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Obesity and hypertension contribute to prolong QRS complex duration among middle-aged adults

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Abstract

Background: Obesity and hypertension are comorbid diseases, which influence cardiac structure, and are associated with increased risk for cardiovascular events. The QRS duration (QRSd) reflects ventricular depolarization, and increased QRSd is associated with poor cardiovascular outcomes. QRS duration may be influenced by obesity and HTN, and reflect the increased risk factor for poor cardiovascular outcomes. The purpose of this analysis was to assess the relations between obesity, hypertension, and the interaction between them on QRSd.

Methods: In this secondary data analysis, firefighters without documented cardiovascular disease in normal sinus rhythm were included. Twelve-lead 24-hr ECG Holter monitors measured mean QRSd. Body mass index (BMI) and resting blood pressure (BP) were measured and categorized. Univariate linear regression models were produced using BMI, BP, and the interaction between BMI and BP as factors associated with QRSd. Multivariate models adjusting for multiple covariates were also produced.

Results: Seventy-seven firefighters were included and most (89.4%) were overweight or obese. After covariate adjustment, BMI ($p = 0.028$), BMI categorization ($p = 0.020$), and the interaction between BMI and systolic BP ($p = 0.021$) were associated with prolong QRSd.

Conclusions: Increased BMI and an interaction between BMI and systolic BP were independently associated with prolongation of the QRS complex. Determination of the underlying cardiac structures responsible prolongation of the QRSd is recommended for further research.

KEYWORDS
electrocardiography, hypertension, obesity, risk factors

1 | INTRODUCTION

Obesity and hypertension are significant complications among adults in the United States and associated with poor cardiovascular outcomes. The prevalence of obesity among adults in the United States increased from 33.7% in 2007–2008 to 39.6% in 2015–2016, and the prevalence of severe obesity has increased 5.7% in 2007–2008 to 7.7% in 2015–2016 (Hales, Fryar, Carroll, Freedman, & Ogden, 2018). Obesity is highly comorbid with hypertension (Lavie, Milani, & Ventura, 2009). A 2017 epidemiological study of 324,199 patients found that the prevalence of antihypertensive medication increased with BMI and that more than 50% of individuals with a BMI > 35 kg/m^2 were prescribed one (Pantalone et al., 2017). According to the Guyton hypothesis, hypertension develops when the relationship between arterial pressure and natriuresis is asynchronous; in obesity, this occurs due to increased sympathetic activity, increased cardiac output, and activation of the renin–angiotensin system altering natriuresis consequently rising arterial pressure (Re, 2009). Prolong, sustained hypertension manifests as structural heart disease including left ventricular hypertrophy (LVH) which increases the susceptibility to lethal ventricular arrhythmia and sudden cardiac death (SCD) even among young obese individuals (Jayaraman et al., 2018; Kahan & Bergfeldt, 2005; Re, 2009). Thus, it is important to understand the influences of obesity and hypertension on cardiac health among asymptomatic middle-aged adults for proper risk stratification. The interaction between obesity and hypertension contributes to the development of structural changes in the heart and leads to poor cardiovascular outcomes.

The electrocardiogram (ECG) is a relatively inexpensive, noninvasive, and widely accessible instrument to characterize cardiac risk. The effects of obesity and hypertension on ECG waveforms have been investigated in previous studies. Obesity is associated with a wide variety of ECG abnormalities including prolong JT and QT interval, leftward shifts in the P wave, LVH pattern, flattening of the T wave, and prolongation of the QRS complex duration (QRSd) (Alpert et al., 2000; Fraley, Birchem, Senkottaiyan, & Alpert, 2005; Inair et al., 2019; Sun et al., 2013). Similarly, the effects of hypertension on ECG-based measures have been previously investigated, and such effected measures include the Tp-Te interval as well as fragmentation and prolongation of the QRS complex (Ferrucci et al., 2015; Kadi et al., 2013). Since prolong QRSd has been associated with both hypertension and obesity independently, this ECG measure was chosen specifically for the described study. Moreover, increased QRSd has been suggested to be an independent marker for poor cardiac outcomes including SCD (Kurl, Mäkikallio, Rautaharju, Kiviniemi, & Laukkanen, 2012; Morin et al., 2009; Teodorescu et al., 2011). Among research examining the role of both obesity and hypertension, one previous study evaluated the effect of both conditions on P-wave indices (Vaidean, Manczuk, & Magnani, 2016). The researchers demonstrated that both conditions were associated with increased in P-wave indices which potentially increased the risk for atrial fibrillation (Vaidean et al., 2016). Obesity and hypertension independently impact numerous ECG waveforms such as QRSd; yet, the impact of the interaction between the two conditions on QRSd remains unquantified.

