An association between right ventricular dysfunction and sudden cardiac death

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BACKGROUND The effectiveness of severely reduced left ventricular ejection fraction (LVEF <35%) as a predictor of sudden cardiac death (SCD) has diminished, and improvements in risk stratification await discovery of novel markers. Right ventricular (RV) abnormalities can be observed in conditions such as chronic obstructive pulmonary disease and sleep apnea, which have been linked to SCD.

OBJECTIVE The purpose of this study was to evaluate whether RV abnormalities were associated with SCD after accounting for LVEF and other patient characteristics.

METHODS In a large, prospective ongoing community-based study of SCD in the Portland, Oregon, metropolitan area, SCD cases (age ≥18 years; 2002–2014) were compared to controls with coronary artery disease but no SCD. Using a novel archive of digital echocardiograms, a standardized approach was used to evaluate RV basal diameter, RV end-diastolic area, and right ventricular fractional area change (RVFAC).

RESULTS A total of 350 subjects were studied, including 81 SCD cases (age 68.7 ± 13.6 years; 73% male) and 269 controls (age 66.5 ± 10.2 years; 69% male). In multivariate analysis, RVFAC was significantly associated with SCD (odds ratio 1.14 for each 5% decrease; 95% confidence interval 1.03–1.25; \(P = .01\)). When modeled with LVEF ≤35%, RVFAC ≤35% was significantly associated with increased risk of SCD. Individuals with both left ventricular and RV dysfunction had a 3× higher odds of SCD than those with neither (odds ratio 3.19; 95% confidence interval 1.33–7.68; \(P = .01\)).

CONCLUSION RV dysfunction was associated with a significantly increased risk of SCD independent of LVEF and, when combined with LVEF, had additive effects on SCD risk.

KEYWORDS Cardiac arrest; Echocardiogram; Right ventricle; Risk prediction; Sudden death

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Introduction

Sudden cardiac death (SCD) remains a significant public health problem worldwide, with an annual incidence in the United States of approximately 350,000.1 In the landmark primary prevention trials, implantable cardioverter-defibrillators (ICDs) were shown to reduce mortality from SCD by delivering timely shock therapies.2,3

Indications for ICDs, however, remain narrow and rely primarily on a reduced left ventricular ejection fraction (LVEF) <35%.4 Recent studies have indicated that LVEF <35% is likely to be inadequate as the sole risk stratification criterion for SCD, especially because it accounts for only approximately one-third of cases.5–8 In a recent meta-analysis performed from the ARIC (Atherosclerosis Risk in Communities) and CHS (Cardiovascular Health Study) cohorts, Konyet et al.10 reported associations between SCD and multiple echocardiographic predictors of SCD, including LVEF, mitral annular calcification, increased left ventricular (LV) mass, increased left atrial diameter, and abnormal LV geometry. With these findings, we know that the echocardiogram is likely to provide more SCD predictors than LVEF alone. Recently, conditions such as chronic obstructive pulmonary disease (COPD)11 and obstructive sleep apnea (OSA)12 have been associated with increased SCD risk. Both of these conditions are known to be causal in...
pulmonary hypertension and have detrimental long-term effects on function of the right ventricle (RV). To date, few studies have evaluated the impact of RV dysfunction on the risk of SCD and what may be the best echocardiographic assessment for this purpose. Therefore, this study sought to determine whether standard measures of RV function, including right ventricular fractional area change (RVFAC), right ventricular end-diastolic area (RVEDA), and right ventricular basal diameter (RVBD), could be utilized as novel SCD predictors.

