Importance of heart ultrasound in pulmonary embolism diagnostic algorithm

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IMPORTANCE OF HEART ULTRASOUND IN PULMONARY EMBOLISM DIAGNOSTIC ALGORITHM

Graduate Thesis

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List of Abbreviations

BNP - brain natriuretic peptide

DMI - Doppler myocardial imaging

DVT - deep vein thrombosis

PE - pulmonary embolism

RV- right ventricle

TIPG - tricuspid insufficiency pressure gradient

VTE - venous thromboembolism

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Summary

Title: Importance of Heart Ultrasound in Pulmonary Embolism Diagnostic Algorithm
Name: Ivan Beluškov

Pulmonary embolism (PE) is according to the Guidelines on the diagnosis and management of acute pulmonary embolism a common cardiovascular emergency. By occluding the pulmonary arterial bed it may lead to acute life-threatening but potentially reversible right ventricular failure. PE is a difficult diagnosis that may be missed because of non-specific clinical presentation. PE is not a disease on its own rather it is a complication of other systemic hypercoagulability states; Such as deep vein thrombosis (DVT), pregnancy, smoking, cancer, hormone replacement therapy, contraception, genetic factors and many more. Due to this fact and due to the fact that it usually presents with nonspecific symptoms, PE diagnosis is sometimes very difficult. Acute PE is usually a consequence of primarily haemodynamic problems and can be seen in when as much as 30-50% of the pulmonary arterial bed is occluded by thrombus. Symptoms of PE are usually nonspecific and this makes the diagnosis of PE a hard one to make even in this day and age. The clinical presentation has a high importance in order to interpret the diagnostic test results and to plan the diagnostic strategies. In as much as 90 % of cases suspicion on PE is raised because of the presence of symptoms such as dyspnoea, chest pain and syncope, either singly or in combination. From all the symptoms pleuritic chest pain, whether or not combined with dyspnoea, is one of the most frequent presentations of PE. The levels of the D-dimer, compression ultrasonography and computed tomographic venography, as well as computed tomography which if it shows that a thrombus is shown up to the segmental level than it can be taken as good evidence for PE, are other diagnostic tools that can be used to diagnose DVT and PE, "Ventilation–perfusion scintigraphy is a robust and well-established diagnostic test for suspected PE. Pulmonary angiography is one of the more reliable diagnostic procedures of PE but its invasiveness limits its use to times when noninvasive tests don't yield satisfactory results. We today have available tools to guide us and support our decision making; we have tools to classify the stability of our patients, and with diagnostic tools such as heart ultrasound, which is most important for patient suffering from high-risk PE, we can do bedside evaluation of the patient and appropriate treatment would be given to the patient on time. The availability of diagnostic tools as well as the availability of different treatment options will hopefully in the future give us the ability to even better understand this complication and treat the underlying cause of it.

Keywords: Heart Ultrasound, Pulmonary Embolism, Algorithm, Diagnosis, Venous Thromboembolism, Hypercoagulability.

Pulmonary Embolism

What is pulmonary embolism? Pulmonary embolism (PE) is according to the Guidelines on the diagnosis and management of acute pulmonary embolism a common cardiovascular emergency. By occluding the pulmonary arterial bed it may lead to acute life-threatening but potentially reversible right ventricular failure. PE is a difficult diagnosis that may be missed because of non-specific clinical presentation. [1] PE is not a disease on its own rather it is a complication of other systemic hypercoagulability states; Such as deep vein thrombosis (DVT), pregnancy, smoking, cancer, hormone replacement therapy, contraception, genetic factors and many more. Due to this fact and due to the fact that it usually presents with nonspecific symptoms, PE diagnosis is sometimes very difficult.

Guidelines on the diagnosis and management of acute pulmonary embolism

Table 3
Predisposing factors for venous thromboembolism

Predisposing factor	Patient-related	Setting-related
Strong predisposing factors (odds ratio >10)		
Fracture (hip or leg)		✓
Hip or knee replacement		✓
Major general surgery		✓
Major trauma		✓
Spinal cord injury		✓
Moderate predisposing factors (odds ratio 2–9)		
Arthroscopic knee surgery		✓
Central venous lines		✓
Chemotherapy		✓
Chronic heart or respiratory failure	✓	
Hormone replacement therapy	✓	
Malignancy	✓	
Oral contraceptive therapy	✓	
Paralytic stroke	✓	
Pregnancy/postpartum		✓
Previous VTE	✓	
Thrombophilia	✓	
Weak predisposing factors (odds ratio <2)		
Bed rest >3 days		✓
Immobility due to sitting (e.g. prolonged car or air travel)		✓
Increasing age	✓	
Laparoscopic surgery (e.g. cholecystectomy)		✓
Obesity	✓	
Pregnancy/antepartum	✓	
Varicose veins	✓	

Data are modified from reference 2. This article was published in *Circulation*, Vol. 107, Anderson FA Jr, Spencer FA. Risk factors for venous thromboembolism, I-9–I-16. © (2003) American Heart Association, Inc.

