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KOMPLEXNÍ KARDIOVASKULÁRNÍ CENTRUM
FAKULTNÍ NEMOCNICE OLOMOUC

Zobrazovací metody u akutní plicní embolie a CTEPH: ECHOKARDIOGRAFIE

Martin Hutyra

4.–7. KVĚTNA 2024 | VELETRHY BRNO
XXXII.
VÝROČNÍ SJEZD
ČESKÉ KARDIOLOGICKÉ
SPOLEČNOSTI

ESC European Society of Cardiology

European Heart Journal (2019) **00**, 1–61
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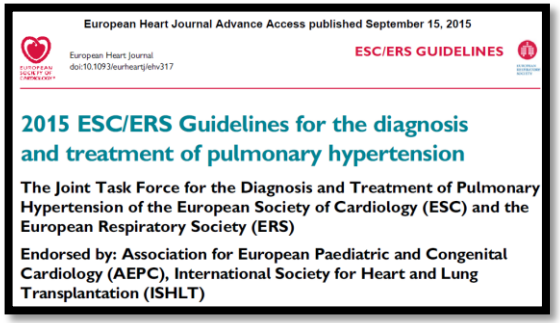
European Heart Journal Advance Access published September 15, 2015

European Heart Journal
doi:10.1093/eurheartj/ehv317

ESC/ERS GUIDELINES

2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension

The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS)

Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT)

ESC GUIDELINES

ESC Guidelines 25 YEARS

2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS)

The Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC)

Konstantinides SV. Eur Respir J. **2019** Aug 31. pii: 1901647. doi: 10.1183/13993003.01647-**2019**.

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I. INTERNÍ KLINIKA KARDIOLOGICKÁ
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Epidemiologie

ESC European Heart Journal (2019) 00, 1–61 doi:10.1093/eurheartj/ehz405
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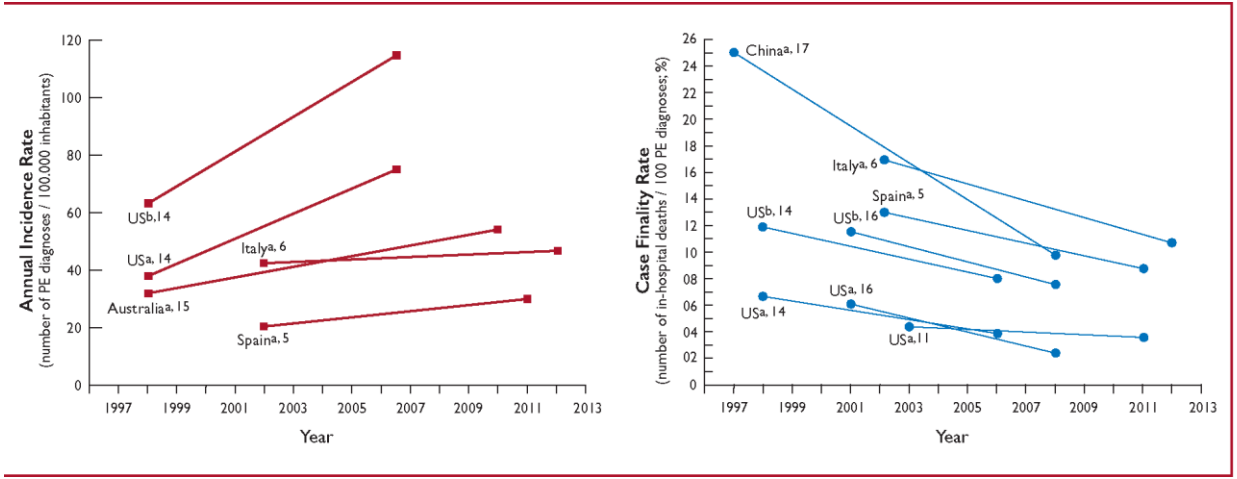
The Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC)



Trends in annual incidence rates (left panel) and case fatality rates (right panel) of pulmonary embolism.

Prevalence 0,4% populace

Incidence 100-200/100 000/1 rok

Autopsie 2356 (79% všech zemřelých z populace 200 tis.) s nálezem PE u 25% a u 18% jako hlavní příčina smrti

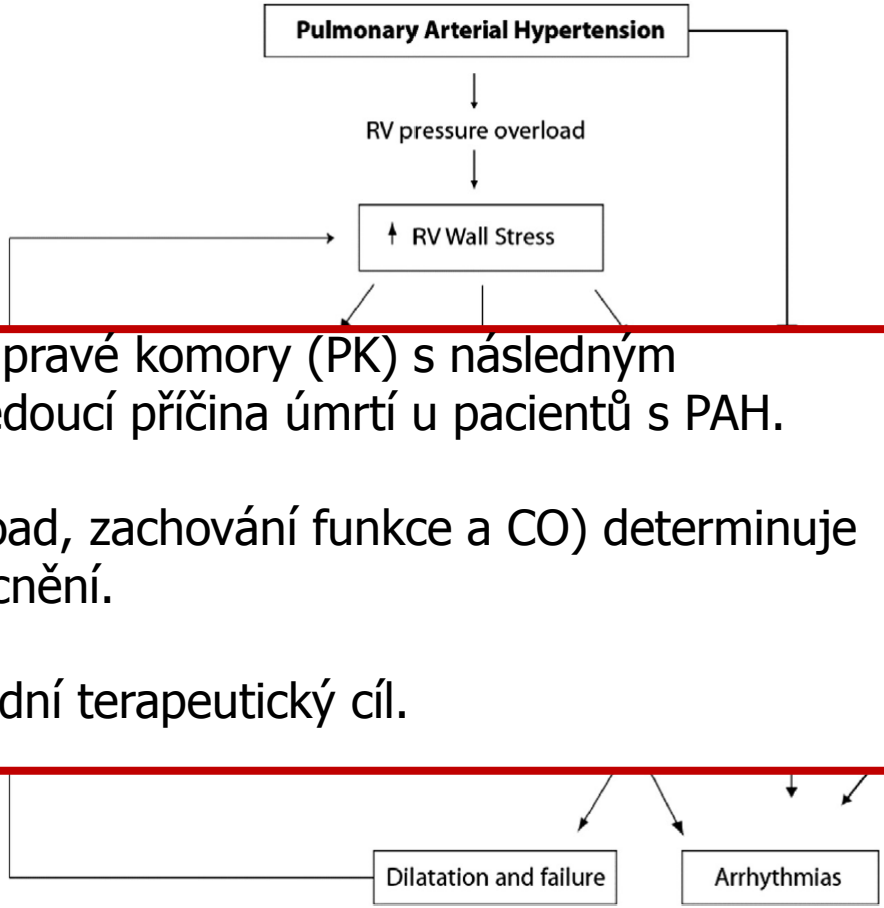
Závislost výskytu na věku a rizikových faktorech

Jen 30-45% pacientů, kteří zemřeli v důsledku plicní embolie bylo adekvátně léčeno

Až 80% pacientů s rizikovými faktory tromboembolie je špatně diagnostikováno

Pravá komora…

- An **afterload mismatch** - increased RV afterload, driven by increased PVR, leads to right heart failure.
- At an early stage, the RV adapts to the increased afterload to preserve stroke volume (**homeometric adaptation**), followed by an
- In contrast, *maladapted ventricles* usually are characterized by **uncoupling of the RV to the pulmonary circulation**



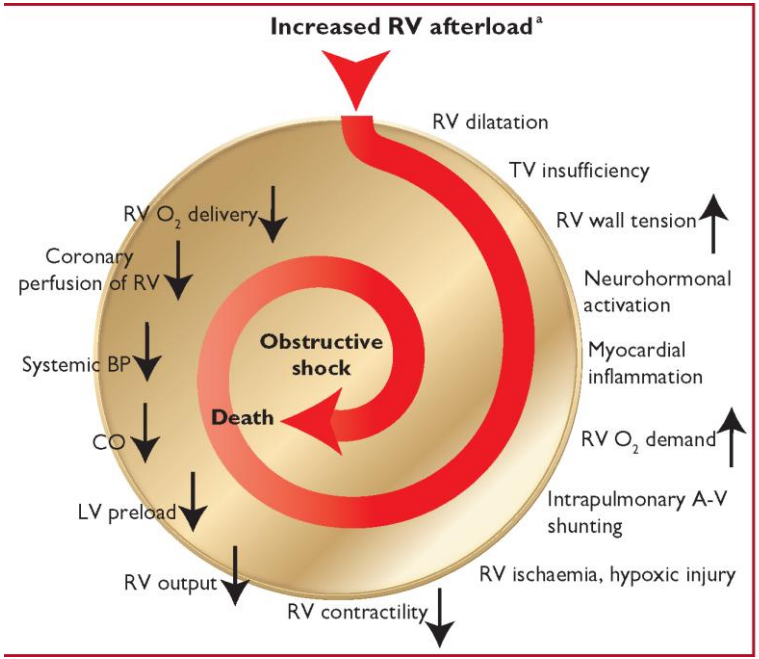
- Vyčerpání kompenzačních mechanismů pravé komory (PK) s následným pravostranným srdečním selháním je vedoucí příčina úmrtí u pacientů s PAH.
- Funkce PK (adaptace na zvýšený afterload, zachování funkce a CO) determinuje funkční status a klinický průběh onemocnění.
- Zachování/zlepšení funkce PK jako zásadní terapeutický cíl.

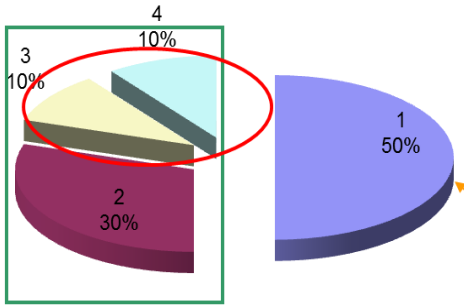
Vonk-Noordegraaf A et al. JA Am Coll Cardiol 2013; Vol. 62, No. 25, Suppl D; D22–33;

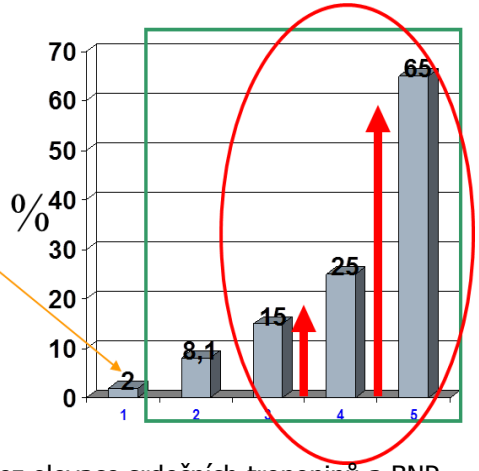
Miotti C et al. J Clin Med 2021, 10, 619.