Obesity and hypertension impact more than a third of adults living in the United States and significantly increase the risk for poor cardiovascular outcomes. This secondary analysis sought to examine the relations between obesity, hypertension, and the interaction term between the two conditions on ventricular depolarization. The objective of this analysis was to evaluate the association between obesity, hypertension, and the interaction between obesity and hypertension on QRSd, specifically, among a sample of asymptomatic middle-aged adults.

Highlights

1. Obesity and hypertension are concomitant conditions plaguing more than a third of the population in the United States increasing risk for poor cardiovascular outcomes though most individuals are asymptomatic.
2. The electrocardiogram (ECG) remains the most widely used tool for cardiac risk stratification; thus, determining the influence of obesity, hypertension, and the interaction between the two on ECG waveforms may be informative for risk characterization among asymptomatic adults.
3. QRS duration (QRSd) corresponds to ventricular depolarization, and prolong QRSd may reflect myocardial thickening delaying electrical transduction.
4. In this analysis, obesity and the interaction between obesity and systolic blood pressure were statistically significant variables which prolong QRSd among asymptomatic middle-aged adults.

2 | METHODS

Subjects were firefighters participating in a cross-sectional, NIH-funded study conducted in Western New York. All on-duty firefighters were eligible to participate in the study. The primary results from the nurse-led study have been previously reported (Al-Zaiti & Carey, 2015). The study was approved by the Institutional Review Board at the State University of New York at Buffalo. On-duty firefighters provided written consent after understanding the potential benefits and risks involved with this study in the firehouse. Self-reported demographic data were collected which included age, sex, number of years as a firefighter, race (white, black, or other), whether a current smoker (yes/no), and whether the firefighter had a current medical diagnosis of sleep apnea (yes/no). Afterward, registered nurses obtained anthropometric measures including height and weight for body mass index (BMI) calculation. Height was recorded using a tape measure, and weight was recorded once using a calibrated bathroom scale. Body mass index was categorized in accordance with the Centers for Disease Control and Prevention (CDC) standard BMI range categories for adults (Calle, Thun, Petrelli, Rodriguez, & Heath, 1999).

2.1 | Blood pressure

Resting state blood pressure (BP) was recorded in the quiet bunkroom with the firefighters sitting and resting for 5 min. A second reading was taken 5 min later with the firefighter still resting in the sitting position. The mean of the two BP values was considered the resting state BP which was then categorized. Blood pressure categorization was then conducted in accordance with the 2017 American Heart Association BP guidelines (Whelton et al., 2018).

2.2 | 12-lead 24-hr Holter ECG monitoring

This study used 12-lead, H12 + Holter ECG monitors (V3.12; Mortara Instruments) which captured continuous ECG data for 24 hr. The leads were placed on shaven and cleaned areas of the torso, in the Mason–Likar lead configuration, under the firefighters' uniforms, and monitors were securely fastened to their belt (Papouchado, Walker, James, & Clarke, 1987). Firefighters were instructed to continue their activities of daily living with the 12-lead Holter monitor attached for 24 hr. After, the firefighters were instructed to remove the electrodes and return the Holter monitor to the firehouse for their next shift. To ensure quality of the QRSd measurement, high-fidelity and high-resolution (1,000 samples per second) recordings were obtained with a frequency of 0.05–60 Hz. Recordings were downloaded to a computer with H-Scribe 4 software (Mortara Instruments) for ECG processing and analysis. After semi-automatic annotation was performed to filter noticeable artifact, all ECGs were manually reviewed for quality assurance by a reviewer blinded to the study. High-frequency ECG waveforms like QRSd, especially during periods of tachycardia, can be missed by automatic computer software using the standard upper filter setting of 60 Hz. Thus, to ensure accuracy of the measurement, the first 10-s ECG tracing of the 24-hr monitoring period was exported into a portable document format with the standard filter setting at 0.05–150 Hz using the ELI LINK program (Mortara Instruments). The average QRSd from each of the 12 leads during the entire 24-hr monitoring session was calculated and reported. Lastly, since LVH may be a result of prolong and undertreated hypertension, LVH was measured and reported using Cornell's criteria (lead SV3 + lead RaVL; cutoff for LVH > 20 mm in women or > 28 mm in men). Su et al. (2017) recently reported that Cornell's criteria had a sensitivity of 22.2% and specificity of 95.2% for LVH.