Figure 1:Predisposing factors for venous thromboembolism,
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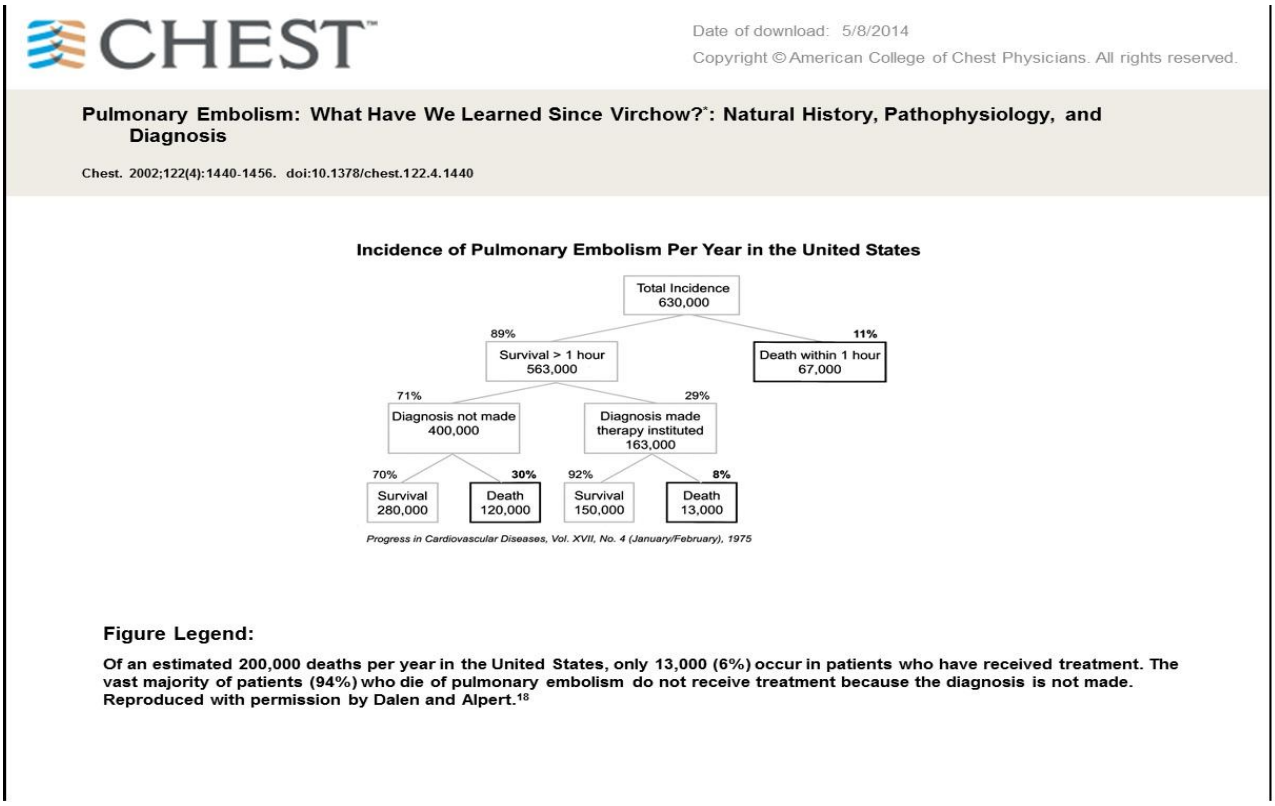
Dalen JE believes that we will discover additional genetic defects that lead to hypercoagulability, such that the majority of patients with venous thromboembolism (VTE) will be found to have a hypercoagulable state.[2] According to some data even up to 50% of patient that have symptomatic DVT have ventilation perfusion scan that suggests high chance of having PE.[3] It is difficult to estimate the proportion of cases of PE that are undiagnosed, and it is due to this that the true incidence of PE is underreported in my opinion. Undiagnosed and untreated PE can have dire consequences for any patient, and for the doctor that examines this patient. It can be as much as 10% of patient that have symptomatic PE will die in the first 1 hour. [3][4][5] And on the other hand in Barritt and Jordan's trial , 26% (5 of 19) of untreated patients with clinically diagnosed PE (severe end of the spectrum) died of PE during a follow-up period of ≈2 weeks, and another 26% of patients experienced nonfatal recurrences.[3][6]



Pulmonary Embolism: What Have We Learned Since Virchow?: Natural History, Pathophysiology, and Diagnosis

Chest. 2002;122(4):1440-1456. doi:10.1378/chest.122.4.1440

Incidence of Pulmonary Embolism Per Year in the United States

Progress in Cardiovascular Diseases, Vol. XVII, No. 4 (January/February), 1975

Figure Legend:

Of an estimated 200,000 deaths per year in the United States, only 13,000 (6%) occur in patients who have received treatment. The vast majority of patients (94%) who die of pulmonary embolism do not receive treatment because the diagnosis is not made. Reproduced with permission by Dalen and Alpert.[18]

Figure 2: Incidence of PE per year in United States,

With permission of "Copyright © 2002, American College of Chest Physicians"

Guidelines on the Diagnosis of Pulmonary Embolism

Acute PE is usually a consequence of primarily haemodynamic problems and can be seen in when as much as 30-50% of the pulmonary arterial bed is occluded by thrombus. [1][7] In the Guidelines on the diagnosis and management of acute pulmonary embolism it also says that non-thrombotic pulmonary emboli are rare and have different pathophysiological consequences and clinical characteristics. [1] As it was said previously symptoms of PE are usually nonspecific and this makes the diagnosis of PE a hard one to make even in this day and age. One of the first things that need to be considered when examining a patient suspected on having PE is to go over the clinical presentation. The clinical presentation has a high importance in order to interpret the diagnostic test results and to plan the diagnostic strategies. In as much as 90 % of cases suspicion on PE is raised because of the presence of symptoms such as dyspnoea, chest pain and syncope, either singly or in combination. From all the symptoms pleuritic chest pain, whether or not combined with dyspnoea, is one of the most frequent presentations of PE. [1]

Guidelines on the diagnosis and management of acute pulmonary embolism

Table 6
Prevalence of symptoms and signs in patients with suspected PE according to final diagnosis

	PE confirmed (*n* = 219)	PE excluded (*n* = 546)
Symptoms		
Dyspnoea	80%	59%
Chest pain (pleuritic)	52%	43%
Chest pain (substernal)	12%	8%
Cough	20%	25%
Haemoptysis	11%	7%
Syncope	19%	11%
Signs		
Tachypnoea (≥20/min)	70%	68%
Tachycardia (>100/min)	26%	23%
Signs of DVT	15%	10%
Fever (>38.5°C)	7%	17%
Cyanosis	11%	9%

Data are form references 53 and 55.