Patofyziologie a prognóza PE

Key factors contributing to haemodynamic collapse and death in acute pulmonary embolism







1. **Normotenze** bez dilatace a dysfunkce PK, bez elevace srdečních troponinů a BNP
2. **Normotenze s dilatací nebo dysfunkcí PK, plicní hypertenzí**, (elevace srdečních troponinů a BNP)
3. **Systémová hypotenze bez klinických známek šokové cirkulace** (pokles TKs<90 mmHg nebo pokles TKs>40 mmHg, bez nutnosti použití vazopresorů s výjimkou dobutaminu do maximální dávky 5 μg/kg/min)
4. **Kardiogenní obstrukční šok** s orgánovou hypoperfuzí a multiorgánovým selháním
5. Nutnost **iniciální kardiopulmonální resuscitace** a **náhlá srdeční smrt**

Kasper W, Konstantinides S, Greibel A, et al. Management strategies and determinants of outcome in acute pulmonary embolism: results of a multicenter registry. J Am Coll Cardiol 1997;30:1165–71
Goldhaber SZ, Visani L, DeRosa M. for ICOPER. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999; 353: 1386-9

Klinická pravděpodobnost

Geneva skóre:

klinická pravděpodobnost

nízká 0-3 body

střední 4-10

vysoká ≥ 11

Predisponující faktory	
Věk nad 65 let	+1
Předchozí TEN	+3
Chirurgický výkon nebo trauma do 1 měsíce	+2
Malignita	+2
Symptomy	
Bolesti končetiny	+3
Hemoptýza	+2
Fyzikální vyšetření	
Srdeční frekvence	
75-95/min.	+3
>95/min.	+2
Asymetrický otok nebo bolestivost končetiny	+4

Wellsovo skóre:

klinická pravděpodobnost

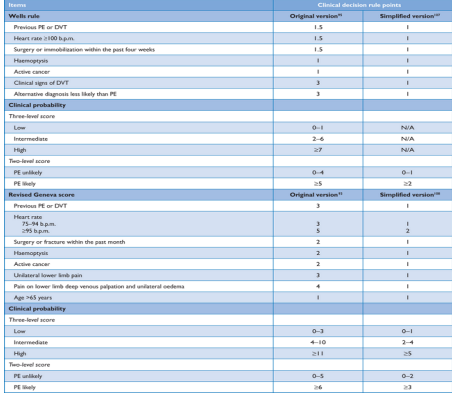
nízká 0-1 body

střední 2-6, vysoká ≥ 7

plicní embolie nepravděpodobná 0-4

pravděpodobná ≥ 5

Predisponující faktory	
Předchozí TEN	+1,5
Recentní chirurgický výkon nebo imobilizace	+1,5
Malignita	+1,0
Symptomy	
Hemoptýza	+1,0
Fyzikální vyšetření	
Tepová frekvence > 100/min.	+1,5
Klinické známky hluboké žilní trombózy	+3,0
Klinické hodnocení	
Jiná diagnóza je méně pravděpodobná než PE	+3,0



Items	Clinical decision rule points	
Wells rule	**Original version[41]**	**Simplified version[107]**
Previous PE or DVT	1.5	1
Heart rate ≥100 b.p.m.	1.5	1
Surgery or immobilization within the past four weeks	1.5	1
Haemoptysis	1	1
Active cancer	1	1
Clinical signs of DVT	3	1
Alternative diagnosis less likely than PE	3	1
Clinical probability		
Three-level score		
Low	0–1	N/A
Intermediate	2–6	N/A
High	≥7	N/A
Two-level score		
PE unlikely	0–4	0–1
PE likely	≥5	≥2
Revised Geneva score	**Original version[41]**	**Simplified version[108]**
Previous PE or DVT	3	1
Heart rate 75–94 b.p.m. ≥95 b.p.m.	3 5	1 2
Surgery or fracture within the past month	2	1
Haemoptysis	2	1
Active cancer	2	1
Unilateral lower limb pain	3	1
Pain on lower limb deep venous palpation and unilateral oedema	4	1
Age >65 years	1	1
Clinical probability		
Three-level score		
Low	0–3	0–1
Intermediate	4–10	2–4
High	≥11	≥5
Two-level score		
PE unlikely	0–5	0–2
PE likely	≥6	≥3

Guo DJ. Chin Med J (Engl). 2015 Apr 20; 128(8): 1052–1057.

Annals of Internal Medicine
ESTABLISHED IN 1927 BY THE AMERICAN COLLEGE OF PHYSICIANS

Performance of 4 Clinical Decision Rules in the Diagnostic Management of Acute Pulmonary Embolism

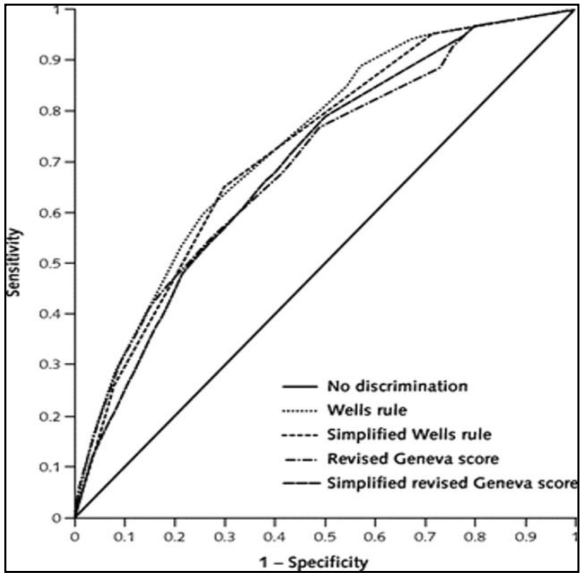
A Prospective Cohort Study from the Promethius Study Group

Ann Intern Med. 2011;154(11):709-718.

Table 4. Accuracy Indexes of the Clinical Decision Rules in Combination With a Normal D-Dimer Result in Patients With a Suspected Event*

Variable	Original Wells Rule (n = 796)	Simplified Wells Rule (n = 803)	RGS (n = 796)	Simplified RGS (n = 795)
Sensitivity†				
Number/number	190/191	191/192	188/189	187/188
Percentage (95% CI)	99.5 (97–100)	99.5 (97–100)	99.5 (97–100)	99.5 (97–100)
Specificity‡				
Number/number	183/605	177/611	184/607	189/607
Percentage (95% CI)	30 (27–34)	29 (25–33)	30 (27–34)	31 (28–34)
Negative predictive value§				
Number/number	183/184	177/178	184/185	189/190
Percentage (95% CI)	99.5 (97–100)	99.4 (97–100)	99.5 (97–100)	99.5 (97–100)

RGS = revised Geneva rule.
* Patients with a clinical decision rule indicating that PE was unlikely but in whom the D-dimer result was missing (protocol violation) were not included in this analysis; this number differed among the 4 clinical decision rules. Sensitivities did not differ among the 4 clinical decision rules in combination with D-dimer test. Specificity differed significantly between the Wells rule and the simplified Wells rule (P = 0.031) and the simplified Wells rule and the simplified RGS (P = 0.017). Other differences in specificity were not statistically significant.
† The number of patients correctly identified as having pulmonary embolism by the combination of clinical decision rules and D-dimer testing divided by the total number of patients with proven pulmonary embolism identified by computed tomography at the time of initial evaluation or venous thromboembolism at 3-mo follow-up.
‡ The number of patients correctly identified as not having pulmonary embolism by the combination of clinical decision rules and D-dimer testing divided by the total number of patients in whom pulmonary embolism was excluded by computed tomography at the time of initial evaluation or venous thromboembolism at 3-mo follow-up.
§ The number of patients correctly identified as not having pulmonary embolism by the combination of clinical decision rules and D-dimer testing divided by the total number of patients with the combination of clinical decision rule and D-dimer testing indicating that pulmonary embolism was excluded (i.e., pulmonary embolism and deep venous thrombosis).



Receiver-operating characteristic curves of the 4 clinical decision rules:
Area under the receiver-operating characteristic curves were 0.73 (95% CI, 0.69 to 0.77) for the Wells rule, 0.72 (CI, 0.68 to 0.76) for the simplified Wells rule, 0.70 (CI 0.65 to 0.74) for the revised Geneva score, and 0.69 (CI, 0.65 to 0.74) for the simplified revised Geneva score.

Diagnostický algoritmus

ESC European Society of Cardiology
European Heart Journal (2019) 00, 1–61 doi:10.1093/eurheartj/ehz405
ESC GUIDELINES

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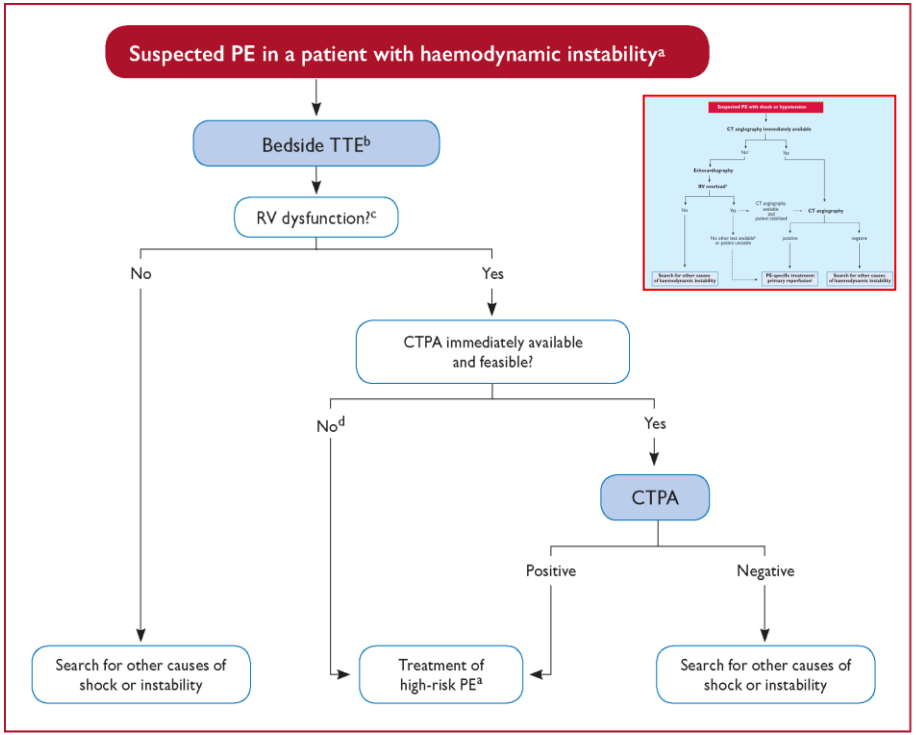

