in detecting LVH, ECG remains the preferred screening methodology for LVH in the clinical setting and is known to confer an independent risk for cardiovascular outcomes and mortality. Prior studies have shown echocardiographic evidence of left ventricular mass reduction [14,15,22,23], and electrocardiographic evidence of LVH regression [6,7], following prolonged sodium restriction. However, the demonstration that *acute* dietary sodium restriction can significantly alter ECG voltage and detection of LVH is a particularly relevant observation; acute volumetric or structural changes in myocardial physiology are not typically considered when obtaining ECG's, and variations in interpreting LVH from ECG data can impair the accurate screening and risk stratification of cardiovascular risk. Although many factors can contribute to the detection of LVH on ECG, prior studies have carefully shown that the main determinant of LVH on an ECG is the product of the myocardial wall thickness and ventricular chamber diameter [17]. Thus, our prior findings in hypertensives demonstrating alterations in ECG voltage and LVH, likely reflected the modulation of ventricular chamber diameter (with acute dietary salt modulation) in the setting of increased myocardial wall thickness due to chronic hypertension. In contrast, the current study evaluated phenotypically normotensive individuals who were young and lean with no personal or family history of cardiac disease; myocardial wall thickness in this population was presumably normal, and thus, the modulation of dietary salt and intravascular volume did not result in perceivable changes in ECG voltage or LVH specific voltage criteria.

In contrast to prior observations in hypertension, where overall ECG voltage uniformly increased with high dietary salt intake and uniformly declined with salt restriction [16], this pattern was not observed in normotensives. Nearly equal numbers of the study population exhibited ECG voltage regression or voltage increments with one week of dietary salt restriction, suggesting that dietary sodium balance and intravascular volume status (reflected in the levels of renin-angiotensin aldosterone system activity) are not significant determinants of myocardial physiology that would result in LVH on ECG in normotensive individuals. In our effort to restrict inclusion criteria to specifically select those with a normal phenotype, it is worth noting that the demographics of our current normotensive population differed from our prior study in hypertensives in that they were more male-predominant, younger, and leaner. We cannot exclude the possibility that these demographic

differences may have contributed to the differential ECG responses to acute dietary modulations.

Our study findings must be interpreted in the context of the study design. Notable strengths of this study include the randomized crossover study design facilitating paired statistical analyses, the meticulous phenotyping of healthy normotensives, the absence of confounding by medications and medical conditions, and control for body posture and time of day when conducting study measurements. In addition, all subjects underwent a strict dietary protocol to mimic the typical Western diet on HS diet conditions [18,24], and a sodium restricted diet on LS diet conditions to elaborate the maximal effect size of resultant observations. A notable limitation of our study was the small sample size; however, we were likely adequately powered to see a dietary sodium induced change in AVL, which we did not. In contrast to the expected lowering of LVH voltage criterion for ECG with a sodium restricted diet [16], we observed no difference with sodium modulation, suggesting that a larger sample size would be unlikely to show a difference. The lack of corroborative data from more sensitive measures of cardiac structure such as echocardiography and magnetic resonance imaging, and the absence of long-term follow-up are also potential limitations of our study. Our observations highlight an interesting physiologic disparity in the response to acute dietary sodium modulation between normotensives and hypertensives; the availability of cardiac imaging to characterize these changes could add significant value to our interpretations and should be considered for future studies. Although all ECG's were read by two independent observers with high concordance, small heterogeneities in ECG interpretation likely introduced variation in our statistical analyses.

In summary, both dietary sodium intake and LVH on ECG are known to be important determinants of cardiovascular disease. While acute fluctuations in dietary sodium intake have been shown to significantly influence ECG voltage amplitude and detection of LVH in hypertensive individuals [16], this was not observed in healthy normotensives. Our findings may identify unique myocardial electrophysiologic differences between normotensive and hypertensive hearts in response to acute dietary sodium modulation that may be better characterized with cardiac imaging modalities in future studies.