DVT = deep vein thrombosis.

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Figure 3:Prevalence of symptoms and signs in patients suspected PE according to final diagnosis "With permission of Oxford University Press (UK) © European Society of Cardiology, www.escardio.org/guidelines."Cardiology, www.escardio.org/guidelines."

The clinical presentation of a patient can neither confirm for us of exclude the diagnosis of PE, but it can lead us to proceed to using other diagnostic tools to make a definite diagnosis.

Guidelines on the diagnosis and management of acute pulmonary embolism

Table 7
Clinical prediction rules for PE: the Wells score and the revised Geneva score

Revised Geneva score[64]		Wells score[65]	
Variable	**Points**	**Variable**	**Points**
Predisposing factors		Predisposing factors	
Age >65 years	+1		
Previous DVT or PE	+3	Previous DVT or PE	+1.5
Surgery or fracture within 1 month	+2	Recent surgery or immobilization	+1.5
Active malignancy	+2	Cancer	+1
Symptoms		Symptoms	
Unilateral lower limb pain	+3		
Haemoptysis	+2	Haemoptysis	+1
Clinical signs		Clinical signs	
Heart rate		Heart rate	
75–94 beats/min	+3	>100 beats/min	+1.5
≥95 beats/min	+5		
Pain on lower limb deep vein at palpation and unilateral oedema	+4	Clinical signs of DVT	+3
		Clinical judgement	
		Alternative diagnosis less likely than PE	+3
Clinical probability	**Total**	**Clinical probability (3 levels)**	**Total**
Low	0–3	Low	0–1
Intermediate	4–10	Intermediate	2–6
High	≥11	High	≥7
		Clinical probability (2 levels)	
		PE unlikely	0–4
		PE likely	>4

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Figure 4: Clinical predicted rules for PE; "With permission of Oxford University Press (UK) © European Society of Cardiology, www.escardio.org/guidelines."Cardiology, www.escardio.org/guidelines."

Another diagnostic test that can be used to increase the probability of PE is the plasma D-dimer levels. The levels of the D-dimer will be elevated if there is acute clot formed. But because this is not the only reason why the level of D-dimers can be elevated we cannot use this test to confirm PE, but rather we can use it to either to increase or decrease the probability of PE depending on the D-dimer levels. Because negative D-dimer result can be said that safely excludes PE. [1]

Compression ultrasonography and computed tomographic venography are other diagnostic tools that can be used to diagnose DVT and PE, as well as computed tomography which if it shows that a thrombus is shown up to the segmental level than it can be taken as good evidence for

PE.[1] “Ventilation–perfusion scintigraphy is a robust and well-established diagnostic test for suspected PE.”[1] It is a safe test with little or no side effects. This test is one of the better ones because normal scan can safely exclude where as high-probability ventilation–perfusion scan establishes the diagnosis of PE with high probability. [1] Pulmonary angiography is one of the more reliable diagnostic procedures of PE but its invasiveness limits its use to times when noninvasive tests don’t yield satisfactory results. [1]

Guidelines on the diagnosis and management of acute pulmonary embolism

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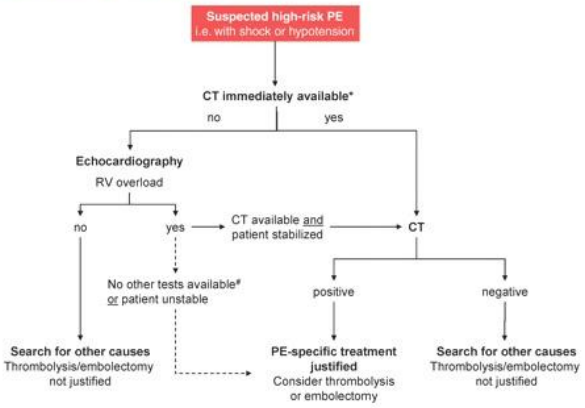


Figure 1
Proposed diagnostic algorithm for patients with suspected high-risk PE, i.e. presenting with shock or hypotension. *CT is considered not immediately available also if the critical condition of a patient allows only bedside diagnostic tests.
#Transoesophageal echocardiography may detect thrombi in the pulmonary arteries in a significant proportion of patients with RV overload and PE that is ultimately confirmed by spiral CT; confirmation of DVT with bedside CUS might also help in decision-making.