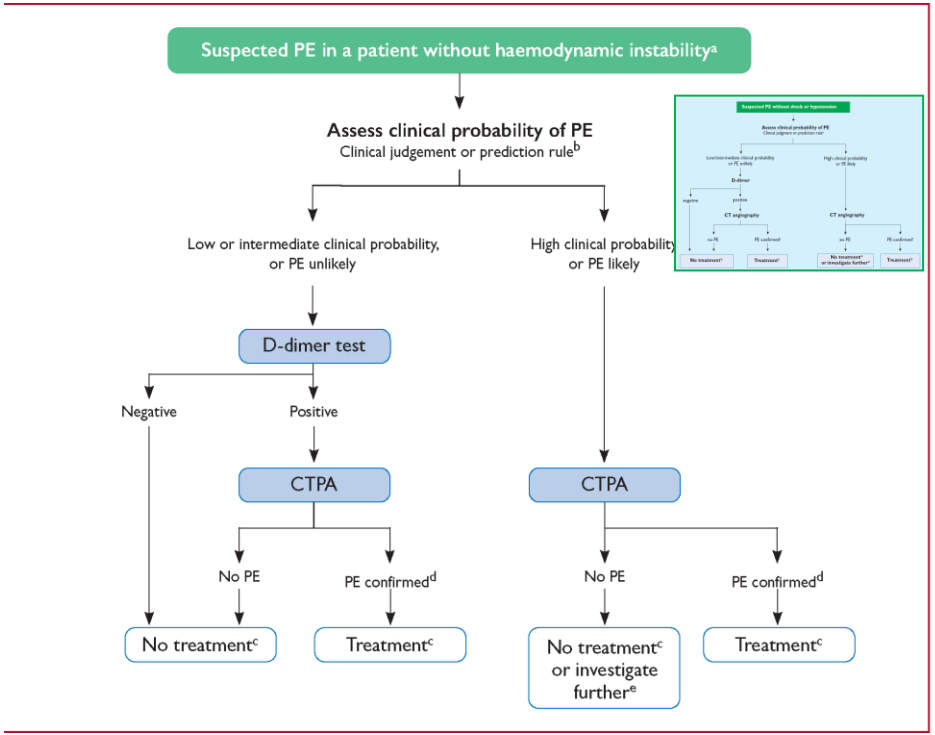

Konstantinides SV. Eur Respir J. **2019** Aug 31. pii: 1901647. doi: 10.1183/13993003.01647-**2019**.

ESC European Society of Cardiology European Heart Journal (2019) 00, 1–61 doi:10.1093/eurheartj/ehz405
ESC GUIDELINES

Klasifikace a riziková stratifikace

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Early mortality risk		Indicators of risk			
		Haemodynamic instability[a]	Clinical parameters of PE severity and/or comorbidity: PESI class III–V or sPESI ≥I	RV dysfunction on TTE or CTPA[b]	Elevated cardiac troponin levels[c]
High		+	(+)[d]	+	(+)
Intermediate	Intermediate–high	-	+[e]	+	+
	Intermediate–low	-	+[e]	One (or none) positive	
Low		-	-	-	Assesment optional; if assessed, negative

BP = blood pressure; CTPA = computed tomography pulmonary angiography; H-FABP = heart-type fatty acid-binding protein; NT-proBNP = N-terminal pro B-type natriuretic peptide; PE = pulmonary embolism; PESI = Pulmonary Embolism Severity Index; RV = right ventricular; sPESI = simplified Pulmonary Embolism Severity Index; TTE = transthoracic echocardiogram.
[a] One of the following clinical presentations (*Table 4*): cardiac arrest, obstructive shock (systolic BP <90 mmHg or vasopressors required to achieve a BP ≥90 mmHg despite an adequate filling status, in combination with end-organ hypoperfusion), or persistent hypotension (systolic BP <90 mmHg or a systolic BP drop ≥40 mmHg for >15 min, not caused by new-onset arrhythmia, hypovolaemia, or sepsis).
[b] Prognostically relevant imaging (TTE or CTPA) findings in patients with acute PE, and the corresponding cut-off levels, are graphically presented in *Figure 3*, and their prognostic value is summarized in Supplementary Data *Table 3*.
[c] Elevation of further laboratory biomarkers, such as NT-proBNP ≥600 ng/L, H-FABP ≥6 ng/mL, or copeptin ≥24 pmol/L, may provide additional prognostic information. These markers have been validated in cohort studies but they have not yet been used to guide treatment decisions in randomized controlled trials.
[d] Haemodynamic instability, combined with PE confirmation on CTPA and/or evidence of RV dysfunction on TTE, is sufficient to classify a patient into the high-risk PE category. In these cases, neither calculation of the PESI nor measurement of troponins or other cardiac biomarkers is necessary.
[e] Signs of RV dysfunction on TTE (or CTPA) or elevated cardiac biomarker levels may be present, despite a calculated PESI of I–II or an sPESI of 0.[234] Until the implications of such discrepancies for the management of PE are fully understood, these patients should be classified into the intermediate-risk category.

ESC European Heart Journal (2019) 00, 1–61
European Society of Cardiology doi:10.1093/eurheartj/ehz405
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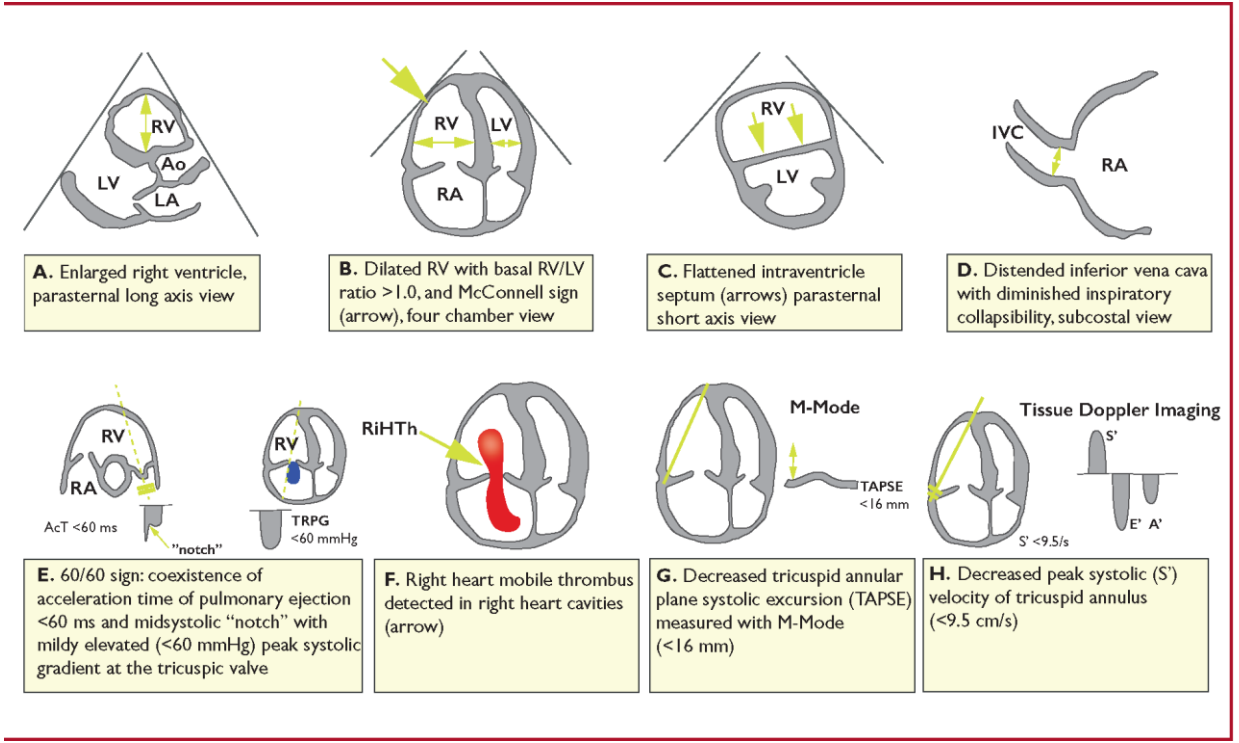
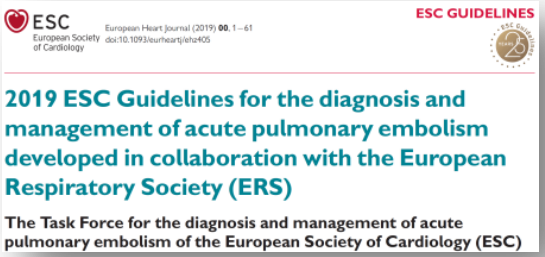
PESI

Parameter	Original version[214]	Simplified version[218]
Age	Age in years	1 point (if age >80 years)
Male sex	+10 points	–
Cancer	+30 points	1 point
Chronic heart failure	+10 points	1 point
Chronic pulmonary disease	+10 points	
Pulse rate ≥110 b.p.m.	+20 points	1 point
Systolic blood pressure <100 mm Hg	+30 points	1 point
Respiratory rate >30 breaths per minute	+20 points	–
Temperature <36 °C	+20 points	–
Altered mental status	+60 points	–
Arterial oxyhaemoglobin saturation <90%	+20 points	1 point
	Risk strata[a]	
	Class I:≤65 points very low 30-day mortality risk (0–1.6%) **Class II: 66–85 points** low mortality risk (1.7–3.5%) **Class III: 86–105 points** moderate mortality risk (3.2–7.1%) **Class IV: 106–125 points** high mortality risk (4.0–11.4%) **Class V: >125 points** very high mortality risk (10.0–24.5%)	**0 points**= 30-day mortality risk 1.0% (95% CI 0.0%–2.1%) **≥1 point(s)**= 30-day mortality risk 10.9% (95% CI 8.5%–13.2%)

Konstantinides SV. Eur Respir J. **2019** Aug 31. pii: 1901647. doi: 10.1183/13993003.01647-**2019**.

Echokardiografie v rizikové stratifikaci PE





Konstantinides SV. Eur Respir J. **2019** Aug 31. pii: 1901647. doi: 10.1183/13993003.01647-**2019**.

Odhad systolického tlaku v plicnici - trikuspidální regurgitační gradient

základní parametr pro screening PH pomocí echokardiografie

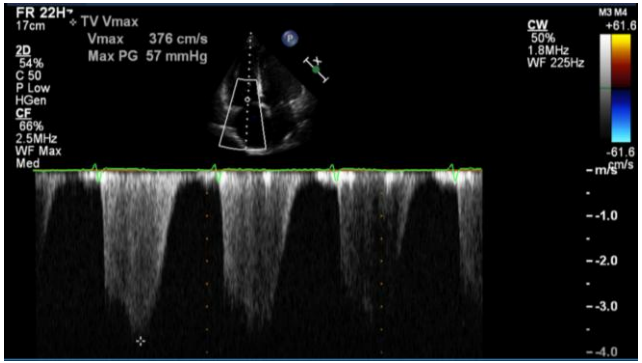
pro **hodnocení prognózy a rozhodnutí o terapii nemá význam**

vzestup odhadovaného sPAP nemusí nutně ukazovat progresi onemocnění, pokles odhadovaného sPAP nemusí nutně odpovídat zlepšení PAP

invazivně měřený mPAP má rovněž poměrně malý prognostický význam (kromě responderů akutní vazoreaktivity)

Tabulka 4A – Pravděpodobnost zjištění plicní hypertenze echokardiografickým vyšetřením symptomatických pacientů s podezřením na plicní hypertenzi

Maximální rychlost proudu krve při nedomykavosti trojcípé chlopně (m/s)	Přítomnost jiných „známek PH" při echokardiografickém vyšetření[a]	Možnost plicní hypertenze podle echokardiografického vyšetření
≤ 2,8 nebo neměřitelná	Ne	Nízká
≤ 2,8 nebo neměřitelná	Ano	Středně vysoká
2,9–3,4	Ne	
2,9–3,4	Ano	Vysoká
> 3,4	Není nutno provádět	



Sitbon O et al. J Am Coll Cardiol. 2002 Aug 21;40(4):780-8. McLaughlin VV et al. Eur Respir J. 2005 Feb;25(2):244-9. Nickel N et al. Eur Respir J. 2012 Mar;39(3):589-96.