**Methods**

**Study population**

Case subjects included in this analysis were drawn from the ongoing Oregon Sudden Unexpected Death Study (Oregon SUDS), from SCD cases that occurred between February 1, 2002, and January 31, 2015, in the Portland, Oregon, metropolitan area (population ~1 million). As previously published from Oregon SUDS, SCD cases were identified via multiple sources, including fire department, ambulance services, local hospital emergency rooms, and the county medical examiner’s office. SCD was defined as a sudden and unexpected pulseless condition of likely cardiac etiology if witnessed, and a sudden death within 24 hours of last having been seen in a usual state of health if unwitnessed. All identifiable noncardiac causes of death, including trauma, drug overdose, pulmonary embolism, cerebrovascular accident, and chronic terminal illness, were excluded. Survivors of SCD were included as cases. All cases of SCD were adjudicated via a 3-physician review panel with access to all available medical records and autopsy reports. Control subjects were obtained from multiple sources, including chest pain patients attended by emergency medical services, outpatient clinics, patients undergoing angiography, and patients from a large health maintenance organization in the Portland metropolitan area. Control subjects were selected to be enriched for coronary artery disease, and 91% of controls included in the analysis had coronary artery disease, defined as ≥50% stenosis of a major coronary artery, history of myocardial infarction, or history of coronary artery bypass grafting or percutaneous coronary intervention. Controls with a previous history of ventricular arrhythmia or cardiac arrest were excluded. For this analysis, we included all subjects age ≥18 years with a digital echocardiogram available in the electronic medical records. For SCD cases, the digital echocardiogram closest and before the SCD event was used for analysis. Digital echocardiogram files of cases and controls were de-identified and stored on a password-protected digital archive in a core laboratory. This study was approved by the institutional review boards of Cedars-Sinai Medical Center, Oregon Health and Science University, and all participating hospitals and health systems. All survivors of cardiac arrest provided informed consent; this requirement was waived for nonsurvivors.

**Echocardiographic assessment**

Using a 3-physician blinded and standardized reading protocol, direct measurements were made from the digital echocardiograms of SCD cases and controls. The primary reader (SP) made all quantitative measurements of echocardiograms using ScImage PICOM 365 software (ScImage, Los Altos, CA). All measurements were then overseen by a second reader (TN), a cardiologist with special expertise in echocardiography. In case of disagreement, a second cardiologist with special expertise in echocardiography (TS) reviewed the echocardiogram, and the majority vote determined the evaluation. LVEF was measured using the standard Simpson biplane method in the 4- and 2-chamber views. To determine the presence of left ventricular hypertrophy (LVH), measurements were made in the parasternal long axis of the left ventricular internal diameter in diastole (LVIDD), posterior wall thickness in diastole (PWTD), and interventricular septal thickness in diastole (IVSTD). Using the formula recommended by the American Society of Echocardiography, LV mass index was then calculated as (0.8 × (LVIDD + PWTD + IVSTD)² - (LVIDD)²) + 0.6) g divided by the body surface area in m². LVH was defined as an LV mass index >134 g/m² for men and 110 g/m² for women. Using the 4-chamber view, measurements were made of RVEDA, RV end-systolic area (RVESA), and RVBD (Figure 1) per the 2010 American Society of Echocardiography guidelines. RVFAC was calculated as the difference between RVEDA and RVESA, divided by RVEDA, and multiplied by 100%. Other measures of RV function, such as tricuspid annular plane systolic excursion (TAPSE), RV index of myocardial performance, tricuspid annular S’ wave velocity, RV strain, and RV systolic pressure, were not consistently measurable in the majority of digital echocardiograms and thus were not included for comparison in this study.

**Statistical analysis**

Baseline characteristics of SCD cases and controls, including age, sex, and presence of hypertension, diabetes mellitus, obesity (body mass index ≥30), sleep apnea, and COPD, were compared using independent-samples Student t tests with statistical significance set at a 2-tailed P ≤.05. Additionally, the presence of LVH, mean LVEF, and proportion with LVEF ≤35% were compared using available data from the digital echocardiograms. Similarly, the measured RV parameters of RVBD, RVEDA, and RVFAC were compared. Correlations were evaluated between RV measures of function, and, based on this, a multivariable logistic regression analysis was used to calculate an odds ratio and 95% confidence interval for standard unit changes in RVEDA and RVFAC. An additional multivariable analysis was performed to evaluate SCD risk in subjects with only LVEF ≤35% or RVFAC ≤35%, and those with both LVEF ≤35% and RVFAC ≤35%. A joint distribution χ² analysis was also performed between these groups and the reference group of neither LVEF ≤35% nor RVFAC ≤35%. Both logistic regression models were adjusted for age, sex, diabetes...
mellitus, LVH, and LVEF. All analyses were performed using SAS version 9.3 (SAS Institute Inc, Cary, NC).