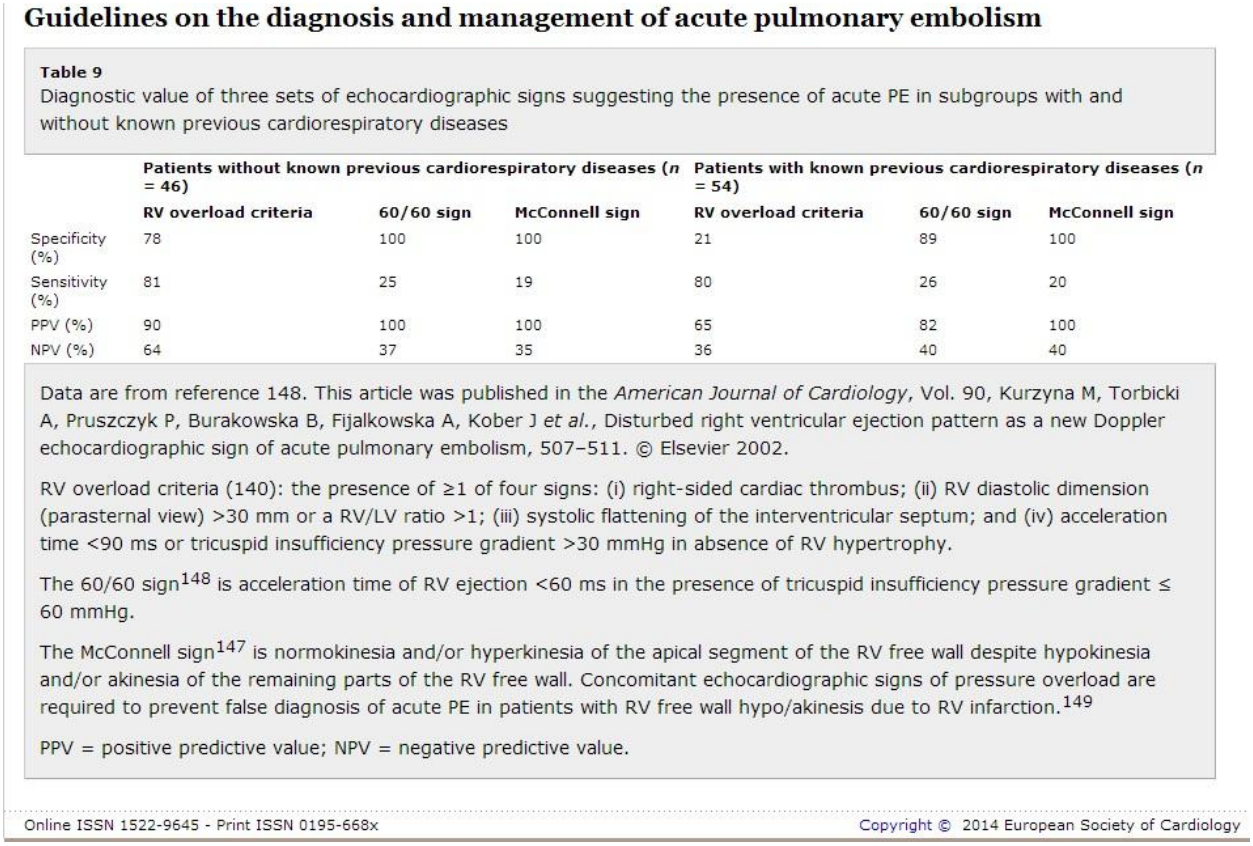
Figure 5: Proposed diagnostic algorithm for patients with suspected high risk PE;"With permission of Oxford University Press (UK) © European Society of Cardiology, www.escardio.org/guidelines."Cardiology, www.escardio.org/guidelines."

So far I have discussed some of the most commonly used and most reliable tests in order to diagnose PE. The only diagnostic tool that I have not discussed so far is heart ultrasound, which is the main topic for this paper. In the upcoming part of this paper I will shortly try to emphasize why heart ultrasound is important and where does it find its place in the diagnostic algorithm of PE.

Heart Ultrasound as a Diagnostic Tool for Pulmonary Embolism

Acording to Ruxandra Jurcut: “For decades, the right ventricle (RV) has been considered ‘dispensable’ for cardiac function and consequently ignored. Echocardiography, being non-invasive, widely available, relatively inexpensive, and having no side effects, is the modality of choice for the assessment of morphology and function of the RV in clinical practice. Recent developments have provided several new methods for analysing the RV, each having advantages and disadvantages. Doppler myocardial imaging (DMI), speckle tracking, or 3D echocardiography (3D Echo) are some of the techniques that may now add to a better understanding of RV function.”[8] On the other hand we have A. Torbicki who says that the place of echocardiography in diagnostic assessment of patients with suspected pulmonary embolism is neither strong nor definitely established. But if we have a patient that is acutely unstable echocardiography may be highly suggestive of PE. [9] This approach had been formally accepted by a multidisciplinary ESC Task Force and published in the ESC Guidelines in 2000.[9][10] In most of the cases heart ultrasound gives indirect signs of PE. There are some signs and among them the McConnell's (defined as right ventricular (RV) free wall hypokinesis in the presence of normal RV apical contractility) and “60/60 sign” (acceleration time below 60ms in the presence of tricuspid insufficiency pressure gradient (TIPG) above 30 but below 60mmHg) are the most reliable ones and combining them together gives more reliable data on which we can diagnose PE. At least 25% of patient with PE have RV dilatation.[1] The criteria for heart ultrasound in the diagnosis of PE have been different in different trials but are usually based on the RV dimensions and the tricuspid jet velocity of insufficiency. The sensitivity of it was 60-70% which means that a negative result cannot exclude PE. [1] Even thou heart ultrasound is not routinely used as a diagnostic test its use is really helpful in finding patients

with PE that have poor prognosis. It can also be used for rapid and accurate risk assessment. And based on the severity of RV hypokinesis, persistent pulmonary hypertension, a patent foramen ovale, and free-floating right-heart thrombus are echocardiographic markers that identify the risk of a patient for recurrent thromboembolism. [11] Bedside echocardiography is really useful in emergency decision making. Because if we have a patient with hypotension and shock the absence of RV overload with high probability excludes PE.[1] And it lies in this point the importance of heart ultrasound, because according to this prognosis the treatment of PE is guided and further prognostic stratification is done based on the echocardiography imaging results.