Longitudinální funkce PK

European Heart Journal – Cardiovascular Imaging (2015) 16, 233–271
doi:10.1093/ehjci/jev014
POSITION PAPER

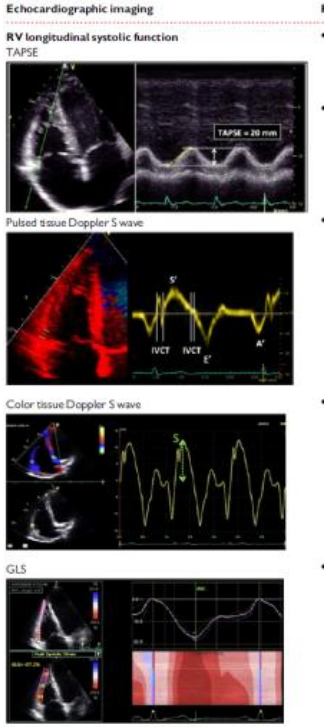
Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging

Tricuspid annular plane systolic excursion (TAPSE) reflects longitudinal shortening of the RV. TAPSE is measured in the A4C by placing an M-mode cursor on the lateral tricuspid annulus and measuring the peak distance travelled by this reference point during systole. A greater distance travelled during systole implies greater RV systolic function, with the normal reference limit being a TAPSE of ≥1.7 cm.

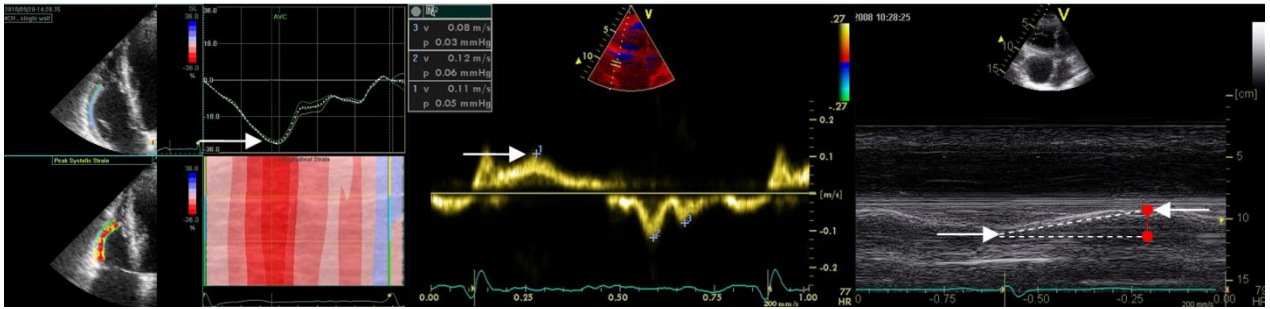
The primary limitation of TAPSE is that it only represents one component of RV motion within one single segment of RV myocardium. The RV may be frankly dysfunctional despite relatively preserved TAPSE, as in some cases of severe pulmonary arterial hypertension. Alternatively, the RV function may be globally preserved despite significantly reduced TAPSE, as often seen after cardiac surgery. In healthy individuals, TAPSE correlates with RV size.

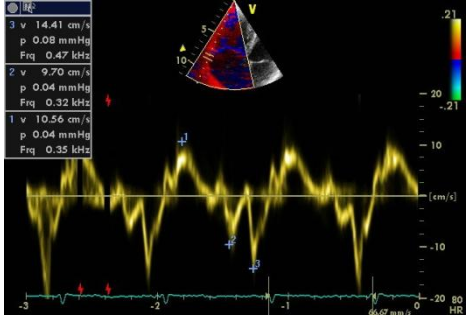
Two common sources of error with TAPSE are:

1. Not placing the M-mode cursor parallel to the plane of longitudinal motion, which results in angle-dependent underestimation of TAPSE.
2. Incorrectly measuring the magnitude of displacement from the M-mode image.



Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1.





Tricuspid annular velocity reflects the longitudinal velocity of the tricuspid annulus during systole.

S' is measured in the A4C by placing a tissue Doppler cursor on the lateral tricuspid annulus and measuring the peak velocity of this reference point during systole. Care should be taken to measure the peak of the ejection waveform and not the earlier isovolumetric contraction waveform.

A greater velocity during systole implies greater RV systolic function, with the **normal reference limit** being an S' of ≥9.5 cm/s. Both pulsed tissue Doppler and color-coded tissue Doppler can be used to measure S', although the color-coded method yields mean velocities that are usually slightly lower.

The **advantages and limitations** are the same as TAPSE:

1. S' is simple to perform and has prognostic data, yet it is angle-dependent and only represents the longitudinal annular component of RV motion.
2. S' has been shown to correlate with CMR-derived RVEF and predicts outcomes in patients with pulmonary hypertension, inferior myocardial infarction, chronic heart failure, and arrhythmogenic RV cardiomyopathy (ARVC).

Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1.

Frakční změna plochy PK

Fractional area change (FAC) is the % change in RV area from diastole to systole, a two-dimensional surrogate for RV EF, and thereby reflects the systolic function of the inflow and apical portions of the RV. FAC encompasses longitudinal shortening as well as radial thickening and the contribution of the septum.

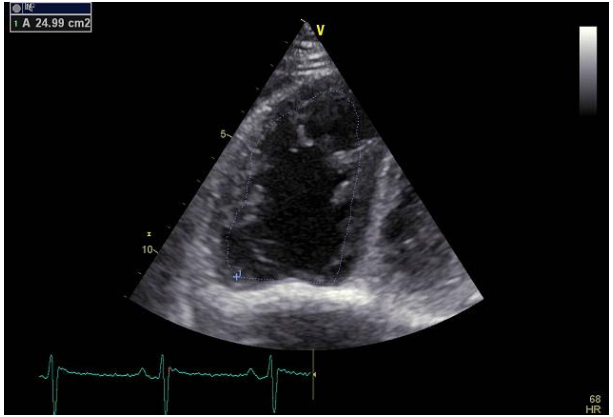
FAC = [(end-diastolic RV area – end-systolic RV area)/end-diastolic RV area] x 100

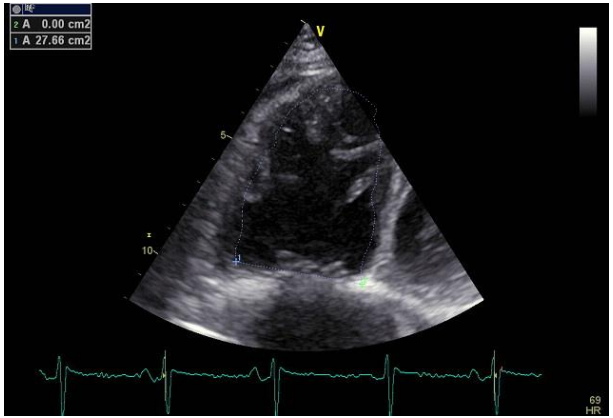
The normal reference limit for FAC is ≥35%.

The primary challenge and main limitation of FAC is the accurate tracing of the true RV endocardial border.

Compared with TAPSE and S', FAC was found to correlate best with the reference standard of CMR-derived RVEF *(r=0.80)*.

1. In substudies from the SAVE and VALIANT trials, 416 and 522 patients with **AMI** and evidence of LV dysfunction underwent complete echocardiographic assessment. Four independent predictors of subsequent all-cause mortality were identified: age, Killip classification, LV ejection fraction, and FAC; with FAC <35 percent carrying an adjusted hazard ratio of 3.56.
2. In the **Multidisciplinary Study of Right Ventricular Dysplasia**, FAC was found to be significantly reduced in probands compared with normal controls. The revised ARVC Task Force Criteria list FAC ≤33% as a major diagnostic criterion and FAC 34-40% as a minor criterion





Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1.

Strain PK

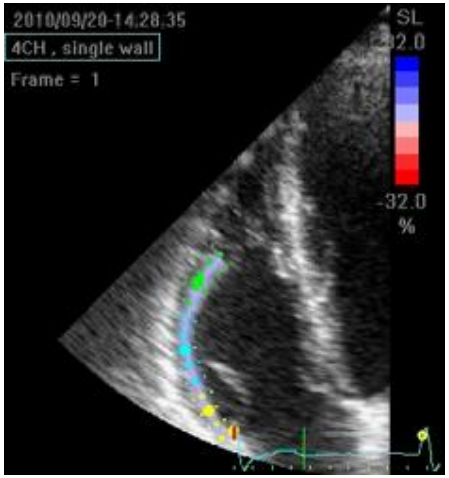
2D strain imaging is defined as the % change in myocardial deformation (RV longitudinal shortening). Strain is currently measured principally by the speckle-tracking (non-angle-dependent) approach.

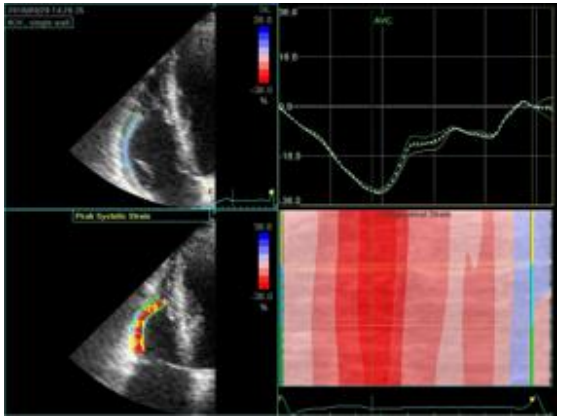
Potential pitfalls include technical challenges in image acquisition and analysis (need for high frame rates, high signal-to-noise, experienced observers for reproducible measurements).

Contemporary speckle-tracking algorithms have enhanced reproducibility and are beginning to yield **clinically relevant** observations:

1. In a large cohort of 575 patients with pulmonary arterial hypertension, free wall longitudinal strain by 2D speckle tracking was predictive of functional capacity and 18-month mortality.
2. In 200 patients with heart failure and seemingly normal RV systolic function (TAPSE >16 mm), a substantial proportion of patients was found to have abnormal RV free wall strain indicative of subclinical RV dysfunction, which was in turn predictive of death and hospitalization.
3. To identify signs of RV infarction in patients presenting with acute myocardial infarction, RV free wall strain was superior to conventional echocardiographic parameters.