Results
Baseline characteristics
Digital echocardiograms were available for a total of 81 SCD cases and 269 controls. For 50 of the 81 cases (62%), echocardiograms had been performed within 1 year of arrest. For 210 of 269 controls (78%), echocardiograms had been performed within 1 year of ascertainment. There was no significant difference between the groups with regard to age or sex, with a male predominance in both groups (Table 1). There was no difference in the prevalence of most comorbid conditions, including hypertension, obesity, COPD, and sleep apnea. However, the prevalence of diabetes mellitus was higher in SCD cases than controls (59% vs 40%; \(P=.001\)), as was prevalence of LVH (38% vs 17%; \(P<.001\)). Mean LVEF was lower in SCD cases (0.44 ± 0.14 vs 0.47 ± 0.12; \(P=.04\)), and the proportion with LVEF ≤35% was higher in SCD cases (32% vs 19%; \(P=.01\)).

Measures of RV function
Differences were observed between SCD cases and controls for the mean values of each measure of RV function (Table 2). RVBD was higher in SCD cases (45.2 ± 9.9 vs 42.2 ± 8.7; \(P=.008\)). RVEDA was higher in SCD cases (24.7 ± 8.5 vs 22.4 ± 7.1; \(P=.03\)). RVFAC was lower in SCD cases (0.38 ± 0.14 vs 0.45 ± 0.14; \(P<.001\)). The proportion of subjects with RVFAC <35% was also somewhat higher in SCD cases (38% vs 28%; \(P=.07\)). A boxplot distribution of RVFAC is shown in Figure 2.

Before simultaneous modeling of RV and other measures, we examined correlations between RV variables. RVBD and RVEDA were strongly positively correlated (\(r = 0.75\); \(P<.001\)), whereas RVFAC was less strongly and negatively correlated with both RVBD and RVEDA (\(r = -0.20\); \(P<.001\) and \(r = -0.22\); \(P<.001\), respectively). Because RVBD is a component of RVEDA, we included only RVEDA in the combined model with RVFAC. In a multivariable analysis including both RVFAC and RVEDA, RVFAC was significantly associated with SCD (odds ratio [OR] 1.14 for each 5% decrease; 95% confidence interval [CI] 1.03–1.25; \(P=.01\)) (Table 3). In this model, however, RVEDA did not significantly affect the risk of SCD (OR 1.10 for each 5-cm² increase; 95% CI 0.92–1.32; \(P=.29\)). A separate multivariable analysis was also performed to determine the additive effect of RVFAC to LVEF in SCD risk prediction by modeling them together (Table 4). For those with only either LVEF ≤35% or RVFAC ≤35% there was no significant increase in the risk of SCD. However, in those with both LVEF ≤35% and RVFAC ≤35%, there was a significant increase in the risk of SCD (OR 3.19; 95% CI 1.33–7.68; \(P=.01\)). Joint distribution for subjects with and without LV or RV dysfunction shows that 16% of SCD cases...