Guidelines on the diagnosis and management of acute pulmonary embolism

Table 9
Diagnostic value of three sets of echocardiographic signs suggesting the presence of acute PE in subgroups with and without known previous cardiorespiratory diseases

	Patients without known previous cardiorespiratory diseases (*n* = 46)			Patients with known previous cardiorespiratory diseases (*n* = 54)		
	RV overload criteria	**60/60 sign**	**McConnell sign**	**RV overload criteria**	**60/60 sign**	**McConnell sign**
Specificity (%)	78	100	100	21	89	100
Sensitivity (%)	81	25	19	80	26	20
PPV (%)	90	100	100	65	82	100
NPV (%)	64	37	35	36	40	40

Data are from reference 148. This article was published in the *American Journal of Cardiology*, Vol. 90, Kurzyna M, Torbicki A, Pruszczyk P, Burakowska B, Fijalkowska A, Kober J *et al.*, Disturbed right ventricular ejection pattern as a new Doppler echocardiographic sign of acute pulmonary embolism, 507–511. © Elsevier 2002.

RV overload criteria (140): the presence of ≥1 of four signs: (i) right-sided cardiac thrombus; (ii) RV diastolic dimension (parasternal view) >30 mm or a RV/LV ratio >1; (iii) systolic flattening of the interventricular septum; and (iv) acceleration time <90 ms or tricuspid insufficiency pressure gradient >30 mmHg in absence of RV hypertrophy.

The 60/60 sign[148] is acceleration time of RV ejection <60 ms in the presence of tricuspid insufficiency pressure gradient ≤ 60 mmHg.

The McConnell sign[147] is normokinesia and/or hyperkinesia of the apical segment of the RV free wall despite hypokinesia and/or akinesia of the remaining parts of the RV free wall. Concomitant echocardiographic signs of pressure overload are required to prevent false diagnosis of acute PE in patients with RV free wall hypo/akinesis due to RV infarction.[149]

PPV = positive predictive value; NPV = negative predictive value.

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Figure 6: Echocardiographic signs, "With permission of Oxford University Press (UK) © European Society of Cardiology, www.escardio.org/guidelines."Cardiology, www.escardio.org/guidelines."

It is because of this that in the figure below that summarizes the guidelines on the diagnosis and management of acute pulmonary embolism in patients suspected of non-high risk PE [1], we don't see heart ultrasound as a diagnostic tool. Whereas as we saw in the figure summarizing the patients suspected of high risk PE heart ultrasound is after CT, which in this case is not immediately available due to the need of bedside diagnosis, most reliable and available for bedside diagnosis and fast assessment. This makes heart ultrasound indispensable tool in the algorithm for diagnosis and management of PE.

Guidelines on the diagnosis and management of acute pulmonary embolism

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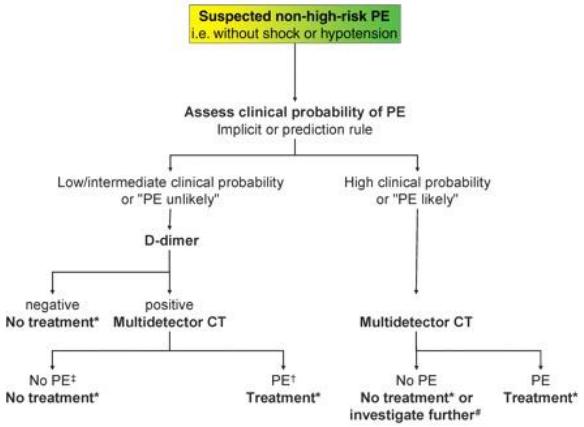

Figure 2

Proposed diagnostic algorithm for patients with suspected non-high-risk PE (i.e. without shock and hypotension). Two alternative classification schemes may be used to assess clinical probability: a three-level scheme (clinical probability low, intermediate or high) or a two-level scheme (PE unlikely or PE likely). When using a moderately sensitive assay, D-dimer measurement should be restricted to patients with a low clinical probability or a 'PE unlikely' classification, while highly sensitive assays may be used in patients with a low or intermediate clinical probability of PE. Plasma D-dimer measurement is of limited use in suspected PE occurring in hospitalized patients. *Anticoagulant treatment for PE. †CT is considered diagnostic of PE if the most proximal thrombus is at least segmental. ‡If single-detector CT is negative, a negative proximal lower limb venous ultrasonography is required in order to safely exclude PE. #If multidetector CT is negative in patients with high clinical probability, further investigation may be considered before withholding PE-specific treatment (see text). PE, pulmonary embolism.