The **normal reference limit** for 2DS of the RV free wall is -23%/-20%.





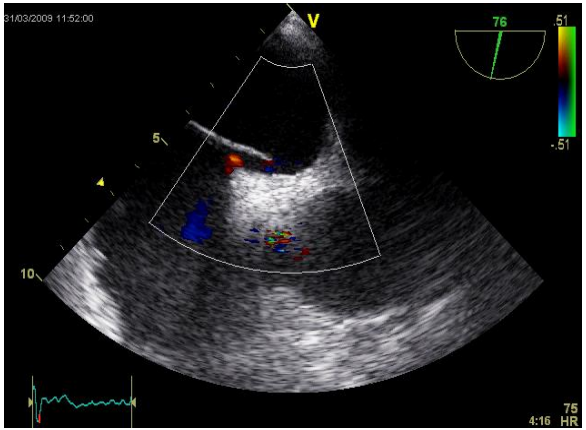
PFO jako zdroj paradoxní embolizace

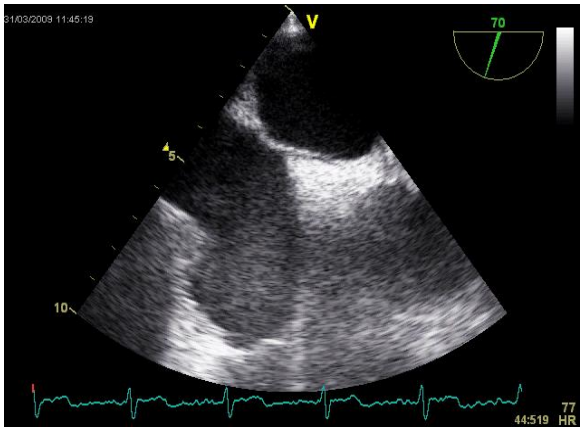
<u>Nemocní s PFO a plicní embolií mají:</u>

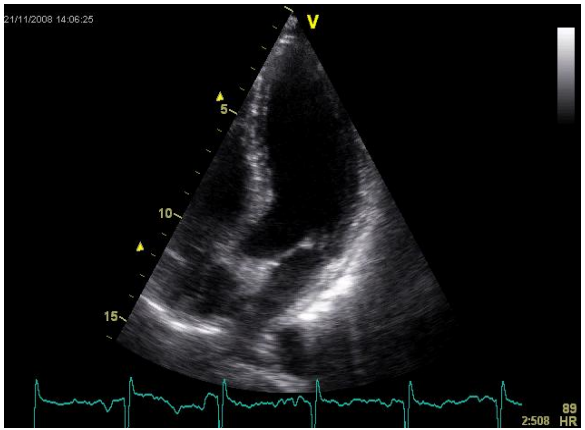
Vyšší riziko mortality (33% vs. 14%)

Vyšší výskyt ischemické CMP (13% vs. 2%)

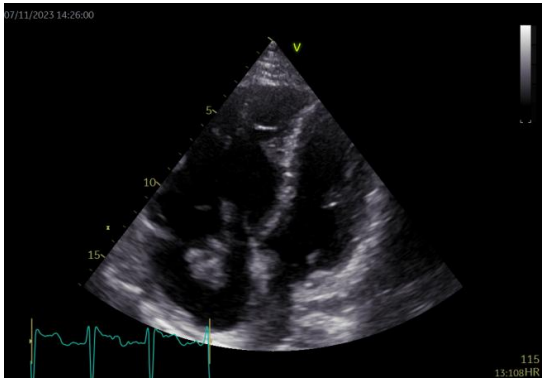
Vyšší incidenci periferní arteriální embolizace (15% vs. 0%)

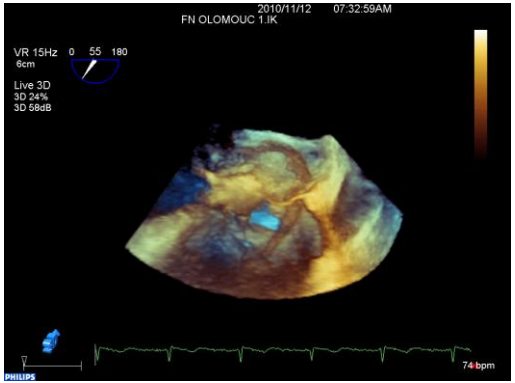


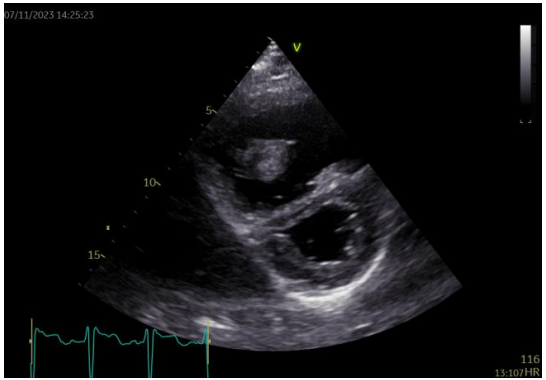


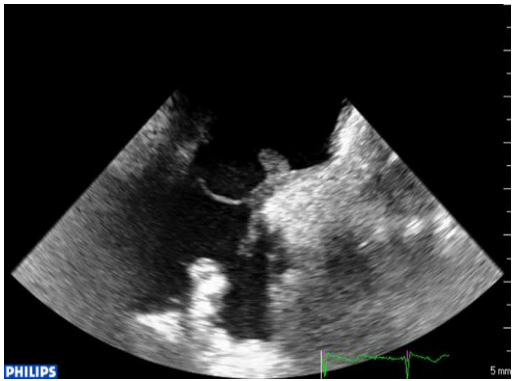


Tranzientní tromby









Přítomny až u 4 (18)% nemocných s plicní embolií

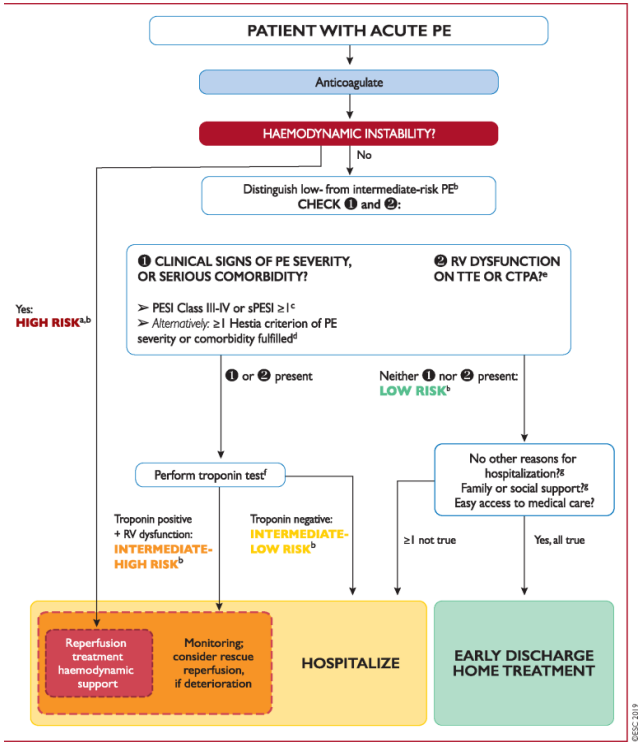
Závažný nález indikující trombolýzu

Při kontraindikaci (při PFO z rizikem vzniku paradoxní embolizace) k embolektomii

Casazza F, Bongarzoni A. Prevalence and prognostic significance of right-sided cardiac mobile thrombi in acute massive pulmonary embolism. Am J Cardiol 1997; 79:1433-5
Chartier L, Bera J. et al. Free-Floating Thrombi in the right Heart: Diagnosis, management and prognostic indexes in 38 consecutive patients. Circulation 1999; 99: 2779-83

ESC doporučení 2019 – diagnostika a management

Diagnosis	
A D-dimer test, using an age-adjusted cut-off or adapted to clinical probability, should be considered as an alternative to the fixed cut-off level.	IIa
If a positive proximal CUS is used to confirm PE, risk assessment should be considered to guide management.	IIa
V/Q SPECT may be considered for PE diagnosis.	IIb
Risk assessment	
Assessment of the RV by imaging or laboratory biomarkers should be considered, even in the presence of a low PESI or a sPESI of 0.	IIa
Validated scores combining clinical, imaging, and laboratory prognostic factors may be considered to further stratify PE severity.	IIb



Risk-adjusted management strategy for acute pulmonary embolism. CTPA = computed tomography pulmonary angiography/angiogram; PE = pulmonary embolism; PESI = Pulmonary Embolism Severity Index; RV = right ventricular; sPESI = simplified Pulmonary Embolism Severity Index; TTE = transthoracic echocardiogram.

Konstantinides SV. Eur Respir J. **2019** Aug 31. pii: 1901647. doi: 10.1183/13993003.01647-**2019**.

(high-risk) plicní embolie se zástavou oběhu a KPR

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rt-PA při zástavě oběhu způsobené plicní embolií

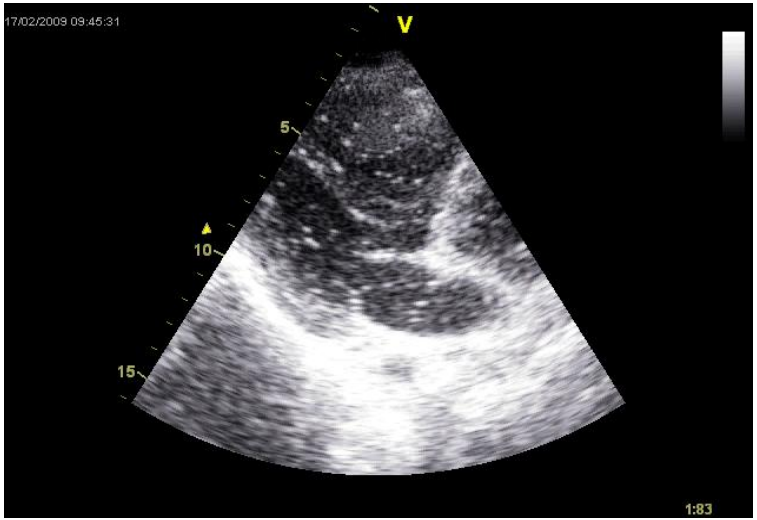
p<0,05

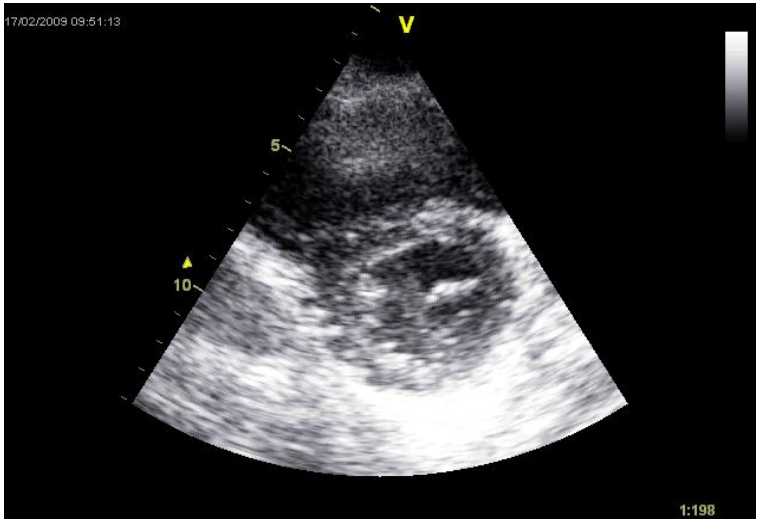
Prospektivní studie srovnávající **rt-PA** 50 mg/2 min. (n=40) + heparin i.v. s **konvenčním postupem** (n=50) u pacientů s masivní PE a >15 min. trvající oběhovou zástavou s nutností KPR

1 zachráněný život na 8 trombolyzovaných pac.