Inclusion criteria for this study required on-duty firefighters to have a normal sinus rhythm for an entire 24-hr recording session. This meant that firefighters with any type of arrhythmia as well as either a complete or incomplete right bundle branch block (RBBB) or left bundle branch block (LBBB) were excluded. Firefighters were excluded if they were receiving pharmacotherapy for either cardiovascular or respiratory disease, underwent cardiovascular surgery including cardiac catheterization, or had a pacemaker. Previous studies suggest that common antihypertensives such as beta-blockers may increase QRSd; thus, firefighters receiving therapy were excluded (Madias, 2008).

2.3 | Statistical analysis

All analyses were conducted using Statistical Package for Social Sciences (SPSS version 21.0; IBM Corp.). Statistical significance was considered when $p < 0.05$. Categorical variables are provided as % (n) and continuous variables as mean ± standard deviation (SD). One-way analysis of variance (ANOVA) was performed to analyze the differences in QRSd based on obesity and BP classification. Continuous variables were assessed for normality using the Shapiro–Wilk test, and the presence of outliers (>3SD) was evaluated which there were none. Body mass index was deemed skewed (Shapiro–Wilk statistic 0.931; $p < 0.05$), and the normal distribution curve was skewed in a positive direction. A $\log_{10}$ transformation was performed and corrected the normality issue (Shapiro–Wilk statistic 0.974; $p = 0.109$). LogBMI analyses were performed for all analyses as described below, and the results are provided. For the ease of interpretation of logarithms, results were also reported as percent changes. There were no missing data in this secondary analysis.

Linear regression models were used to assess the association between BMI and BP as continuous variables at the univariate level. Similarly, linear regression was used to assess the association between BMI and BP classification as categorical variables at the univariate level as well. This was conducted to determine whether current BMI and BP guidelines were reflected of cardiac structural changes as measured by QRSd. Multivariate linear regression models were used to assess the association between BMI and BP on QRSd after adjustment for covariates. Covariates were determined from the literature and included: age, race/ethnicity, length at occupation as a proxy of occupational exposure, history of sleep apnea, and mean 24-hr heart rate. Multivariate linear regression models using the enter selection method were performing using BMI and BP as continuous variables and as categorical variables.

Lastly, interaction terms were computed to estimate the effect of BMI and systolic BP and the effect of BMI and diastolic BP on QRSd. Interaction terms only involved continuous variables to better account for the variance. Both non-mean-centered and mean-centered values were reported. Interaction terms were evaluated on both the univariate and multivariate levels adjusting for the same potential covariates mentioned above.

3 | RESULTS

This secondary analysis included 77 on-duty firefighters with a mean age of 43.42 (±7.75 years) and a mean career length of 15.42 years (±7.00 years). The majority of this sample was male (96.1%, n = 74) and white (79.2%, n = 61), as seen in Table 1. The mean BMI was 29.39 kg/m^2 (±4.44 kg/m^2), mean systolic BP was 129.12 mmHg (±13.04 mmHg), and mean baseline diastolic BP was 81.53 mmHg (±10.69 mmHg). Thus, the average on-duty firefighter in this sample was overweight and had Stage I HTN per current guideline recommendations. The mean QRSd for the 24-hr ambulatory Holter monitoring period was 95.18 milliseconds (±7.41). Based on the two one-way ANOVAs performed, there were no statistically significant differences in QRSd based on obesity ($p = 0.123$) or hypertension classification ($p = 0.126$). The results of the ANOVAs as well as the group means can be seen in Table 2. Overall, this was a predominantly white male sample of professional on-duty firefighters, whom would be categorized, on average, as hypertensive and overweight with a normal QRSd.

Linear regression models were produced to evaluate the association between BMI as a measure of obesity and QRSd. On the univariate level, logBMI was not statistically significantly associated with QRSd, but, after controlling for known variables which influence QRSd, logBMI was statistically significantly related to QRSd ($B = 31.100$; $p = 0.034$; 95% CI 33.358–59.843; $R^2 = 0.120$). Table 3

TABLE 1 Firefighter demographics, $n = 77$

Continuous variables	
Characteristic (n = 77)	**Mean (±*SD*)**
Age (years)	43.42 (±7.75)
Career length (years)	15.42 (±7.00)
BMI (kg/m^2)	29.39 (±4.44)
Systolic BP (mmHg)	129.12 (±13.04)
Diastolic BP	81.53 (±10.69)
QRSd (ms)	95.18 (±7.41)
24-hr heart rate (bpm)	77.13 (±9.56)
Categorical variables	
Characteristic (n = 77)	**% (n)**
Sex	
Male	96.1 (74)
Female	3.9 (3)
BMI	
Normal (BMI 18.5–24.9)	11.7 (9)
Overweight (BMI 25–29.9)	48.1(37)
Obese class I (BMI 30–34.9)	32.5 (25)
Obese class II (BMI 35–39.9)	3.9 (3)
Obese class III (BMI > 40)	3.9 (3)
Hypertension	
Normal (<120/<80 mmHg)	23.4 (18)
Elevated (120–129/<80 mmHg)	6.5 (5)
Stage I (130–139 or 80–89 mmHg)	37.7 (29)
Stage II (>140/>90 mmHg)	32.5 (25)
Sleep apnea	3.9 (3)
LVH	2.6 (2)
Currently smoking	13 (10)