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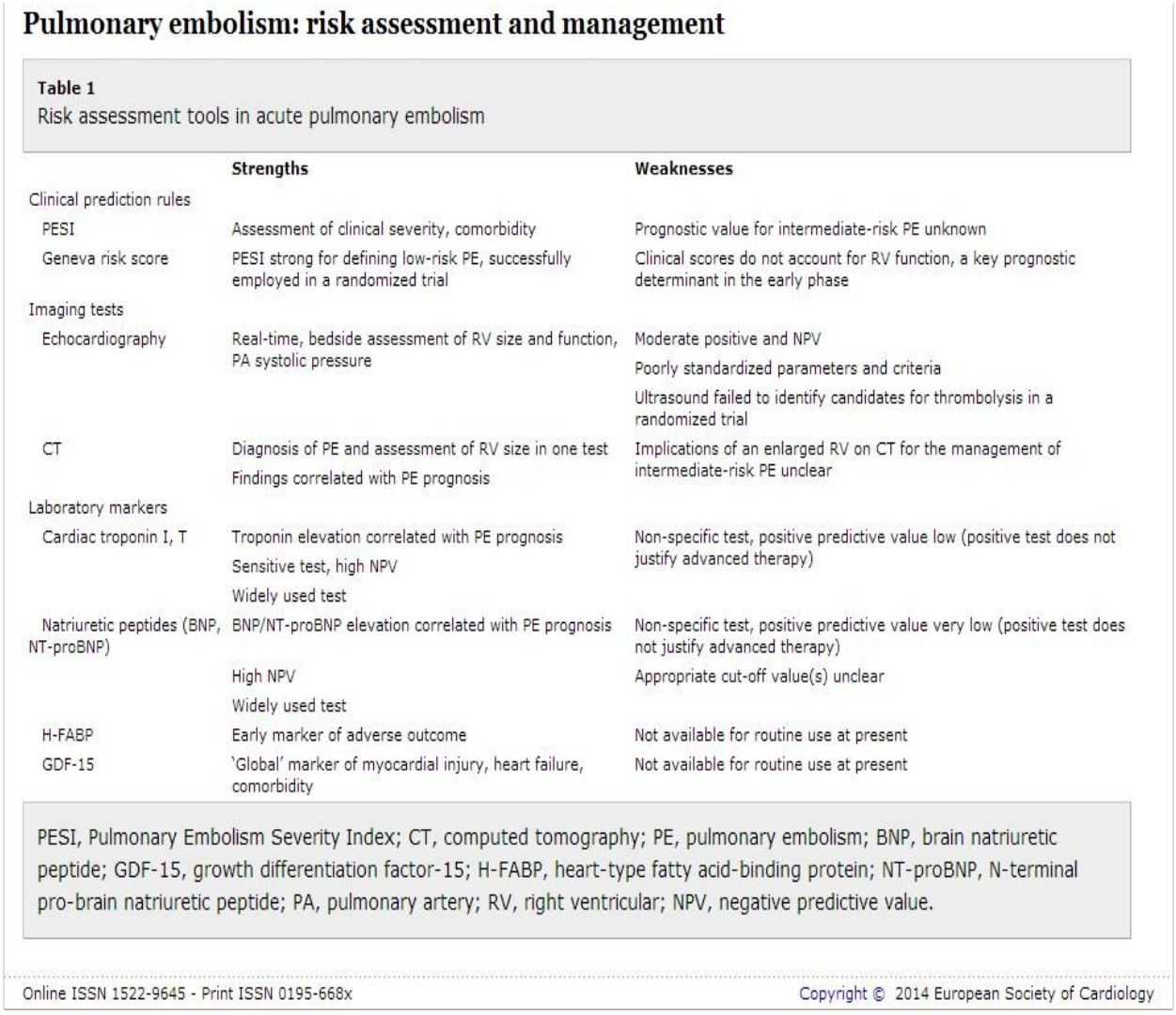
Figure 7: Proposed diagnostic algorithm for patients with suspected non high risk PE "With permission of Oxford University Press (UK) © European Society of Cardiology, www.escardio.org/guidelines."Cardiology, www.escardio.org/guidelines."

Prognostic Assessment of the Patient with Pulmonary Embolism

Acute PA caries huge burden on the health and the survival of the patient as well as on the health care system. Because of this we need to asses every patient fast and with great care in order to benefit both the patient and system from unwanted complications and difficult recovery, and to ensure the highest quality of care. [12] One of the major issues in the assessment of patients with PE is to accurately assess the risk in order to prevent the early death of the patient. We can do this as we previously said with heart ultrasound assessment of the RV, in order to determine its dysfunction and overload. [12][13] [14] In their critical review paper Stavros Konstantinides and Samuel Z. Goldhaber explain and define the initial prognostic assessment as: "The definition of high-risk (European classsification) or massive (North American classification) PE is usually straightforward and relies on the presence of clinically overt RV failure which results in haemodynamic compromise. This condition, which is encountered in <5% of all patients presenting with acute PE, constitutes a medical emergency, since it is associated with at least a 15% risk of in-hospital death, particularly during the first hours after admission."[12] Heart ultrasound can detect the morphological changes and function of the RV. And Stavros Konstantinides and Samuel Z. Goldhaber have found registries of cohort studies that make the connection between heart ultrasound parameters of RV dysfunction and poor in-hospital outcome.[12] But this is only true for haemodynamically unstable patients. Additional clinical signs are needed to indicate severe PE in haemodynamically stable patients. Circulating biochemical markers and especially circulating natriuretic peptides are additional tools for assessing these haemodynamically stable patients and assessing and stratifying their risk. [12] Stavros Konstantinides and Samuel Z. Goldhaber also found that: "A meta-analysis of 13 studies found that 51% of 1132 patients with acute PE had elevated brain natriuretic peptide

(BNP) or N-terminal (NT)-proBNP concentrations; these were associated with an increased risk of early death and a complicated in-hospital course."[12][15] Other diagnostic test such as levels of troponin I or T can be used based on meta-analysis of published studies, as well as Heart-type fatty acid-binding protein, Cardiac expression of growth-differentiation factor-15 which both appear to be promising in the evaluation of the prognosis of patients with non-high risk PE.[12] All of these tools and many more are summarized in the figures below, and combining these tools is as important in the assessment of the prognosis of patients with PE.[12]



