# RIGHT VENTRICULAR OUTFLOW TRACT FRACTIONAL SHORTENING – A PROGNOSTIC MARKER IN NON-HIGH RISK PATIENTS WITH ACUTE PULMONARY EMBOLISM

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***Abstract:*** *In normotensive, non-high risk patients with acute pulmonary embolism (APE), the presence of right ventricular dysfunction is a marker of increased mortality risk, although it still has no generally accepted clear definition. The aim of the study is to evaluate right ventricular outflow tract (RVOT) fractional shortening as a prognostic marker for increased early mortality. We performed echocardiography on 68 consecutive patients with confirmed, non-high risk APE following a standardized protocol, and correlated markers of right ventricular dysfunction with the occurrence of 30-day mortality and adverse events. Mean RVOT fractional shortening was lower in the composite end-point group (19% vs. 24%), but not significant (p=0.148) due to the low number of events (6 patients). Also, it was strongly significantly correlated with the presence of markers of right ventricular dysfunction with proven prognostic value. RVOT fractional shortening is a marker of increased mortality or adverse events in non-high risk APE patients.*

## INTRODUCTION

Acute pulmonary embolism (APE) is an urgent, common and possibly life-threatening condition, bordering on the activity of many medical specialties. Early mortality in these patients is high, with approximately 10% of all patients dying during the first three months.(1) Current management of suspected APE patients is based on an initial division into patients with a high risk of 30-day mortality, in excess of 15%, and non-high risk patients.(2) The basis for prognostic stratification is the presence of systemic hypotension, defined as a systolic blood pressure below 90 mmHg, or shock, which is indicative of patients at high-risk.(2) Patients with APE presenting with hypotension or shock require more aggressive management strategies, including primary reperfusion treatment with systemic thrombolysis.(2)

In hemodynamically stable, non-high risk APE patients, 30-day mortality risk stratification is performed with the purpose of guiding therapeutic strategies and the duration of hospitalization.(2) In these patients, no individual clinical, laboratory or imaging finding has been shown to predict adverse in-hospital events with enough power in order to justify a more aggressive management strategy. As a result, 30-day mortality risk is evaluated using validated clinical prognostic scores that use combinations of various parameters (3,4) and by identifying right ventricular dysfunction (RVD), through echocardiography or computer tomography angiography, and elevated plasma cardiac biomarker levels, cardiac troponins or natriuretic peptides.(5)

Echocardiography is the mainstay of RVD evaluation in APE patients.(6) RVD is now acknowledged as an important part of the risk stratification criteria in APE,(2) but it still has no generally accepted clear definition. The right ventricle (RV) has a complex geometry and no one single measurement capable of defining its systolic function,(7) thus, different criteria are in use for assessing RVD.(8) The right ventricle outflow tract (RVOT) is a distinct anatomic region and its function is closely linked with pulmonary artery pressure.(9) Based upon its unique anatomy, we hypothesized that the systolic function of the RVOT might be correlated with the severity of APE and be associated with an increased early mortality risk.

## PURPOSE

The aim of this study was to assess right ventricular outflow tract fractional shortening in patients with non-high risk acute pulmonary embolism and to determine its predictive value for increased early mortality. The objectives were to evaluate echocardiographic parameters of right ventricular dysfunction in normotensive patients with APE and to identify their correlations with 30-day mortality and adverse events occurrence.

## MATERIALS AND METHODS

We performed a prospective observational study of patients with non-high risk APE, consecutively admitted in the adult cardiology ward of the Institute of Emergency Cardiovascular Diseases and Transplant of Tîrgu-Mures. APE was confirmed by contrast medium enhanced computer tomography, performed at admittance, which identified filling defects in at least one of the main pulmonary arteries or branches. After the diagnosis of APE was established, high-risk patients were identified by the presence of systemic hypotension, identified by a systolic arterial blood pressure of less 90 mm Hg, or other signs of peripheral hypoperfusion. The time passed from symptom occurrence was evaluated for all patients, and these included new onset dyspnea, chest pain, syncope or hemoptysis.

The exclusion criteria consisted of patients presenting high-risk APE with systemic hypotension, patients with symptom onset older than 7 days and patients with prior pulmonary or heart disease suspected of chronic elevated

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pulmonary artery pressure. The inclusion of patients was performed after obtaining written informed consent.