Abbreviations: BMI, body mass index; LVH, left ventricular hypertrophy. Note Categories in *italics* have multiple levels.

shows the values generated from the univariate linear regression models, while Table 4 shows the results from the adjusted models. These models were repeated for obesity categories per CDC recommendations and results were reproducible (B = 2.338; p = 0.024; 95% CI 0.318–4.358; R^2 = 0.128). That is, a one-category increase was associated with a 2.403 ms increase in QRSd. This multivariate model using BMI categorization accounted for 12.8% of the variance in the QRSd variable, a small measure of association. From these models, it appears BMI was positively associated with QRSd after adjustment for potential covariates.

Next, linear regression models were generated to evaluate the association between BP and QRSd. On the univariate level, neither systolic nor diastolic BP was statistically significantly associated with QRSd. However, on the multivariate level, only systolic BP (B = 0.117; p = 0.087; 95% CI −0.018 to 0.252; R^2 = 0.100) was nearing statistical significance in relation to QRSd. On the univariate and multivariate level, BP classification was not statistically significant though the multivariate model was nearing significance (B = 1.069; p = 0.164; 95% −0.447 to 2.584; R^2 = 0.087). In summary, multivariate adjusted models using systolic BP and BP classification to explain QRSd partially explained the increase in QRSd.

Lastly, the interaction between logBMI and each systolic and diastolic BP measure was evaluated on QRSd. A statistically significant univariate relationships existed between the interaction term of systolic BP and logBMI (B = 0.090; p = 0.020; 95% CI 0.014–0.165; R^2 = 0.070). After mean centering of both variables, the interaction term was nonstatistically significant (B = 1.038; p = 0.353; 95% CI −1.176 to 3.253; R^2 = 0.011). However, interaction term between diastolic BP and logBMI was not statistically significant (B = 0.077; p = 0.129; 95% CI −0.023 to 0.129; R^2 = 0.030), and this remained nonsignificant after mean centering of both variables (B = −0.924; p = 0.501; 95% CI −3.647 to 1.798; R^2 = 0.006). On the multivariate level, the interaction term between systolic BP and logBMI remained statistically significant (B = 0.093; p = 0.022; 95% CI 0.014–0.172; R^2 = 0.129), and the interaction term between diastolic BP and

TABLE 2 Differences in QRSd means between BMI and BP groups, n = 77

Category	Mean (±*SD*)	95% CI for mean	*p*-Value for ANOVA
BMI classification			
Normal (BMI 18.5–24.9)	89.11 (±4.910)	85.34–92.89	0.123
Overweight (BMI 25–29.9)	96.14 (±6.780)	93.87–98.40	
Obese I (BMI 30–34.9)	95.44 (±8.476)	91.94–98.94	
Obese II (BMI 35–39.9)	97.67 (±5.508)	83.99–111.35	
Obese III (BMI > 40)	97.00 (±8.544)	75.78–118.22	
Hypertension classification			
Normal	91.89 (±8.210)	87.81–95.97	0.120
Elevated	99.60 (±5.225)	93.11–106.09	
Stage I	95.83 (±7.051)	93.15–98.85	
Stage II	95.92 (±7.100)	92.99–98.85	

Note: Categories in *italics* have multiple levels.

TABLE 3 Univariate models of logBMI, BP, and logBMIxBP on QRSd, $n = 77$

Predictor	Variable type	Unstandardized *B*	Standardized *B*	*p*-value	95% CI	R^2	*F* statistic
Obesity	logBMI (continuous)	21.336	0.180	0.117	−5.482 to 48.154	0.032	2.512
	BMI classification (categorical)	1.526	0.184	0.110	−0.352 to 3.404	0.034	2.620
Hypertension	SYS (continuous)	0.125	0.221	0.053	−0.004 to 0.250	0.049	3.853
	DYS (continuous)	0.090	0.131	0.256	−0.067 to 0.248	0.017	1.310
	HTN classification (categorical)	1.165	0.179	0.119	−0.308 to 2.637	0.032	2.483
Obesity * Hypertension	logBMI * SYS (non-mean-centered; continuous)	0.090	0.264	0.020[a]	0.014 to 0.165	0.070	5.639
	logBMI * SYS (centered; continuous)	1.038	0.107	0.353	−1.176 to 3.253	0.011	0.872
	logBMI * DYS (non-mean-centered; continuous)	0.077	0.174	0.129	−0.023 to 0.177	0.030	2.354
	logBMI * DYS (centered; continuous)	−0.924	−0.078	0.501	−3.647 to 1.798	0.006	0.457

