

Utility of electrocardiographic findings in acute pulmonary embolism

Alexander E. Sullivan ^{1,*†}, Tara A. Holder^{1,2,†}, Joshua A. Beckman³,
Cynthia L. Green^{4,5}, Manesh R. Patel^{4,6}, Terry A. Fortin⁶, and W. Schuyler Jones^{4,6}

¹Department of Medicine, Vanderbilt University Medical Center, 1215 21st Ave S, Suite 5468A, Nashville, TN 37232, USA; ²Department of Medicine, Division of Cardiology, Prisma Health, Greenville, SC 29605, USA; ³Division of Vascular Medicine, Department of Medicine, University of Texas Southwestern, Dallas, TX 75390, USA; ⁴Duke Clinical Research Institute, Duke University School of Medicine, Durham, NC 27701, USA; ⁵Department of Biostatistics and Bioinformatics, Duke University School of Medicine, Durham, NC 27701, USA; and ⁶Division of Cardiology, Department of Medicine, Duke University School of Medicine, Durham, NC 27710, USA

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Utilization of the electrocardiogram (ECG) to assess the diagnostic probability and severity of pulmonary embolism (PE) remains common in clinical settings, yet its role is unclear in clinical practice guidelines and is excluded from common clinical prediction rules.¹ Historically, ECG findings considered specific for PE have been used as an initial low-cost screening tool for the presence or absence of PE with right ventricular (RV) dysfunction. Many ECG findings have been noted to predict RV strain and dysfunction, including sinus tachycardia, the presence of an S-wave in lead I, Q-wave in lead III, and T-wave inversion in lead III (S1Q3T3), complete and incomplete right bundle branch block (RBBB), T-wave inversions in V₁–V₄, and ST-segment elevation in aVR and V₁ with and without concomitant ST-segment depressions in V₄–V₆. These ECG changes, however, are rare and have not been consistently associated with RV dysfunction.^{2,3} We investigated the frequency of traditional ECG markers of PE and its complications in a large, diverse cohort of patients with acute PE.

Consecutive patients with imaging-confirmed acute PE who presented to Duke University Medical Center from 1 January 2016 to 30 June 2017 were retrospectively identified using International Classification of Diseases, Tenth Revision codes, as previously described.⁴ Clinical data and ECG data (closest in time to diagnostic imaging study) were abstracted by two physicians (T.A.H. and T.A.F.) that were blinded to clinical data and outcomes. To limit inclusion of chronic ECG abnormalities, ST-segment deviation, T-wave inversion, and S1Q3T3 were coded if new from prior ECG or if no baseline ECG was available. Pulmonary embolism risk classification was performed in accordance with European Society of Cardiology 2019 Guidelines for Acute Pulmonary Embolism.¹ This study was Institutional Review Board exempt, and no informed consent was required.

Of 829 patients diagnosed with PE, 676 had an available ECG within 24 h of PE diagnosis and were included in the study. Sinus rhythm was the most common rhythm found in 86.9% of patients (Table 1). Heart rates were between 60 and 99 beats per minute in 45.6% and ≥ 100

beats per minute in 41.3% of patients. Atrial tachyarrhythmias were seen in 8.7% of patients. Electrocardiogram patterns of RV strain were noted in 29.4% of patients. The S1Q3T3 pattern was the most common ($n = 70$, 10.4%) followed by T-wave inversions in V₁–V₄ ($n = 66$, 9.8%), atrial fibrillation or flutter ($n = 56$, 8.3%), ST-segment elevation in aVR ($n = 35$, 5.2%), and RBBB ($n = 31$, 4.6%) (Table 1). All but 36 patients with an abnormal ECG pattern underwent echocardiography. Hypotension (15.6% vs. 6.9%), tachycardia (69.3% vs. 58.7%), elevated troponin (20.1% vs. 8.8%), elevated pro-B-type natriuretic peptide (46.7% vs. 25.4%), and echocardiographic RV enlargement (53.3% vs. 23.1%) and hypokinesia (48.2% vs. 18.4%) were more common in patients with abnormal ECG patterns (all $P < 0.01$). Intermediate high-risk (35.7% vs. 14.5%) and high-risk ESC classifications (12.6% vs. 4.2%) and intensive care unit admission (38.2% vs. 22.2%) were also more common in patients with ECG abnormalities. In-hospital mortality (4.5% vs. 3.8%, $P = 0.65$) was similar between the two groups. The frequency of specific ECG patterns and PE-related clinical parameters are shown in Table 1.

The current data demonstrate that one-third of patients with PE will have an abnormal ECG pattern and are more likely to have RV dysfunction, but the utility of ECG as a point-of-care surrogate for an individual patient remains limited. Nearly, 50% of patients with an abnormal ECG pattern did not have evidence of RV dysfunction on echocardiography. Furthermore, 51% of patients with RV enlargement and 48% of patients with RV hypokinesia had a normal ECG. This limitation is compounded when considering each pattern individually. S1Q3T3 has been classically associated with RV dysfunction in acute PE and was the most common pattern in this cohort. This finding was only seen in 20% and 22% of patients with RV enlargement and hypokinesia, respectively, and 37% of patients with S1Q3T3 had no echocardiographic evidence of RV dysfunction. Others have suggested that these uncommon ECG patterns are useful because they have high specificity and negative predictive

* Corresponding author. Tel: 615-936-4986, Fax: 615-936-1872, Email: alexander.sullivan@vumc.org

† The first two authors contributed equally to the study.