Echocardiography was performed as soon as possible after admission by experienced physicians, always using the same machine, a GE Vivid S5 system (General Electric, Fairfield, CT, USA), following a standardized protocol that included the measurements and views in which these were acquired, in order to reduce interobserver variability. Patients were examined in the supine position. The examinations were recorded and reinterpreted when needed. Parameters of right ventricular function were measured as following: in the parasternal long-axis view RV and left ventricle (LV) end-diastolic diameters, in the parasternal short-axis view pulmonary ejection acceleration time (PAT) using continuous Doppler, in the apical 4-chamber view tricuspid annulus plane systolic excursion (TAPSE) using M-mode by placing the cursor at the level of the lateral annulus of the tricuspid valve, tricuspid regurgitation pressure gradient (TRPG) using continuous Doppler and the presence of McConnell's sign and in the subcostal view the diameter and inspiratory collapse of the inferior vena cava (IVC). End diastolic RV/LV diameter ratio was calculated, right atrial pressure was estimated using IVC diameter and inspiratory collapse and systolic pulmonary artery pressure (sPAP) was calculated by adding the right atrial pressure and the TRPG.

The RVOT diameter was measured using the parasternal short axis view at the level of the aortic root using two-dimensional imaging, by measuring the end-diastolic and end-systolic diameters of the RVOT parallel to the transversal axis of the RVOT. RVOT fractional shortening was calculated as the percentage of the end-systolic RVOT diameter reduction in respect to the end-diastolic RVOT diameter (figure no. 1).

**Figure no. 1. Measurement of right ventricular outflow tract fractional shortening from the parasternal short-axis view**


After examination on admission, patients were followed for 30 days. The composite endpoint was 30-day APE-related mortality and the occurrence of APE recurrence, ischemic stroke or acute myocardial infarction. Local ethics committee approval was obtained for the study protocol.

Statistical analysis was performed using SPSS v20 (IBM, Armonk, NY, USA) and Microsoft Excel 2010 (Microsoft Corporation, Redmond, WA, USA). Numerical data was analysed for normal distribution using Kolmogorov-Smirnov and Shapiro-Wilk tests and non-parametric tests were used when needed. The student t test was used to compare the means of two groups and the Chi-square test with Yates correction was used to compare discrete variables. Receiver operating characteristic (ROC) curves were analysed to assess the predictive values of RVD parameters for the composite endpoints. Correlations between RVOT fractional shortening and other RVD parameters were analysed using Pearson's product-moment or Spearman's rank correlation coefficients. A p value of less than 0.05 was considered significant.

## RESULTS

The study population consisted of 68 consecutive patients with confirmed non-high risk APE. Of these, 10 patients were excluded due to late presentation, more than seven days after symptom onset, or due to previous chronic respiratory or cardiac diseases. In all, 58 patients were included in the study, of which most were males of older age who presented within 24 hours of symptom onset. Patient characteristics and echocardiographic measurements are presented in table no. 1.

All patients were treated according to guideline recommendations with initial parenteral anticoagulation with dose-adjusted low molecular weight or unfractionated heparin, followed by oral anticoagulation with INR adjusted vitamin K antagonists or non-vitamin K oral anticoagulants, along with other recommended therapies.

**Table no. 1. Patient characteristics**

| Characteristic, unit | Value |
|---|---|
| Age, years | 67 ± 13 |
| Sex | |
| Male, pts. | 30 (52%) |
| Female, pts. | 28 (48%) |
| Time from symptom onset | |
| Mean, hours | 45 ± 46 |
| 0-24 hours, pts. | 33 (57%) |
| 1-3 days, pts. | 18 (31%) |
| 3-7 days, pts. | 7 (12%) |
| Systolic Blood Pressure, mmHg | 129 ± 27 |
| RV end-diastolic diameter, mm | 37 ± 8 |
| LV end-diastolic diameter, mm | 46 ± 7 |
| RV/LV end-diastolic diameter ratio | 0.84 ± 0.26 |
| PAT, msec | 82 ± 18 |
| TAPSE, mm | 15 ± 5 |
| sPAP, mmHg | 53 ± 20 |
| McConnell's sign present, pts. | 14 (24%) |
| RVOT end-diastolic diameter, mm | 39 ± 5 |
| RVOT end-systolic diameter, mm | 30 ± 5 |
| RVOT fractional shortening, % | 23 ± 7 |

*Values are expressed as number of patients (percent) or mean ± SD, RV right ventricle, LV left ventricle, PAT pulmonary ejection acceleration time, TAPSE tricuspid annulus plane systolic excursion, sPAP systolic pulmonary artery pressure, RVOT right ventricular outflow tract*

The composite endpoint of 30-day APE-related mortality and the occurrence of APE recurrence, ischemic stroke or acute myocardial infarction occurred in 6 patients (10.3%). There were four deaths recorded, of which three in the first 72 hours and one after 21 days, and two ischemic strokes. The patients in the composite endpoint group were significantly older than the patients who did not experience such an event ($p<0.001$), but no statistically significant difference in regards to sex ($p=0.631$), systolic blood pressure ($p=0.947$) and time since symptom onset ($p=0.324$) could be found (table no. 2).