Abbreviations: BMI, body mass index; DYS, diastolic BP; HTN, hypertension; SYS, systolic BP.
*Statistical significance at $p < 0.05$.

logBMI remained nonstatistically significant ($B = 0.077$; $p = 0.146$; 95% CI −0.027 to 0.181; $R^2 = 0.089$). Comparatively, mean-centered interaction terms were nonstatistically significant for both systolic ($B = 0.601$; $p = 0.610$; 95% CI −1.738 to 2.941; $R^2 = 0.065$) and diastolic BP ($B = 0.976$; $p = 0.487$; 95% CI −3.758 to 1.806; $R^2 = 0.068$). Through the multivariate model including the interaction term between systolic BP and logBMI, 13% of the variance could be explained. The interaction between logBMI and systolic BP is associated with QRSd prolongation.

4 | DISCUSSION

In this secondary analysis, the effects of obesity, hypertension, and the interaction between the two conditions were assessed on QRSd among a sample of asymptomatic adults free of documented cardiovascular disease and arrhythmia. The results of this study suggest that obesity as measured by increased BMI, as both a continuous and categorical variable, and the interaction between increasing BMI and systolic BP contribute to prolongation of the QRS complex. Alarmingly, even among individuals without known cardiovascular disease, changes in cardiac ECG waveforms suggestive of underlying cardiac pathology may be evident by prolongation of the QRSd.

This study adds to the growing understanding of the changes on ECG associated with obesity and hypertension due to underlying changes in cardiac conduction. In obesity, abnormal increases in sympathetic activity and activation of the renin–angiotensin system lead to increased cardiac output and sodium retention (Re, 2009). The effects of obesity and hypertension on ECG waveforms and intervals have been previously reported as depolarization and repolarization abnormalities among others (Alpert et al., 2000; Ferrucci et al., 2015; Fraley et al., 2005; Inair et al., 2019; Kadi et al., 2013; Sun et al., 2013). Vaidean, Manczuk, and Magnani (2016) recently reported that P-wave indices increase among adults with general and central obesity, and that hypertension exerted an incremental effect in people who were overweight but not obese possibly increases the risk for atrial fibrillation. In the presented study, the interaction term between obesity and hypertension was associated with small changes in QRSd, potentially a sign of changes in the underlying structures responsible for ventricular depolarization. In summary, the presented findings add to the growing body of evidence that obesity, hypertension, and possibly the interaction between the two conditions contribute to measurable ECG changes.

With a growing obesity and hypertension epidemic, proper stratification of cardiovascular risk will be important for practicing clinicians. Current recommendations by the American Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure and the 2013 joint European Society of Hypertension/European Society of Cardiology advise that clinicians routinely perform 12-lead ECG among all patients with arterial hypertension for risk stratification purposes though such guidelines have not been published for management of obese patients (Chobanian et al., 2003; Mancia et al., 2013). Thus, it is important to understand the underlying reasons for changes in ECG. Prolongation of the QRSd due to obesity and the interaction between obesity and systolic hypertension as suggested in this study may reflect the developmental of structural changes within the myocardium which may predispose individuals to left ventricular dysfunction and cardiac arrhythmia leading to SCD (Lavie et al., 2009; Re, 2009). Murkofsky et al. (1998) found that a prolong QRSd was a specific indicator of left systolic dysfunction which may preclude poor cardiac output and SCD. Prolong QRSd has previously been reported as an independent predictor for SCD among men, obese and hypertensive individuals, and the general public (Kurl et al., 2012; Morin et al., 2009; Teodorescu et al., 2011). The stress of volume overload on the ventricular wall induced by obesity and hypertension contributes to the development of LVH and ventricular enlargement which may predispose individuals to SCD,