**Table 1** Baseline clinical characteristics of SCD cases vs controls

<table>
<thead>
<tr>
<th>Total (N = 350)</th>
<th>SCD cases (n = 81)</th>
<th>Controls (n = 269)</th>
<th>(P) value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (y)</strong></td>
<td>68.7 ± 13.6</td>
<td>66.5 ± 10.2</td>
<td>.17</td>
</tr>
<tr>
<td>Male sex</td>
<td>59 (73)</td>
<td>186 (69)</td>
<td>.52</td>
</tr>
<tr>
<td>Hypertension</td>
<td>69 (85)</td>
<td>216 (81)</td>
<td>.35</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>48 (59)</td>
<td>104 (39)</td>
<td>.001</td>
</tr>
<tr>
<td>Obese (body mass index ≥30)</td>
<td>33 (45)</td>
<td>116 (44)</td>
<td>.92</td>
</tr>
<tr>
<td>Sleep apnea</td>
<td>16 (20)</td>
<td>34 (13)</td>
<td>.11</td>
</tr>
<tr>
<td>COPD</td>
<td>19 (23)</td>
<td>44 (16)</td>
<td>.15</td>
</tr>
<tr>
<td>LVH</td>
<td>28 (38)</td>
<td>45 (17)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mean LVEF</td>
<td>0.44 ± 0.14</td>
<td>0.47 ± 0.12</td>
<td>.04</td>
</tr>
<tr>
<td>LVEF ≤35%</td>
<td>25 (31)</td>
<td>47 (17)</td>
<td>.009</td>
</tr>
</tbody>
</table>

Values are given as mean ± SD or n (%) unless otherwise indicated. Diabetes, hypertension, chronic obstructive pulmonary disease (COPD), and sleep apnea history missing for 1 control; obesity missing for 7 cases and 5 controls; left ventricular hypertrophy (LVH) missing for 7 cases and 9 controls.

LVEF = left ventricular ejection fraction; SCD = sudden cardiac death.
Discussion
To our knowledge, this is the first population-based study to report the association between reduced RV function and increased risk of SCD. We evaluated several potential quantitative measures of RV function to determine which could be reliably obtained in clinically acquired echocardiograms. Incremental changes in RVFAC were significantly associated with SCD in a multivariable analysis, independent of LVEF. In addition, our analysis demonstrated that when combined with LVEF ≤35%, RVFAC ≤35% had an additive effect on prediction of SCD. This indicates that RVFAC has the potential to enhance the current approach to SCD risk stratification beyond LVEF.

RV dysfunction is emerging as a novel marker for risk stratification in SCD. Although RVFAC is a standard measure for echocardiographic assessment of RV function, it is not clear at this time which measure of RV function would be the most suitable for risk stratification. Previous work has identified RV dysfunction as a potential predictor of SCD risk, although it has not been shown for quantitative measures in clinically acquired echocardiograms. Aktas et al reported that severe RV dysfunction as subjectively determined by the reader of a 2-dimensional echocardiogram was independently associated with a combined endpoint of ICD therapy or death in a population that received ICDs for primary prevention of SCD. More recently, Makami et al prospectively used right ventricular ejection fraction (RVEF) as measured by cardiac magnetic resonance imaging, the gold standard for determining RV function, and were able to demonstrate that a reduced RVEF was a strong, independent predictor of arrhythmic events in a population with known systolic dysfunction by LVEF ≤54%. Risum et al found that RV free-wall strain as measured by 2-dimensional echocardiographic analysis was significantly associated with ventricular arrhythmias/SCD and superior as a predictor compared to TAPSE in an acute myocardial infarction population. In one of the largest studies to date of 5463 subjects who all had been admitted to the coronary care unit at Mayo Clinic, Rochester, Minnesota, Naksuk et al found that moderate to severe RV dysfunction as determined jointly by TAPSE, RV index of myocardial performance, and tricuspid annular S’ wave velocity was an incremental predictor of SCD in both patients with LVEF ≤35% and those with LVEF >35%. These studies all indicate that RV dysfunction by various means of assessment can be predictive of SCD risk.

In a recent meta-analysis by Lee et al, RVFAC was compared with TAPSE for its ability to correlate with RVEF by cardiac magnetic resonance imaging. RVFAC was found to be superior in this regard, likely because it is a 2-dimensional measurement, which allows it to account for regional differences in RV function.