Almost all of the echocardiographic measurements of RV morphology and function indicated an increased RVD in the composite endpoint group: increased RV end-diastolic diameter and sPAP and decreased PAT and TAPSE. However, the differences in these measured parameters did not reach statistical significance, except for the presence of McConnell's sign which registered a significantly higher incidence in the composite endpoint group ($p=0.027$). The RVOT end-diastolic and end-systolic diameters also showed slight increases in the composite endpoint group, along with a reduction in RVOT fractional shortening, none of which were significant (table no. 2).

Receiver-operating characteristic (ROC) curve analysis showed the area under the curve (AUC) for RVOT fractional shortening in predicting the composite endpoint was

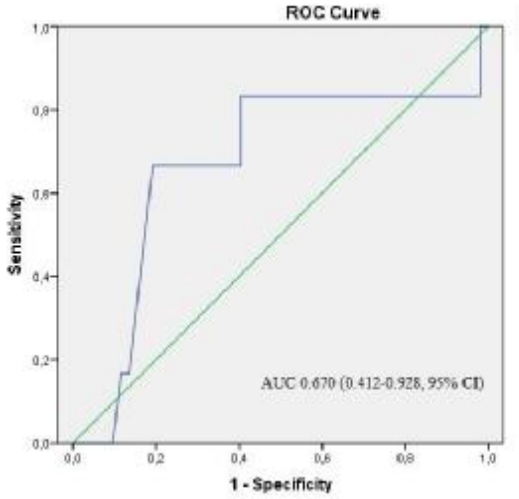
0.670 (0.412-0.928, 95% CI) but with no statistical significance (p=0.176) (figure no. 2). The same analysis for TAPSE showed an AUC of 0.744 (0.537-0.950, 95% CI), but with a non-significant p value (p=0.052) (figure no. 3).

**Table no. 2. Correlations with the composite endpoint**

| Characteristic, unit | Non-endpoint patients (n = 52) | Endpoint patients (n = 6) | p value |
|---|---|---|---|
| Male/Female, pts. | 27/25 | 3/3 | 0.631 |
| Age, years | 66 ± 14 | 77 ± 4 | <0.001 |
| Time from symptom onset, hours | 46 ± 48 | 31 ± 33 | 0.324 |
| Systolic Blood Pressure, mmHg | 128 ± 28 | 127 ± 20 | 0.947 |
| RV end-diastolic diameter, mm | 37 (22-56) | 38 (34-46) | 0.443 |
| LV end-diastolic diameter, mm | 46 ± 7 | 49 ± 10 | 0.372 |
| RV/LV end-diastolic diameter ratio | 0.83 (0.44-1.66) | 0.82 (0.66-1.30) | 0.878 |
| PAT, msec | 82 ± 18 | 74 ± 12 | 0.533 |
| TAPSE, mm | 16 ± 5 | 12 ± 4 | 0.063 |
| sPAP, mmHg | 53 (15-120) | 59 (45-100) | 0.372 |
| McConnell's sign present, pts. | 10 (19%) | 4 (66%) | 0.027 |
| RVOT end-diastolic diameter, mm | 39 ±5 | 40 ± 3 | 0.505 |
| RVOT end-systolic diameter, mm | 30 ± 5 | 32 ± 5 | 0.181 |
| RVOT fractional shortening, % | 24 ± 7 | 19 ± 8 | 0.148 |

*Values are expressed as mean ± SD or median (minimum-maximum range), RV right ventricle, LV left ventricle, PAT pulmonary ejection acceleration time, TAPSE tricuspid annulus plane systolic excursion, sPAP systolic pulmonary artery pressure, RVOT right ventricular outflow tract*