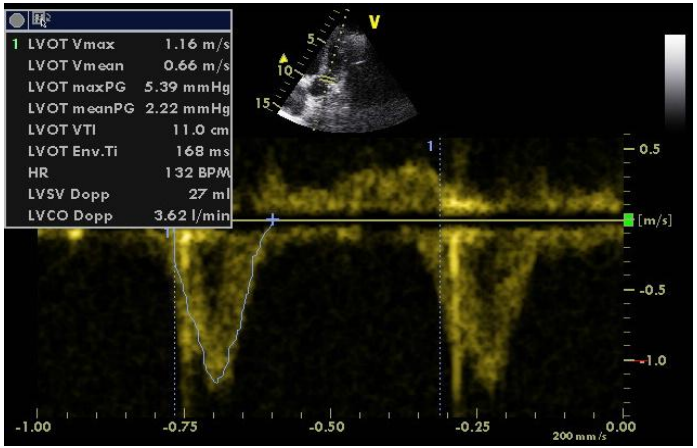
%

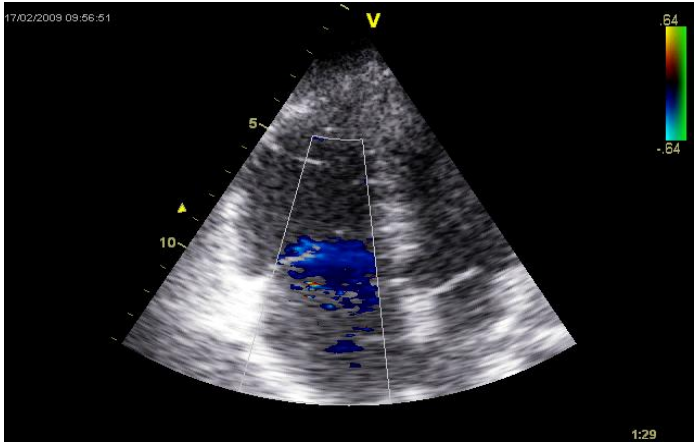
p=NS

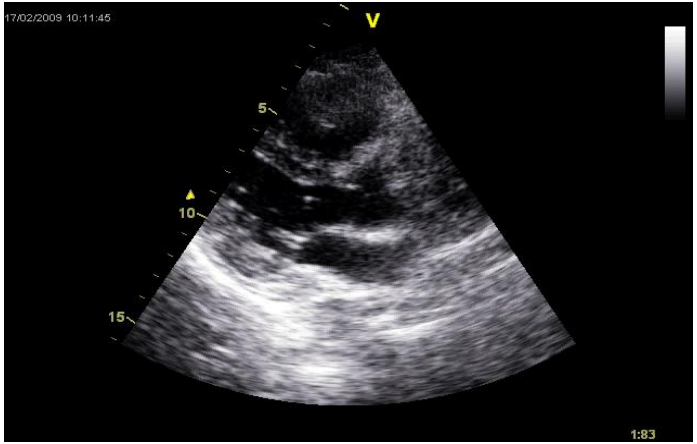

17/02/2009 09:45:31
V
5
10
15
1:83


17/02/2009 09:51:13
V
5
10
1:198


LUCAS™


1 LVOT Vmax 1.16 m/s
LVOT Vmean 0.66 m/s
LVOT maxPG 5.39 mmHg
LVOT meanPG 2.22 mmHg
LVOT VTI 11.0 cm
LVOT Env.Ti 168 ms
HR 132 BPM
LVSV Dopp 27 ml
LVCO Dopp 3.62 l/min
0.5
[m/s]
-0.5
-1.0
-1.00
-0.75
-0.50
-0.25
0.00
200 mm/s


17/02/2009 09:56:51
.64
-.64
1:29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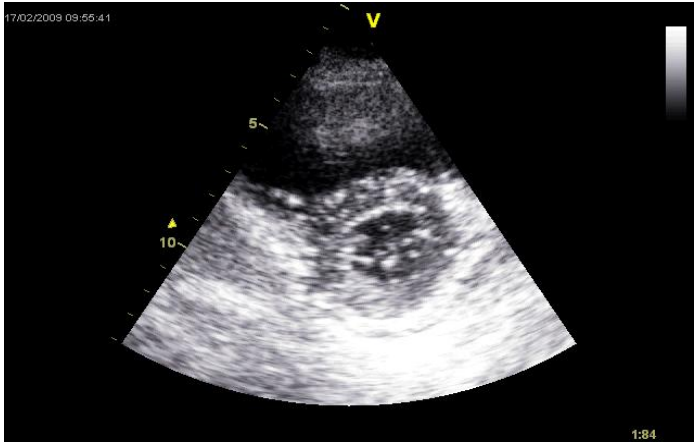
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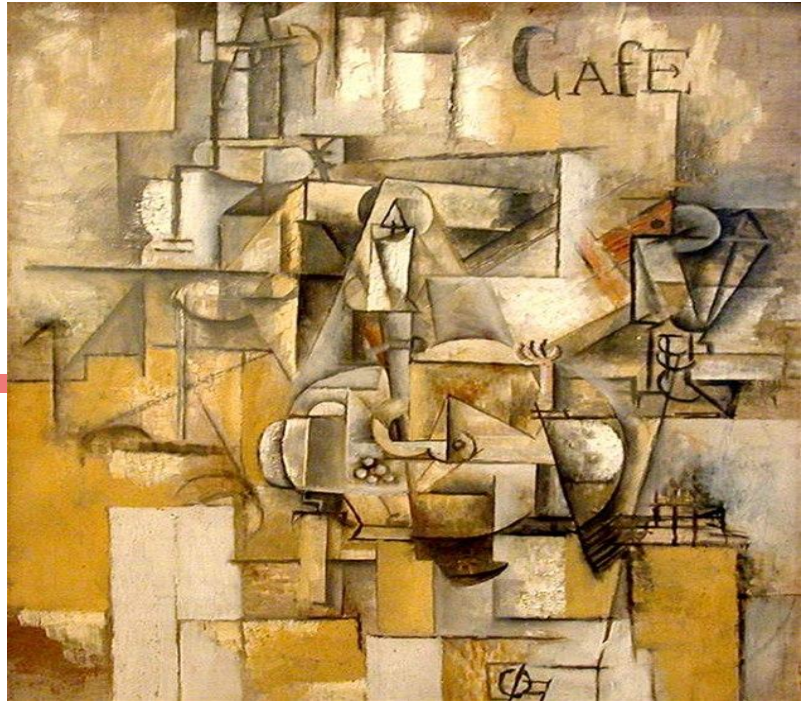

(high-risk) plicní embolie s oběhovou nestabilitou

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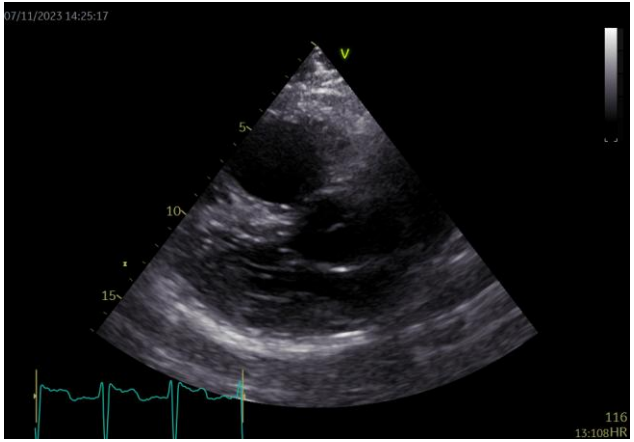
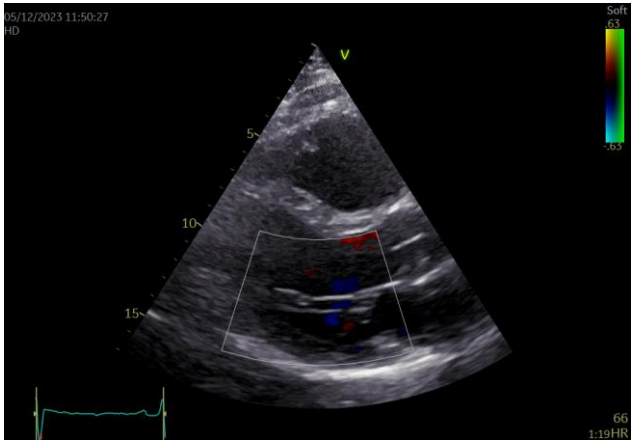
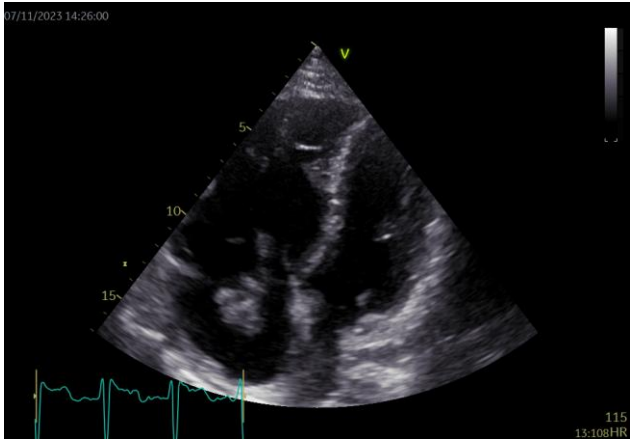
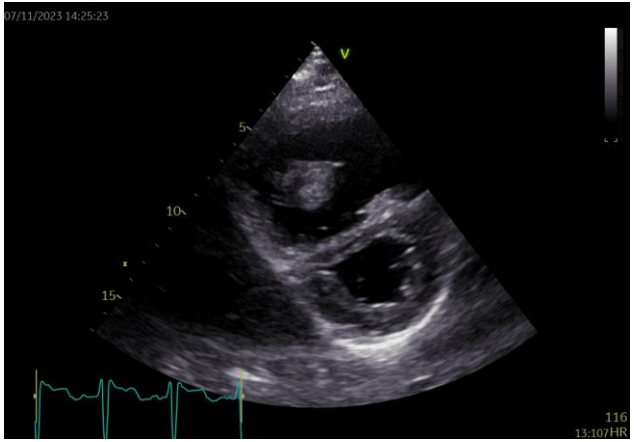
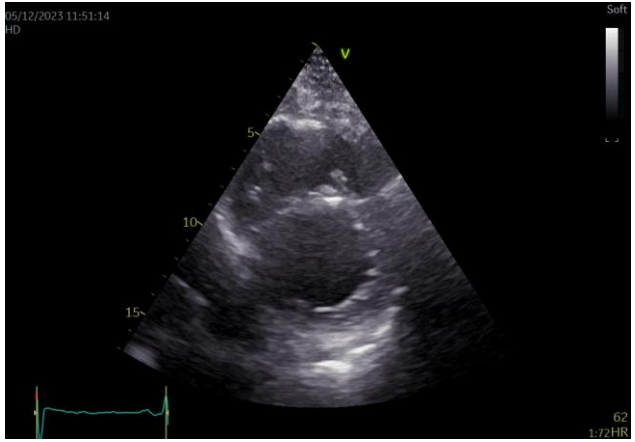
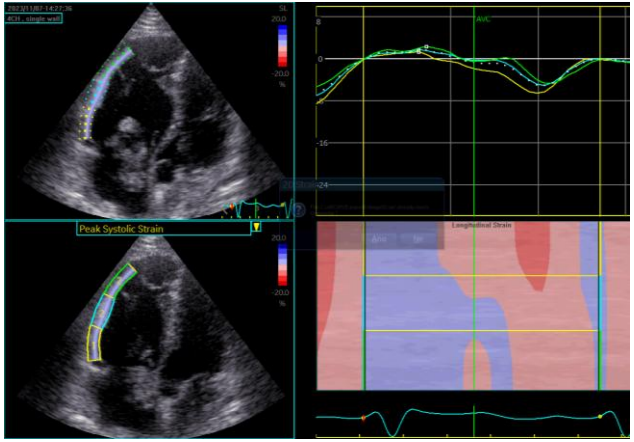