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Table 1
Study Population Characteristics

Values reported as percentages, means with standard error of means for normally distributed variables, and medians with interquartile ranges for non-normally distributed variables.

Total Patients	N= 39		
Gender (% male)	67		
Race (% white)	69		
Age (years)	33.2 ± 12.9		
Body Mass Index (kg/m ²)	24.6 ± 3.2		
	HS	LS	P-value
Systolic Blood Pressure (mmHg)	114.5±1.66	110.2 ±1.39	<0.01
Diastolic Blood Pressure (mmHg)	70.0 ±1.66	66.8 ±1.05	<0.05
Serum Aldosterone (ng/dl)	2.7 (2.5, 4.6)	19.0 (14.2, 28.5)	<0.0001
Plasma renin activity (ng/ml/h)	0.30 (0.10, 0.43)	2.7 (1.7, 3.9)	<0.0001
Urine sodium (mmol/24 h)	248.3±10.4	13.2 ±0.78	<0.0001

Table 2

Comparison of the mean sum voltage in precordial leads (V_1 - V_6) and limb leads (I, II, III, aVL, AVR) in the normotensive study population.

	HS	LS	P-Value
Precordial R (mV)	5.2 ± 0.30	5.4 ± 0.30	0.18
Precordial S (mV)	3.3 ± 0.20	3.5 ± 0.30	0.07
Limb R (mV)	3.5 ± 0.20	2.7 ± 0.30	0.09
Limb S (mV)	1.0 ± 0.01	1.0 ± 0.30	0.39

Table 3

Comparison of LVH specific voltage criteria between HS and LS diets.

	HS	LS	P-value
Cornell (mV)	1.0 ± 0.10	1.1 ± 0.8	0.77
Sokolow-Lyon-V5 (mV)	2.1 ± 0.10	2.2 ± 0.10	0.27
Sokolow-Lyon-V6 (mV)	1.9 ± 0.10	2.0 ± 0.10	0.15
AVL (mV)	0.3 ± 0.5	0.3± 0.01	0.98

Table 4
Comparison of individuals who demonstrated higher LVH-specific voltages on HS versus those who demonstrate higher LVH-specific voltages on LS diet

Values reported as percentages or means with standard error of means; $P=NS$ for all comparisons for each LVH criteria.

	Cornell		Sokolow-Lyon-V5		Sokolow-Lyon-V6		AVL	
	HS < LS	HS > LS	HS < LS	HS > LS	HS < LS	HS > LS	HS < LS	HS > LS
N								
	22	17	22	17	20	19	17	22
Gender (%male)	72	58	77	53	75	58	71	63
Age (years)	34 ± 2.9	32 ± 3.0	34 ± 2.7	33 ± 3.3	31 ± 2.7	35 ± 3.1	34 ± 3.5	33 ± 2.5
Race (%white)	59	76	68	64	80	52	82	55
BMI (kg/m²)	25 ± 0.7	23 ± 0.8	25 ± 0.7	24 ± 0.7	24 ± 0.8	24 ± 0.7	25 ± 0.8	23 ± 0.7
Δ Systolic Blood Pressure (mmHg)	6.2 ± 7.6	1.7 ± 2.0	5.0 ± 5.7	3.4 ± 5.7	2.2 ± 6.0	6.3 ± 6.0	4.1 ± 7.0	4.5 ± 5.3
Δ Serum Aldosterone (ng/dL)	16.1 ± 1.9	16.2 ± 3.0	15.18 ± 2.4	14.4 ± 2.2	16.9 ± 2.7	12.2 ± 1.6	17.2 ± 2.3	13.1 ± 2.3
Δ Plasma renin activity (ng/mL/h)	2.1 ± 0.3	2.2 ± 0.4	1.80 ± 0.3	2.3 ± 0.4	2.1 ± 0.4	2.0 ± 0.3	2.4 ± 0.4	1.7 ± 0.3