**Figure no. 2. ROC curve for RVOT fractional shortening**



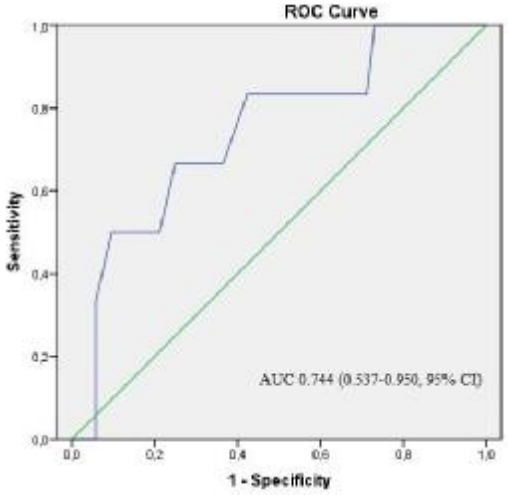

The analysis of correlation between RVOT fractional shortening and other parameters of RV function revealed a strong significant correlation coefficient with TAPSE (r=0.91), a weak significant correlation with RV end-diastolic diameter r=-0.48), RV/LV end-diastolic diameter ratio (r=-0.45), PAT (r=0.49) and sPAP (r=-0.24) and no significant correlation with LV end-diastolic diameter (table no. 3). Also, the presence of McConnell's sign was significantly associated with a reduced RVOT fractional shortening (20% vs. 24%, p=0.043).

**Table no. 3. RVOT fractional shortening correlations with other echocardiographical right ventricular dysfunction parameters**

| Parameter | Correlation coefficient r | p value (significant if p<0.01) |
|---|---|---|
| RV end-diastolic diameter | -0.48 | 0.000 |
| LV end-diastolic diameter | 0.11 | 0.433 |
| RV/LV end-diastolic diameter ratio | -0.45 | 0.000 |
| PAT | 0.49 | 0.005 |
| TAPSE | 0.91 | 0.000 |
| sPAP | -0.24 | 0.000 |

*RV right ventricle, LV left ventricle, PAT pulmonary ejection acceleration time, TAPSE tricuspid annulus plane systolic excursion, sPAP systolic pulmonary artery pressure, RVOT right ventricular outflow tract*

**Figure no. 3. ROC curve for TAPSE**




## DISCUSSIONS

High risk patients with APE, who present with hypotension and shock, have a 30-day mortality risk in excess of 15% (2) and represent a clinical emergency which warrants aggressive therapy. The non-high risk APE patients, with an estimated 30-day mortality risk of 1-15% (2), represent a heterogeneous group that include patients with a benign clinical course and also patients with an increased mortality risk. Recent guidelines have focused attention on this group of patients and have tried to further define the mortality risk by dividing it into a low-intermediate and a high-intermediate risk class with the goal of identifying patients at increased early mortality and adverse events rates, who require more aggressive therapy in order to reduce the risk.

The RV has a complex geometry with two main chambers situated in different planes (10) and prominent trabeculations that make endocardial delineation difficult. Therefore, there is no accurate geometric model to allow calculation of RV ejection fraction (10) and it is difficult for any one echocardiographic parameter to accurately represent global RV function.(11) In addition, some measurements of the RV have proven to be load dependent and inaccurate.(11) TAPSE is an indicator of longitudinal function, RV/LV ratio is indicative of RV morphology and McConnell's sign represents regional dysfunction of the free wall of the RV.

Right ventricular dysfunction, as assessed by echocardiography, is an important predictor of increased short-term mortality in patients with non-high risk APE (6,12,13),

with an incidence between 25 and 55% (14) and a reported odds ratio of predicting death of 2.36.(16) APE patients with normal echocardiographic findings have a reported mortality of lower than 1%.(14) Echocardiographic signs of RVD can be caused by concomitant pulmonary or cardiac diseases which can produce modifications similar to ones produced by acute conditions. For this reason we excluded any patients with previous significant respiratory or cardiac comorbidities. Also, RVD parameters are subject to modifications from the acute to the chronic phase of pulmonary diseases (15) and, because of this reason, patients with a time from symptom onset of more than seven days were considered to have passed the acute phase adaptations of the RV due to APE and were excluded from the study. Echocardiography was performed as soon as possible after admission, in the purpose of identifying the most precocious RV modifications which could predict early mortality.