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Table 1 Baseline characteristics, frequency of ECG findings, and association with adverse clinical endpoints

| Baseline characteristics (N = 676) | | | | | |
|-------------------------------------------|-------------|-------------------|------------------------|-----------------------|--------------------|
| Age at PE diagnosis, mean (SD), years | | 62.4 (16.8) | | | |
| BMI, mean (SD) | | 31.3 (8.7) | | | |
| Female gender | | 357 (52.8%) | | | |
| Race | | | | | |
| White | | 363 (53.7%) | | | |
| Black | | 277 (41.0%) | | | |
| Other | | 36 (5.3%) | | | |
| Heart rate | | | | | |
| Mean (SD) | | 98.2 (21.9) | | | |
| HR <60 | | 19 (2.8%) | | | |
| HR 60–99 | | 328 (48.5%) | | | |
| HR 100–109 | | 138 (20.4%) | | | |
| HR ≥110 | | 191 (28.3%) | | | |
| Heart rhythm | | | | | |
| Sinus and HR <60 | | 18 (2.7%) | | | |
| Sinus and HR 60–99 | | 308 (45.6%) | | | |
| Sinus and HR ≥100 | | 279 (41.3%) | | | |
| Atrial flutter | | 12 (1.8%) | | | |
| Atrial fibrillation | | 44 (6.5%) | | | |
| Multifocal atrial tachycardia | | 3 (0.4%) | | | |
| Other | | 12 (1.8%) | | | |
| ECG finding | | HR >100 | RV Enlargement* | RV hypokinesis | Mortality** |
| Any abnormal ECG finding | 199 (29.4%) | 138 (69.3%) | 106 (53.3%) | 96 (48.2%) | 9 (4.5%) |
| S1Q3T3 | 70 (10.4%) | 48 (68.6%) | 44 (62.9%) | 41 (58.6%) | 1 (1.4%) |
| T-wave inversions V1–V4 | 66 (9.8%) | 45 (68.2%) | 45 (68.1%) | 44 (66.7%) | 3 (4.5%) |
| Atrial fibrillation or flutter | 56 (8.3%) | 42 (75.0%) | 21 (37.5%) | 17 (30.4%) | 6 (10.7%) |
| ST-elevation aVR | 35 (5.2%) | 30 (85.7%) | 19 (54.3%) | 19 (54.3%) | 3 (8.6%) |
| RBBB | 31 (4.6%) | 16 (51.6%) | 15 (48.4%) | 11 (35.5%) | 1 (3.2%) |
| ST-depression V4–V6 | 22 (3.3%) | 17 (77.3%) | 7 (31.8%) | 7 (31.8%) | 1 (4.5%) |
| ST-elevation aVR and ST-depression V4–V6 | 13 (1.9%) | 11 (84.6%) | 6 (46.2%) | 7 (53.8%) | 1 (7.7%) |
| Incomplete RBBB | 13 (1.9%) | 9 (69.2%) | 9 (69.2%) | 9 (69.2%) | 1 (7.7%) |
| ST-elevation aVR and V1 | 2 (0.3%) | 2 (100.0%) | 2 (100.0%) | 2 (100.0%) | 2 (100.0%) |

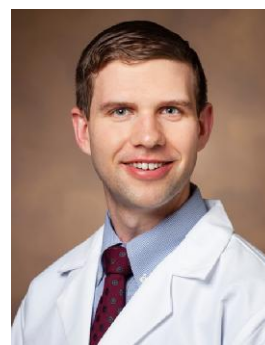
*Basal diameter >4.1 cm.

**In-hospital.

BMI, body mass index; ECG, electrocardiogram; HR, heart rate; PE, pulmonary embolism; RBBB, right bundle branch block; RV, right ventricular; SD, standard deviation.

values for RV dysfunction and 30-day mortality.³ It is important to put these data in the context of the overall incidence of these ECG patterns and clinical outcomes in patients with acute PE. These findings do not obviate other methods of RV assessment, and patients with isolated RV strain on ECG but without dysfunction on computed tomography or transthoracic echocardiogram have a lower rate of adverse events than patients without RV strain on any modality.² While a relationship between ECG findings and RV dysfunction or mortality has been born out of cohorts like ours, the association is not strong enough to have utility for the frontline clinician.^{2,3} While persistent RV strain on serial ECGs is a strong prognostic marker of 30-day mortality, the predictive value from a single admission ECG is not strong enough to alter the initial diagnostic evaluation and management.⁵ The focus should remain on clinical and imaging features associated with adverse outcomes.

Lead author biography



Dr Alexander E. Sullivan is a general cardiology fellow at Vanderbilt University Medical Center in Nashville, TN. He completed medical school at the George Washington University School of Medicine and residency training in internal medicine at Duke University Medical Center. Under the mentorship of Dr W. Schuyler Jones, he developed a passion for vascular disease. He will complete training in interventional cardiology and plans a career as a physician investigator with a focus on the clinical and translational mechanisms of vascular disease.

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Data availability

Research data are not shared.

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