Pulmonary embolism: risk assessment and management

Table 1
Risk assessment tools in acute pulmonary embolism

	Strengths	Weaknesses
Clinical prediction rules		
PESI	Assessment of clinical severity, comorbidity	Prognostic value for intermediate-risk PE unknown
Geneva risk score	PESI strong for defining low-risk PE, successfully employed in a randomized trial	Clinical scores do not account for RV function, a key prognostic determinant in the early phase
Imaging tests		
Echocardiography	Real-time, bedside assessment of RV size and function, PA systolic pressure	Moderate positive and NPV Poorly standardized parameters and criteria Ultrasound failed to identify candidates for thrombolysis in a randomized trial
CT	Diagnosis of PE and assessment of RV size in one test Findings correlated with PE prognosis	Implications of an enlarged RV on CT for the management of intermediate-risk PE unclear
Laboratory markers		
Cardiac troponin I, T	Troponin elevation correlated with PE prognosis Sensitive test, high NPV Widely used test	Non-specific test, positive predictive value low (positive test does not justify advanced therapy)
Natriuretic peptides (BNP, NT-proBNP)	BNP/NT-proBNP elevation correlated with PE prognosis High NPV Widely used test	Non-specific test, positive predictive value very low (positive test does not justify advanced therapy) Appropriate cut-off value(s) unclear
H-FABP	Early marker of adverse outcome	Not available for routine use at present
GDF-15	'Global' marker of myocardial injury, heart failure, comorbidity	Not available for routine use at present

PESI, Pulmonary Embolism Severity Index; CT, computed tomography; PE, pulmonary embolism; BNP, brain natriuretic peptide; GDF-15, growth differentiation factor-15; H-FABP, heart-type fatty acid-binding protein; NT-proBNP, N-terminal pro-brain natriuretic peptide; PA, pulmonary artery; RV, right ventricular; NPV, negative predictive value.

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Figure 8: Risk assessment tools in acute PE;"With permission of Oxford University Press (UK) © European Society of Cardiology. "

Pulmonary embolism: risk assessment and management

Table 2
Thrombolysis for pulmonary embolism

Agents and regimens	
Streptokinase[a]	
250 000 U as a loading dose over 30 min, followed by 100 000 U/h over 12–24 h	
Accelerated regimen: 1.5 million IU over 2 h[b]	
Urokinase[a,c]	
4400 U per kg of body weight as a loading dose over 10 min, followed by 4400 U/kg/h over 12–24 h	
Accelerated regimen: 3 million U over 2 h[b]	
Alteplase[a]	
100 mg over 2 h[d]	
Accelerated regimen: 0.6 mg/kg for 15 min	
Reteplase[a,e]	
Two bolus injections of 10 U 30 min apart	
Tenecteplase[f]	
30–50 mg bolus for 5–10 s adjusted for body weight	
<60 kg	30 mg
≥60 to <70 kg	35 mg
≥70 to <80 kg	40 mg
≥80 to <90 kg	45 mg
≥90 kg	50 mg

Contraindications

Absolute
History of haemorrhagic stroke or stroke of unknown origin
Ischaemic stroke in previous 6 months
Central nervous system neoplasms
Major trauma, surgery, or head injury in previous 3 weeks

Relative
Transient ischaemic attack in previous 6 months
Oral anticoagulation
Pregnancy or first postpartum week
Non-compressible puncture sites
Traumatic resuscitation
Refractory hypertension (systolic blood pressure >180 mmHg)
Advanced liver disease
Infective endocarditis
Active peptic ulcer

Adapted and modified from references 5 and 111.

[a]Unfractionated heparin should not be infused together with streptokinase or urokinase; it can be given during alteplase or reteplase administration. Low-molecular-weight heparins have not been tested in combination with thrombolysis in patients with pulmonary embolism.

[b]Short (2 h) infusion periods are generally recommended.[53]

[c]Urokinase is not available in the USA.

[d]FDA-approved regimen.

[e]Off-label use of reteplase.

[f]Off-label use of tenecteplase; this is the regimen recommended for acute myocardial infarction. A randomized pilot trial[112] found it to be safe and effective in non-high-risk pulmonary embolism.

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Figure 9: Thrombolysis for PE; "With permission of Oxford University Press (UK) © European Society of Cardiology."

Treatment

So far we have discussed the most important points in assessing the risk posed by PE, but we have not said much about how we should manage and what the treatment of these patients should be. After assessing and stratifying the risk of our patients there are many possibilities today how to treat these patients, and one of the most commonly used in acute settings is heparin anticoagulation. Heparin anticoagulation is used in different regiments and the current regiments are summarized in the figure below.[12] Besides Heparin anticoagulation we can also use Vitamin K antagonists and other new oral anticoagulants in the treatment of PE.