Recently, due to advances in early diagnosis and management, 30-day APE-related mortality in normotensive, non-high risk patients was reported, in a meta-analysis, on average as 5% (16), and as low as 3.4% in a large study by Pruszczyk et al.(17) Our study recorded a 30-day APE-related mortality of 6.8% (4 patients) and a composite endpoint rate of 10.3% (6 patients). This low event rate makes it difficult for any one study to identify predictive factors for increased mortality risk. In our population, the only factor significantly associated with the composite endpoint was increased age, which is a proven predictive factor for increased 30-day mortality in APE patients and is included in the most commonly used mortality risk calculators.(3,4)

Our study population, of non-high risk APE patients, was still heterogeneous in respect to APE severity and the presence of RVD. The mean RV morphology parameters registered were modified in comparison with the normal reference values endorsed by the current recommendations for chamber measurements.(7) The mean RV diastolic diameter in the study population was 37 mm, greater than the upper normal limit of 35 mm and the mean RVOT diastolic diameter was 39 mm, greater than the upper normal limit of 35 mm. Also, the functional RV parameters were modified with a reduced mean PAT of 82 msec and an increased mean sPAP of 53 mmHg. Thereby, the majority of both study groups presented with right ventricular dysfunction and, as such, although there were slight differences in the majority of the RVD parameters between the composite endpoint group and the non-endpoint group, none could be proven significant, except the presence of McConnell's sign which had a significantly higher prevalence in the composite endpoint group, suggesting regional wall motion disturbances could be a predictive factor for increased mortality and adverse events.

RVOT fractional shortening has been identified as a simple and important measure of RVOT systolic function and of overall RV systolic function.(18) Moreover, in a study by Yamaguchi et al., reduced RVOT fractional shortening was associated with the presence of reduced left ventricular ejection fraction and showed a predictive value for major cardiovascular events in patients with left ventricular systolic dysfunction.(19) In our study population, the mean RVOT fractional shortening was reduced in comparison with values observed in healthy controls (23% vs. 61%).(18) Also, the value was lower than those measured in patients with RVD in other previous studies, ranging from 26% to 37%.(18,19,20) The composite endpoint group showed a reduced mean RVOT fractional shortening in comparison with the non-endpoint group (19% vs. 24%), but the difference did not reach statistical significance so, although the lower values of RVOT fractional shortening were associated with an increased event ratio, a cut-off value of RVOT fractional shortening for predicting 30 day mortality and adverse events could not be identified.

A large study by Pruszczyk et al. (17) identified a reduced TAPSE as the most important predictor of adverse clinical outcomes in normotensive patients with APE, in comparison with other known markers of RVD, by measuring the AUC of the ROC analysis as the highest compared to the other markers. Although recent recommendations for echocardiographic measurements in adults indicate a TAPSE lower abnormality threshold of 17 mm (7), the study found that in the setting of non-high risk APE, a TAPSE of ≤15 mm had a HR of 14.48 in predicting adverse events(17), while another study by Lobo et al. found a TAPSE of ≤16 mm to have a HR of 2.5 in predicting APE related death.(21) In our population, the composite endpoint group had a mean TAPSE value of 12 mm, in concordance with the values identified as a marker of increased risk. Moreover, the AUC of the ROC analysis for TAPSE in our study was also the highest one, as in the study by Pruszczyk et al., though not as high (0.67 vs. 0.90) and not statistically significant. From these results we can infer that RVOT fractional shortening has a predictive value for increased mortality and adverse events risk in patients with non-high risk APE, and a larger study population could allow us to better evaluate that risk.

While the AUC of the ROC analysis for RVOT fractional shortening did not achieve statistical significance in our study population, the variable dependence analysis showed a strong significant correlation between RVOT fractional shortening and TAPSE (r=0.91). This indicates that a reduced RVOT fractional shortening is associated with a reduced TAPSE, a RVD marker with high predictive value for 30-day mortality.(17) The strong prognostic power of TAPSE has been linked to its simplicity of measurement and good reproducibility (22), characteristics which are shared by RVOT fractional shortening.(18)

The main study limitation was the low number of endpoints observed in the study population. To our knowledge, this is the first study to assess the predictive value of RVOT fractional shortening in the setting of APE, and, as such, future studies are needed to validate the mortality risk predictive value of this echocardiographic measurement. Therefore, our conclusions should be validated in a larger population, or in a larger, multi-centric study.

## CONCLUSIONS

Right ventricular outflow tract fractional shortening is an applicable measure of right ventricular dysfunction in patients with non-high risk acute pulmonary embolism. Reduced RVOT fractional shortening is a marker of increased risk of 30-day mortality or adverse events in non-high risk APE patients. RVOT fractional shortening can be used for risk evaluation, in combination with other known echocardiographic markers of right ventricular dysfunction, in the initial evaluation of patients with non-high risk APE.

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