TABLE 4 Multivariate models of logBMI, BP, and logBMIxBP on QRSd, *n* = 77

Predictor	Variable type	Unstandardized B	Standardized B	*p*-value	95% CI	Adjusted model R^2	Adjusted model *F* statistic	Adjusted model *p*-value
Obesity	logBMI (continuous)	31.100	0.262	0.034[a]	3.358 to 59.843	0.120	1.588	0.163
	BMI classification (categorical)	2.338	0.281	0.024[a]	0.318 to 4.358	0.128	1.707	0.132
Hypertension	SYS (continuous)	0.117	0.208	0.087	−0.018 to 0.252	0.100	1.295	0.271
	DYS (continuous)	0.074	0.108	0.362	−0.087 to 0.236	0.072	0.911	0.492
	HTN classification (categorical)	1.069	0.164	0.164	−0.447 to 2.584	0.087	1.113	0.364
Obesity * Hypertension	logBMI * SYS (non-mean-centered; continuous)	0.093	0.274	0.022[a]	0.014 to 0.172	0.129	1.730	0.127
	logBMI * SYS (centered; continuous)	0.601	0.062	0.610	−1.738 to 2.941	0.065	0.808	0.567
	logBMI * DYS (non-mean-centered; continuous)	0.077	0.174	0.146	−0.027 to 0.181	0.089	1.145	0.346
	logBMI * DYS (centered; continuous)	−0.976	−0.082	0.487	−3.758 to 1.806	0.068	0.848	0.537

Abbreviations: BMI, body mass index; DYS, diastolic BP; HTN, hypertension adjusted for: age, race, career length, sleep apnea, and 24-hr mean heart rate; all nonsignificant ($p > 0.05$); SYS, systolic BP.
*Statistical significance at $p < 0.05$.

even among young adults (Jayaraman et al., 2018; Lavie et al., 2009; Re, 2009). Past research has shown that mass in combination with anatomic and electric remodeling of the left ventricular is responsible for QRS complex changes seen among LVH patients (Bacharova, Szathmary, Kovalcik, & Mateasik, 2010). The small effect sizes reported in this study may be due to the very early changes of LVH ventricular remodeling. The sample was composed of mostly younger adult males who were firefighters and free of documented cardiovascular disease and arrhythmia. As such, small increases in QRSd may provide a potential novel marker of early cardiac remodeling processes.

This study has a number of limitations which need to be addressed. Only individuals without reported cardiovascular disease and free of both ventricular and atrial arrhythmias were included which increases the homogeneity of the sample while potentially reflecting a sample with less cardiac burden. The sizes of association reported in this study are small, and this may potentially be due to the lack of measurable cardiac burden among these individuals. The individuals in this study were on-duty professional firefighters who are younger than most cardiac patients. However, although these individuals were firefighters, antidotal evidence suggests firefighters do not exercise more than the typical patient and engage in a traditional western lifestyle hallmarked by poor dietary choices and inactivity. Obesity is a significant and well-published problem in the firefighting community (Dzikowicz & Carey, 2018; Soteriades et al., 2011). Imaging data are needed to confirm the hypothesis that small increases in QRSd may be an early sign of the development of LVH and ventricular remodeling. Among firefighters which this study used as a sample, it has been reported that the incidence of LVH was 15 per 1,000 person-years of follow-up after approximately 3 years (Soteriades et al., 2011). Moreover, obese firefighters were 2 times more likely (OR = 2; 95% CI, 1.6–6.6) to develop LVH determined by Sokolow–Lyon criterion compared to nonobese firefighters (Soteriades et al., 2011). In the presented study, on-duty firefighters were mostly obese and hypertensive, but only 2 (2.6%) firefighters were found to have LVH per Cornell's criteria. This discrepancy may be attributed to the difference in LVH criteria and sample size (Soteriades et al., 2011). Although limitations are addressed, the findings here demonstrate the association between obesity and hypertension on QRSd contributes to the growing body of literature in this field.

Comprehensively, this study finds that among a sample of asymptomatic adults without documented cardiovascular disease, obesity and the interaction between obesity and systolic hypertension contribute to prolonging the duration of the QRS complex. Further research is necessary to allude to the potential pathological mechanisms responsible for such phenomena such as the development of LVH which increases risk for SCD.

5 | CONCLUSIONS

Obesity and hypertension are prevalent comorbid conditions among adults and known to independently impact ECG. This study finds evidence that obesity and the interaction between obesity and systolic hypertension contribute to prolongation of QRSd. This may reflect early processes of LVH and ventricular remodeling.

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CONFLICT OF INTEREST

None declared.