RV failure can result from many different etiologies, but most notably those that cause pulmonary hypertension by chronic hypoxemia. In 2015, Oregon SUDS demonstrated a link between COPD and SCD using 728 adjudicated cases. COPD was significantly associated with SCD (OR 2.2) independent of LVEF, medications, clinical markers, and electrocardiographic markers using a propensity score matched analysis. This relationship was found to be even stronger in subjects who had COPD and used short-acting beta-agonists but no beta-blockers (OR 3.3). OSA has also been linked to SCD. Gami et al prospectively ascertained 142 cases of SCD in a cohort of >10,000

### Table 2: Measures of RV function in cases vs controls

<table>
<thead>
<tr>
<th>Measures of RV function</th>
<th>Cases (n = 81)</th>
<th>Controls (n = 269)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVBD 5-cm² increase</td>
<td>45.2 ± 9.9</td>
<td>42.2 ± 8.7</td>
<td>.008</td>
</tr>
<tr>
<td>RVFAC 5-cm² increase</td>
<td>24.7 ± 8.5</td>
<td>22.4 ± 7.1</td>
<td>.03</td>
</tr>
<tr>
<td>RVFAC ≤35%</td>
<td>0.38 ± 0.14</td>
<td>0.45 ± 0.14</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>RVEDA ≤5-cm² increase</td>
<td>31 (38)</td>
<td>75 (28)</td>
<td>.07</td>
</tr>
</tbody>
</table>

Values are given as mean ± SD or n (%) unless otherwise indicated.

RV = right ventricle; RVBD = right ventricular basal diameter; RVEDA = right ventricular end-diastolic area; RVFAC = right ventricular fractional area change.

Discussion
To our knowledge, this is the first population-based study to report the association between reduced RV function and increased risk of SCD. We evaluated several potential quantitative measures of RV function to determine which could be reliably obtained in clinically acquired echocardiograms. Incremental changes in RVFAC were significantly associated with SCD in a multivariable analysis, independent of LVEF. In addition, our analysis demonstrated that when combined with LVEF ≤35%, RVFAC ≤35% had an additive effect on prediction of SCD. This indicates that RVFAC has the potential to enhance the current approach to SCD risk stratification beyond LVEF.

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### Table 3: Multivariable analysis for measures of RV function as predictors of SCD

<table>
<thead>
<tr>
<th>Odds ratio</th>
<th>95% Confidence interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>5-cm² increase</td>
<td>1.10</td>
<td>.001</td>
</tr>
<tr>
<td>in RVEDA</td>
<td>1.14</td>
<td>.01</td>
</tr>
<tr>
<td>5% decrease in RVFAC</td>
<td>1.14</td>
<td>.01</td>
</tr>
</tbody>
</table>

Model adjusted for age, sex, diabetes, left ventricular hypertrophy (LVH), and left ventricular ejection fraction (LVEF), including 74 cases and 259 controls with complete data. Model C statistic = 0.702. Model C statistic without right ventricular (RV) measures (including only age, sex, diabetes, LVH, and LVEF) = 0.677.

RVFAC = right ventricular fractional area change; SCD = sudden cardiac death.
patients undergoing routine polysomnography and demonstrated that OSA along with its multiple parameters of severity were significantly predictive of SCD.

The mechanistic link between SCD and chronic hypoxic conditions such as COPD and sleep apnea requires further investigation, but several factors can potentially be implicated. In individuals with heart disease, chronic hypoxemia would be expected to have a detrimental effect due to reduced myocardial oxygen supply, especially during times of activity.26 Chronic hypoxia is also known to cause RV remodeling that over time may increase the arrhythmogenicity of the RV by substrate modification.14 QTc intervals have been demonstrated to have increased duration and dispersion in both COPD and sleep apnea.27–29 These changes over time along with the increased sympathetic tone during hypoxic episodes30 can increase the potential for ventricular ectopy and subsequent deadly arrhythmias. This is further supported in Oregon SUDS by the protective effect against SCD that cardioselective beta blockers seemed to have in COPD patients taking short-acting beta-agonists.11