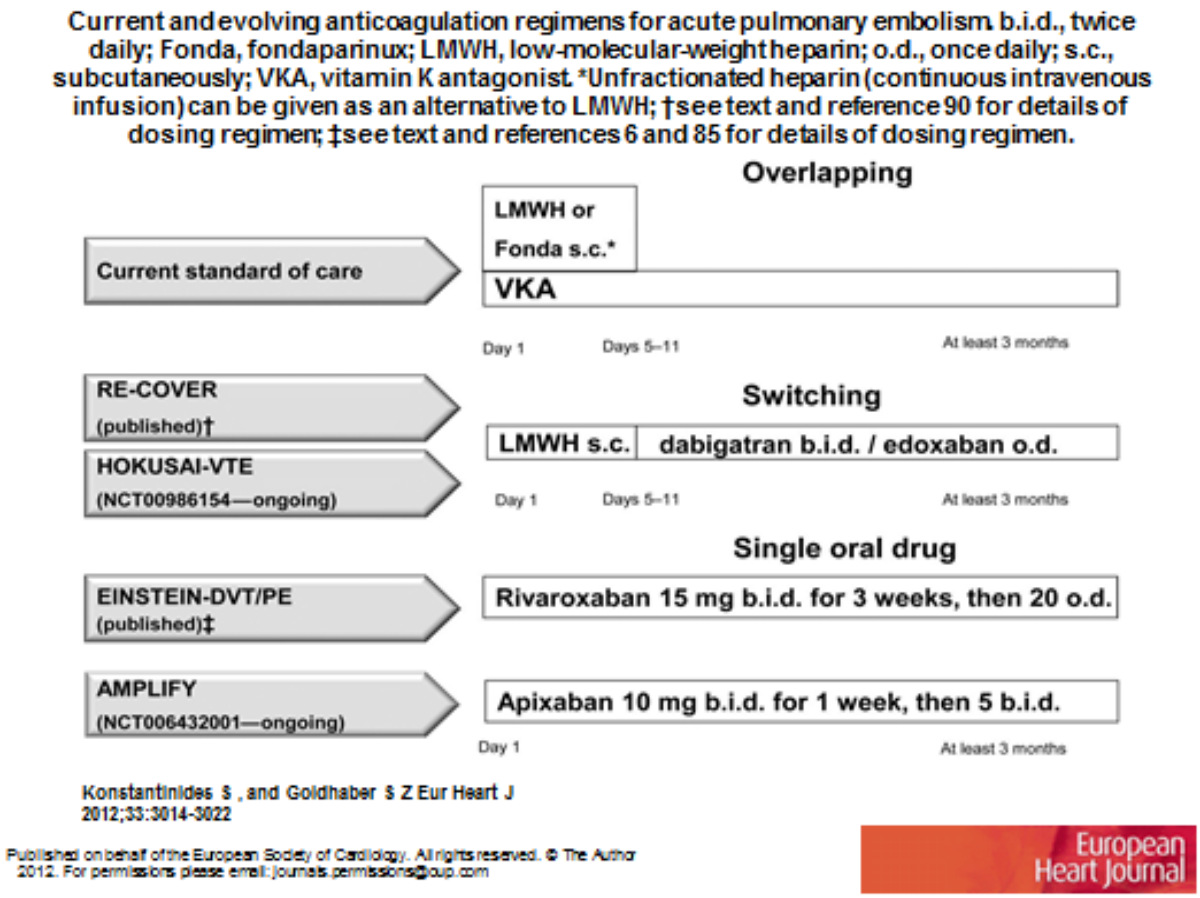

Figure 10: Anticoagulation regiment for acute PE; "With permission of Oxford University Press (UK) © European Society of Cardiology "

Other than heparin anticoagulation we have thrombolysis as a tool for treatment of PE, and it has been shown constantly that is effectively resolves the obstruction made by the thrombus and the hemodynamic functions.[12] Overall, >90% of patients with PE appear to respond favourably to thrombolysis as indicated by clinical and echocardiographic improvement within the first 36 h.[12][16]

We now have several thrombolysis regiments available to us, as seen in figure 9 of the previous section, and it has been shown that in studies that thrombolysis works best if treatment is initiated within 48 h of symptom onset, but thrombolysis can still be useful in patients who have had symptoms for 6–14 days.[12][17]

Other treatments available for PE are surgical or interventional treatment, but these treatment options for now should be considered and are recommended only in situations when there is absolute contraindication to thrombolysis or if thrombolysis treatment failed and the patient has high-risk PE. Inferior vena cava filters on the other hand are not per say treatment options rather a means of both primary and secondary prevention of PE in patient suffering fr1om hypercoagulable states, however there is not enough date to support their use and safety. [12]

Conclusion

Even thou the fatality of PE continues to decrease and today we have diagnostic tools that are more efficient and help us differentiate the severity of PE, we still need to improve our knowledge on PE in order to decrease the burden of PE on health and survival. [18] We today have available tools to guide us and support our decision making; we have tools to classify the stability of our patients, and with diagnostic tools such as heart ultrasound, which is most important for patient suffering from high-risk PE, we can do bedside evaluation of the patient and appropriate treatment would be given to the patient on time. It is my opinion that in the future heart ultrasound as a tool can be further improved, the interpretations and understandings of heart ultrasound signs can be further improved, and as such this tool it can be used not only in the management of high-risk PE patients, but also in low and intermediate risk PE. It is John Locke that said: “The improvement of understanding is for two ends: first, our own increase of knowledge; secondly, to enable us to deliver that knowledge to others.”

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Biography

Ivan Beluškov was born and raised in Radoviš, Macedonia on February 23, 1986. After finishing junior year of high school he moved to the United States as part of a student exchange program to finish his senior year of high school. After graduation from High School he enrolled and Received an Associates in Arts degree, Pre-Medicine and Biology major from the Palm Beach State College (Palm Beach Community College) 2006. He was admitted to the Florida Atlantic University Honors Program but instead decided to move back Macedonia and pursue his medical degree at the University of Zagreb School of Medicine, Medical Studies in English Program, in Croatia in 2007. In the same year he got engaged to his beautiful wife Pavlinka and got married in the following year, on the 6th of September 2008. On March 14th 2009 they were blessed with their first child, their beautiful son, Aleksandar and In the following year on December 25th 2010 they were blessed again with the best Christmas present, their beautiful daughter Anastasija.

After he graduates in July 2014 he plans on getting into the Internship program in order to get fully licensed as a general practitioner. Upon receiving his license he would like to stay together with his family in Croatia to work, specialize and progress in the medical field.