07/11/2023 14:25:17
116
13:108HR

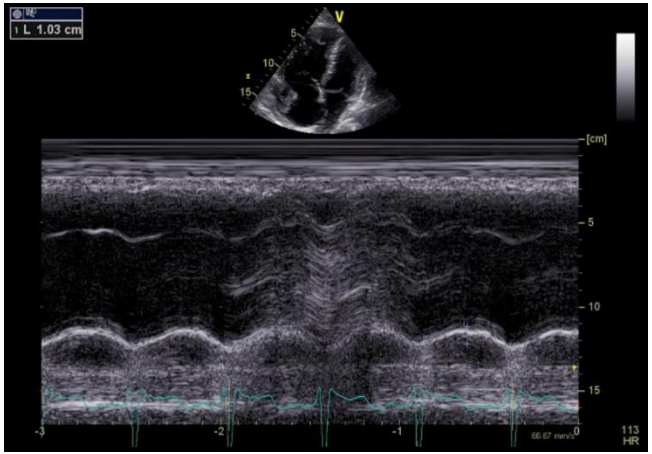

05/12/2023 11:50:27
Soft
66
1:19HR

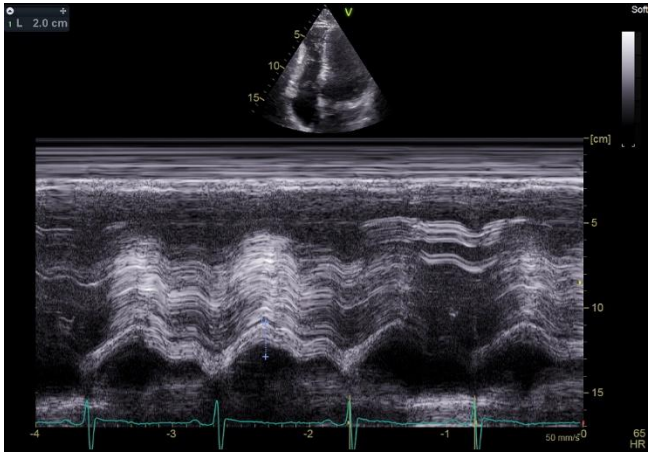

07/11/2023 14:26:00
115
13:108HR

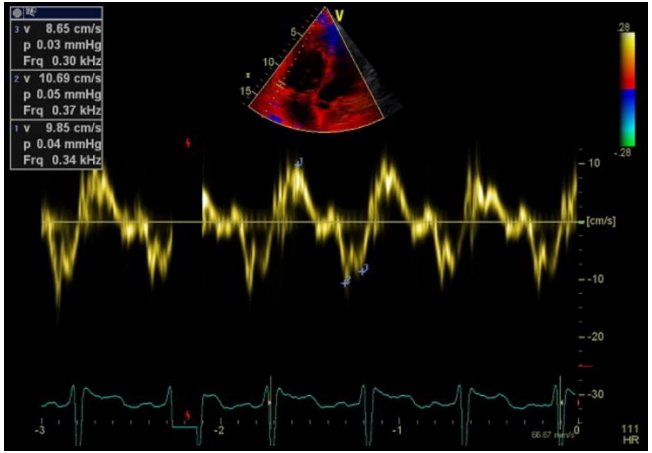

07/11/2023 14:25:23
116
13:107HR

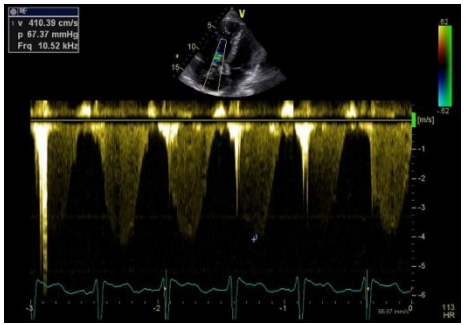

05/12/2023 11:51:14
Soft
62
1:72HR

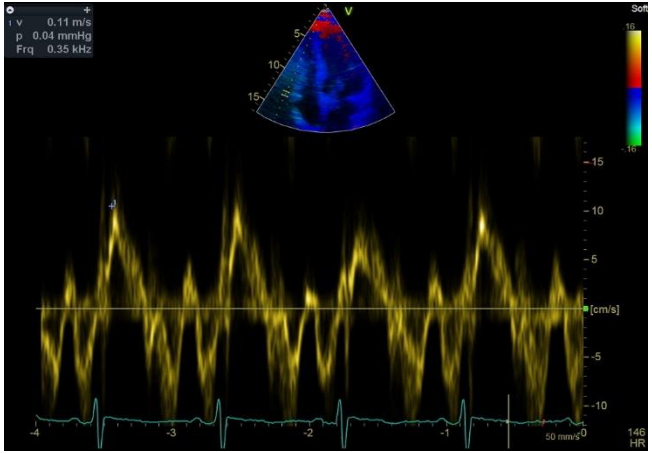

Peak Systolic Strain


L 1.03 cm
[cm]


L 2.0 cm
[cm]


v 8.65 cm/s
p 0.03 mmHg
Frq 0.30 kHz
v 10.69 cm/s
p 0.05 mmHg
Frq 0.37 kHz
v 9.85 cm/s
p 0.04 mmHg
Frq 0.34 kHz
[cm/s]

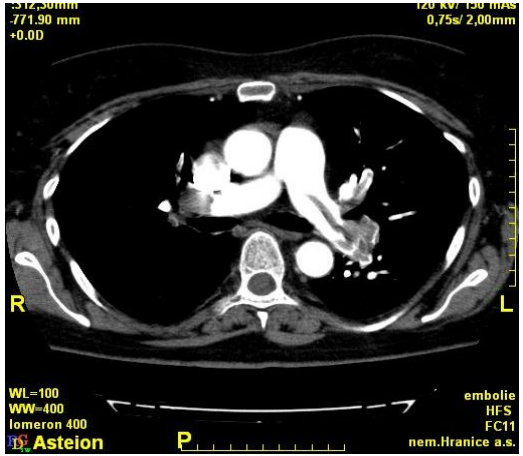

v 410.39 cm/s
p 67.37 mmHg
Frq 10.52 kHz
[m/s]

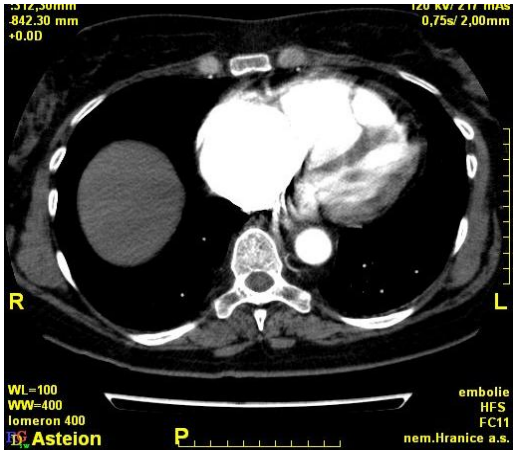

v 0.11 m/s
p 0.04 mmHg
Frq 0.35 kHz
[cm/s]

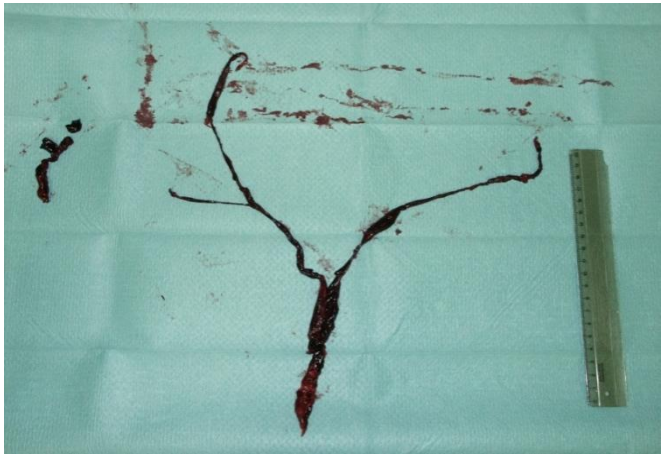
Trombolýza vs. heparin u plicní embolie

Jerjez-Sanchez, J Thromb Thrombolysis 1995;2:227–9

Study	Treatment regimens		No. of patients	Mortality, n (%)	Recurrence n (%)	Major haemorrhage,* n (%)	Comments
Jerjes-Sanchez	*STREPROKINÁZA vs.*	*4*	*0 (0%)*	*0*	*2 (4,3%)*		
et al.	*HEPARIN*	*4*	*4 (100%)*	*NA*	*0*		