At this time, there is significant evidence that LVEF is inadequate as the primary risk stratification tool for SCD.5–9 In order to improve risk stratification, other novel markers need to be identified and studied for their additive benefit to LVEF in risk prediction.31 LVH has been previously demonstrated to predict risk of SCD (OR 1.8) independent of severely reduced LVEF (OR 1.9) in Oregon SUDS,32 with LVH and severely reduced LVEF having an additive effect on SCD risk (OR 3.5). Our study similarly demonstrated that RV dysfunction was independently associated with an increased risk of SCD and had an additive effect when combined with LVEF. When RVFAC ≤35% was combined with LVEF ≤35%, SCD risk prediction improved (OR 3.19). Thus, this novel marker may have significant prognostic value in predicting SCD and improving risk stratification strategies. Given the inherent limitations of a case-control design, these results are not yet definitive, and larger prospective studies of RVFAC in comparison with other measures of RV function are warranted.

### Study limitations

Given that SCD occurs relatively infrequently in the general population (approximately 50 in 100,000 residents), we used a population-based case-control design to accrue feasible numbers for analysis. There are inherent limitations in community-based studies compared to cohort studies, including missing information for patients who may not have seen a cardiologist and therefore did not have an echocardiogram recorded before their SCD event. Our results may be generalizable to individuals who have undergone clinically indicated echocardiograms, a potentially important intermediate-risk population. Furthermore, a digital echocardiogram file from each subject was required to perform a standardized reading of echocardiograms, which also reduced sample size. With this comes the possibility that the selected cases may not be perfectly representative of the parent population. However, the comorbidity profile (obesity, hypertension, COPD, sleep apnea) was not significantly different comparing individuals with available digital echocardiograms to individuals who had echocardiogram results reported in clinical records but for whom no digital image was retrieved. Limiting the analysis to individuals with digital files available allowed standardized reading of all digital echocardiograms. Patients in our study did not have data on severity of pulmonary disease. We were able to assess pulmonary hypertension (by TR velocity) and loading (by diameter of the inferior vena cava) in a subset of echocardiograms, but each variable was missing for approximately 40% of subjects included in this analysis. When TR velocity and inferior vena cava diameter were included in a multivariable model in the subset with available data, the association of RVFAC with SCD was consistent but somewhat attenuated (from OR 1.14 per 5% decrease to OR 1.11 per 5% decrease). Future prospective studies would be well supplemented by including data on loading, pulmonary artery pressures, and severity of comorbid pulmonary disease such as FEV1 for COPD and apnea–hypopnea index for sleep apnea.

### Table 4

<table>
<thead>
<tr>
<th>RVFAC ≤35% only</th>
<th>LVEF ≤35% only</th>
<th>Both LVEF and RVFAC ≤35%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Odds ratio*</td>
<td>1.99</td>
<td>3.19</td>
</tr>
<tr>
<td>95% Confidence interval</td>
<td>0.89–4.48</td>
<td>1.33–7.68</td>
</tr>
<tr>
<td>P value</td>
<td>.10</td>
<td>.01</td>
</tr>
</tbody>
</table>

Model includes 74 cases and 259 controls with complete data. Model C statistic = 0.699.

LV = left ventricle; RV = right ventricle; SCD = sudden cardiac death.

*Reference category: Neither left ventricular ejection fraction (LVEF) nor right ventricular fractional area change (RVFAC) ≤35%. Model adjusted for age, sex, diabetes, and left ventricular hypertrophy.

### Figure 3

Joint distribution for left ventricular and right ventricular dysfunction. The proportion of subjects with both left ventricular ejection fraction (LVEF) and right ventricular fractional area change (RVFAC) ≤35% was 16% of cases vs 7% of controls. $\chi^2 P = .03$. 

...
Conclusion
In this population, RVFAC was independently associated with risk of SCD using a novel digital echocardiogram archive with a standardized reading protocol. When combined with LVEF, RVFAC had additive effects on SCD risk. These findings have potential implications for SCD risk stratification and warrant further prospective evaluation in larger populations.

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The authors would like to acknowledge the significant contribution of American Medical Response, the Portland/Gresham Fire Departments, and the Oregon State Medical Examiner's office.

Appendix
Supplementary data

Interview video associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2019.10.021.

References