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REFERENCES

Alpert, M. A., Terry, B. E., Cohen, M. V., Fan, T. M., Painter, J. A., & Massey, C. V. (2000). The electrocardiogram in morbid obesity. *The American Journal of Cardiology*, *85*(7), 908–910. https://doi.org/10.1016/S0002-9149(99)00894-2

Al-Zaiti, S. S., & Carey, M. G. (2015). The prevalence of clinical and electrocardiographic risk factors of cardiovascular death among on-duty professional firefighters. *The Journal of Cardiovascular Nursing*, *30*(5), 440. https://doi.org/10.1097/JCN.0000000000000165

Bacharova, L., Szathmary, V., Kovalcik, M., & Mateasik, A. (2010). Effect of changes in left ventricular anatomy and conduction velocity on the QRS voltage and morphology in left ventricular hypertrophy: A model study. *Journal of Electrocardiology*, *43*(3), 200–208. https://doi.org/10.1016/j.jelectrocard.2009.07.014

Calle, E. E., Thun, M. J., Petrelli, J. M., Rodriguez, C., & Heath, C. W. Jr (1999). Body-mass index and mortality in a prospective cohort of US adults. *New England Journal of Medicine*, *341*(15), 1097–1105. https://doi.org/10.1056/NEJM199910073411501

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L. Jr, … Roccella, E. J. (2003). The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 report. *JAMA*, *289*(19), 2560–2571.

Dzikowicz, D. J., & Carey, M. G. (2018). Widened QRS-T angle may be a measure of poor ventricular stretch during exercise among on-duty firefighters. *The Journal of Cardiovascular Nursing*, *34*(3), 201–207. https://doi.org/10.1097/JCN.0000000000000554

Ferrucci, A., Canichella, F., Battistoni, A., Palano, F., Francia, P., Ciavarella, G. M., … Tocci, G. (2015). A novel electrocardiographic T-wave measurement (Tp-Te interval) as a predictor of heart abnormalities in hypertension: A new opportunity for first-line electrocardiographic evaluation. *The Journal of Clinical Hypertension*, *17*(6), 441–449. https://doi.org/10.1111/jch.12522

Fraley, M. A., Birchem, J. A., Senkottaiyan, N., & Alpert, M. A. (2005). Obesity and the electrocardiogram. *Obesity Reviews*, *6*(4), 275–281. https://doi.org/10.1111/j.1467-789X.2005.00199.x

Hales, C. M., Fryar, C. D., Carroll, M. D., Freedman, D. S., & Ogden, C. L. (2018). Trends in obesity and severe obesity prevalence in US youth and adults by sex and age, 2007–2008 to 2015–2016. *JAMA*, *319*(16), 1723–1725. https://doi.org/10.1001/jama.2018.3060

Inair, M., Sincer, I., Erdal, E., Gunes, Y., Cosgun, M., & Mansiroglu, A. K. (2019). Evaluation of electrocardiographic ventricular repolarization parameters in extreme obesity. *Journal of Electrocardiography*, *53*(1), 36–39. https://doi.org/10.1016/j.jelectrocard.2018.12.003

Jayaraman, R., Reinier, K., Nair, S., Aro, A. L., Uy-Evanado, A., Rusinaru, C., … Chugh, S. S. (2018). Risk factors of sudden cardiac death in the young: Multiple-year community-wide assessment. *Circulation*, *137*(15), 1561–1570. https://doi.org/10.1161/CIRCULATIONAHA.117.031262

Kadi, H., Demir, A. K., Karaman, K., Damar, İ. H., Ceyhan, K., Zorlu, Ç., & Aygüç, B. (2013). Fragmented QRS complexes on ECG in hypertensive patients are signify more severe left ventricular diastolic abnormalities. *Journal of the American College of Cardiology*, *62*(18), C178. https://doi.org/10.1016/j.jacc.2013.08.504

Kahan, T., & Bergfeldt, L. (2005). Left ventricular hypertrophy in hypertension: It's arrhythmogenic potential. *Heart*, *91*(2), 250–256. https://doi.org/10.1136/hrt.2004.042473

Kurl, S., Mäkikallio, T. H., Rautaharju, P., Kiviniemi, V., & Laukkanen, J. A. (2012). Duration of QRS complex in resting electrocardiogram is a predictor of sudden cardiac death in men. *Circulation*, *125*(21), 2588–2594. https://doi.org/10.1161/CIRCULATIONAHA.111.025577

Lavie, C. J., Milani, R. V., & Ventura, H. O. (2009). Obesity and cardiovascular disease: Risk factor, paradox, and impact of weight loss. *Journal of the American College of Cardiology*, *53*(21), 1925–1932. https://doi.org/10.1016/j.jacc.2008.12.068

Madias, J. E. (2008). Drug-induced QRS morphology and duration changes. *Cardiology Journal*, *15*(6), 505–509.

Mancia, G., Fagard, R., Narkiewicz, K., Redon, J., Zanchetti, A., Böhm, M., … Zannad, F. (2013). 2013 ESH/ESC guidelines for the management of arterial hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *Blood Pressure*, *22*(4), 193–278. https://doi.org/10.3109/08037051.2013.812549

Morin, D. P., Oikarinen, L., Viitasalo, M., Toivonen, L., Nieminen, M. S., Kjeldsen, S. E., … Okin, P. M. (2009). QRS duration predicts sudden cardiac death in hypertensive patients undergoing intensive medical therapy: The LIFE study. *European Heart Journal*, *30*(23), 2908–2914. https://doi.org/10.1093/eurheartj/ehp321

Murkofsky, R. L., Dangas, G., Diamond, J. A., Mehta, D., Schaffer, A., & Ambrose, J. A. (1998). A prolonged QRS duration on surface electrocardiogram is a specific indicator of left ventricular dysfunction. *Journal of the American College of Cardiology*, *32*(2), 476–482. https://doi.org/10.1016/S0735-1097(98)00242-3

Pantalone, K. M., Hobbs, T. M., Chagin, K. M., Kong, S. X., Wells, B. J., Kattan, M. W., … Burguera, B. (2017). Prevalence and recognition of obesity and its associated comorbidities: Cross-sectional analysis of electronic health record data from a large US integrated health system. *British Medical Journal Open*, *7*(11), e017583. https://doi.org/10.1136/bmjopen-2017-017583

Papouchado, M., Walker, P. R., James, M. A., & Clarke, L. M. (1987). Fundamental differences between the standard 12–lead electrocardiograph and the modified (Mason—Likar) exercise lead system. *European Heart Journal*, *8*(7), 725–733. https://doi.org/10.1093/eurheartj/8.7.725

Re, R. N. (2009). Obesity-related hypertension. *The Ochsner Journal*, *9*(3), 133–136.

Soteriades, E. S., Targino, M. C., Talias, M. A., Hauser, R., Kawachi, I., Christiani, D. C., & Kales, S. N. (2011). Obesity and risk of LVH and ECG abnormalities in US firefighters. *Journal of Occupational and Environmental Medicine*, *53*(8), 867–871. https://doi.org/10.1097/JOM.0b013e318221c6fe

Su, F.-Y., Li, Y.-H., Lin, Y.-P., Lee, C.-J., Wang, C.-H., Meng, F.-C., … Lin, G.-M. (2017). A comparison of Cornell and Sokolow-Lyon electrocardiographic criteria for left ventricular hypertrophy in a military male population in Taiwan: The cardiorespiratory fitness and hospitalization events in armed forces study. *Cardiovascular Diagnosis and Therapy*, *7*(3), 244. https://doi.org/10.21037/cdt.2017.01.16

Sun, G. Z., Li, Y., Zhou, X. H., Guo, X. F., Zhang, X. G., Zheng, L. Q., Li, Y., Jiao, Y. D., … Sun, Y. X. (2013). Association between obesity and ECG variables in children and adolescents: A cross-sectional study. *Experimental and Therapeutic Medicine*, *6*(6), 1455–1462. https://doi.org/10.3892/etm.2013.1337

Teodorescu, C., Reinier, K., Uy-Evanado, A., Navarro, J., Mariani, R., Gunson, K., Jui, J., … Chugh, S. S. (2011). Prolonged QRS duration on the resting ECG is associated with sudden death risk in coronary disease, independent of prolonged ventricular repolarization. *Heart Rhythm: the Official Journal of the Heart Rhythm Society*, *8*(10), 1562–1567. https://doi.org/10.1016/j.hrthm.2011.06.011

Vaidean, G. D., Manczuk, M., & Magnani, J. W. (2016). Atrial electrocardiography in obesity and hypertension: Clinical insights from the Polish-Norwegian Study (PONS). *Obesity*, *24*(12), 2608–2614. https://doi.org/10.1002/oby.21678.

Vaidean, G. D., Manczuk, M., & Magnani, J. W. (2016). Atrial electrocardiography in obesity and hypertension: Clinical insights from the Polish-Norwegian Study (PONS). *Obesity* (Silver Spring, Md.), *24*(12), 2608–2614. https:/doi.org//10.1002/oby.21678.

Whelton, P. K., Carey, R. M., Aronow, W. S., Casey, D. E., Collins, K. J., Himmelfarb, C. D., … MacLaughlin, E. J. (2018). 2017 Guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Journal of the American College of Cardiology*, *71*(19), e127–e248.

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