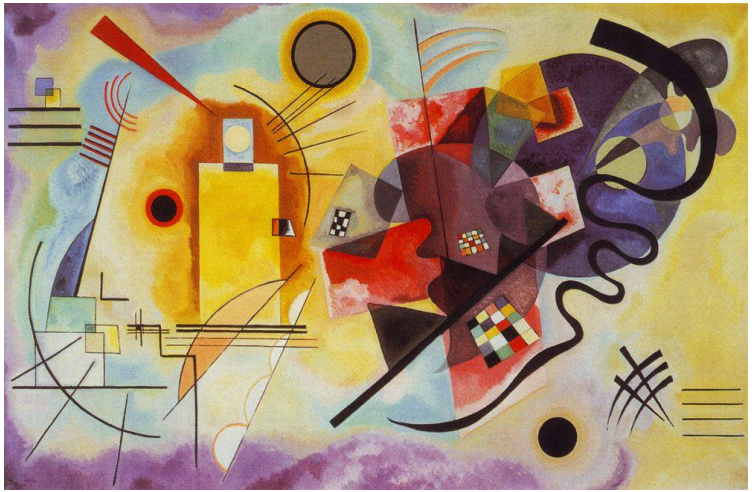


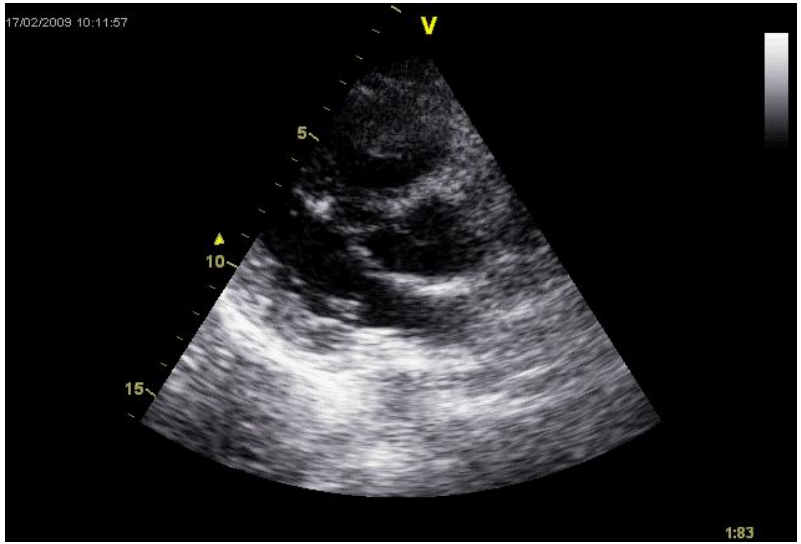


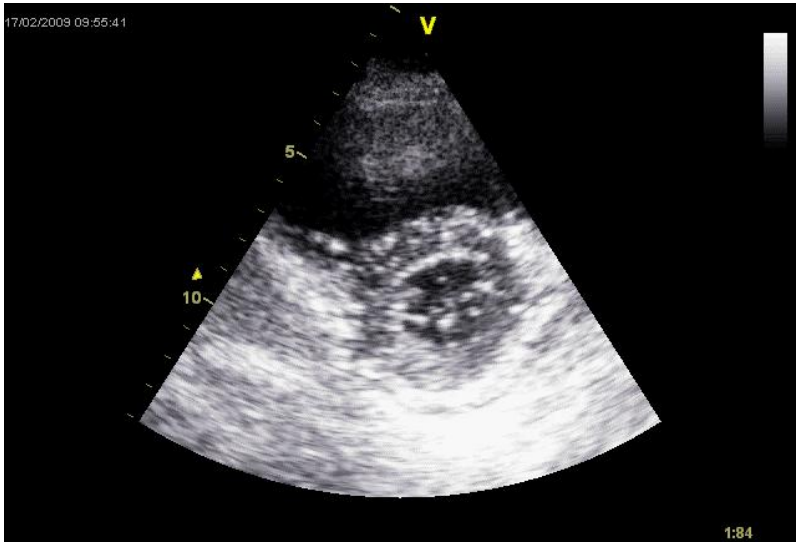


Jerjes-Sanchez C, Ramirez-Rivera A, Garcia M de L, et al. Streptokinase and heparin versus heparin alone in massive pulmonary embolism; a randomized controlled trial. J Thromb Thrombolysis 1995;2:227–9

Středně riziková plicní embolie







Normální systémový tlak, anamnéza synkopy (!), narůstající dušnost při léčbě LMWH, troponin T 240 ng/l, NT-proBNP 11375 ng/l

Chronic Thromboembolic Pulmonary Hypertension (CTEPH)

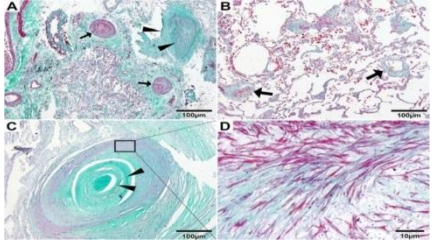
Results From an International Prospective Registry

Joanna Pepke-Zaba, MD; Marion Delcroix, MD; Irene Lang, MD; Eckhard Mayer, MD; Pavel Jansa, MD; David Ambroz, MD; Carmen Treacy, BSc; Andrea M. D'Armini, MD; Marco Morsolini, MD; Repke Snijder, MD; Paul Bresser, MD; Adam Torbicki, MD; Bent Kristensen, MD; Jerzy Lewczuk, MD; Iveta Simkova, MD; Joan A. Barberà, MD; Marc de Perrot, MD; Marius M. Hoeper, MD; Sean Gaine, MD; Rudolf Speich, MD; Miguel A. Gomez-Sanchez, MD; Gabor Kovacs, MD; Abdul Monem Hamid, MD; Xavier Jaïs, MD; Gérald Simonneau, MD

Table 2. Patients' History of Venous Thromboembolism

	All Patients (n=679)	Operable Patients* (n=427)	Nonoperable Patients* (n=247)	*P* (Exploratory)
Confirmed previous acute PE, % (n)	74.8 (678)	77.5 (427)	70.0 (247)	0.0344
PE diagnosed more than once, % (n)	32.8 (469)	35.0 (303)	28.8 (163)	0.2145
Size of previous PE reported as massive, % (n)	40.8 (240)	47.1 (155)	29.4 (85)	0.0090
Confirmed previous DVT, % (n)	56.1 (426)	60.4 (280)	49.0 (143)	0.0295
Acute PE and DVT, % (n)	55.4 (413)	59.3 (270)	48.9 (141)	0.0477
Acute PE no DVT, % (n)	42.6 (413)	39.3 (270)	48.2 (141)	0.0926
Thrombolytic treatment, % (n)	14.4 (404)	18.5 (265)	6.6 (137)	0.0009
Vena cava filter implanted, % (n)	12.4 (491)	13.7 (322)	10.2 (166)	0.3139

P values from Fisher exact test. (n): patients with assessment. DVT indicates deep vein thrombosis; PE, pulmonary embolism.
*5 patients had no data on operability.



Infekce

Zánět

Immunita

Genetická predispozice

Žilní trombóza

Plicní embolie

In situ trombózy

Nekompletní rezoluce a organizace trombu

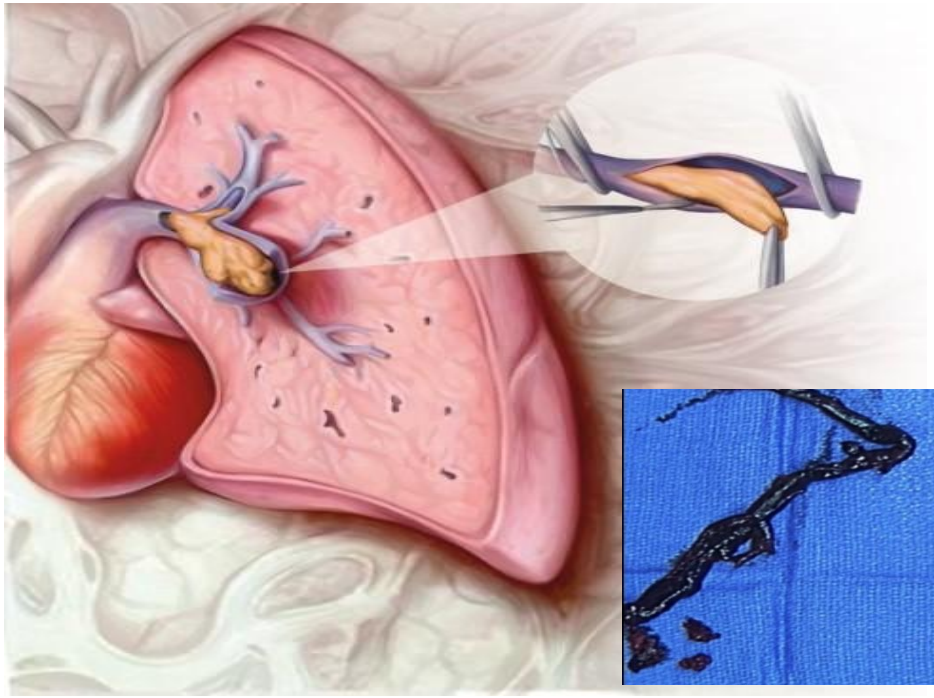
Okluze/obliterace plicní tepny

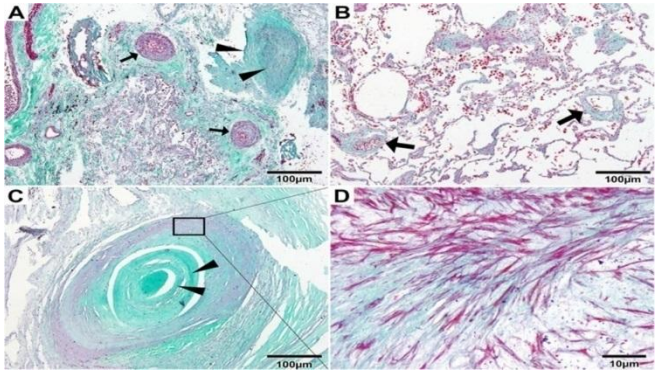
↑ afterload pravé komory

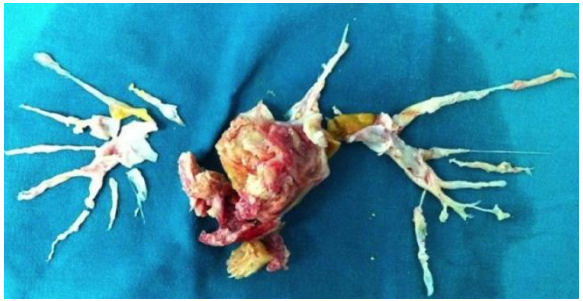
Progresivní ↑ PVR

CTEPH

Zaba et al. Chronic thrombormbolic pulmonary hypertension. Circ. 2011;124(18):1973-1981







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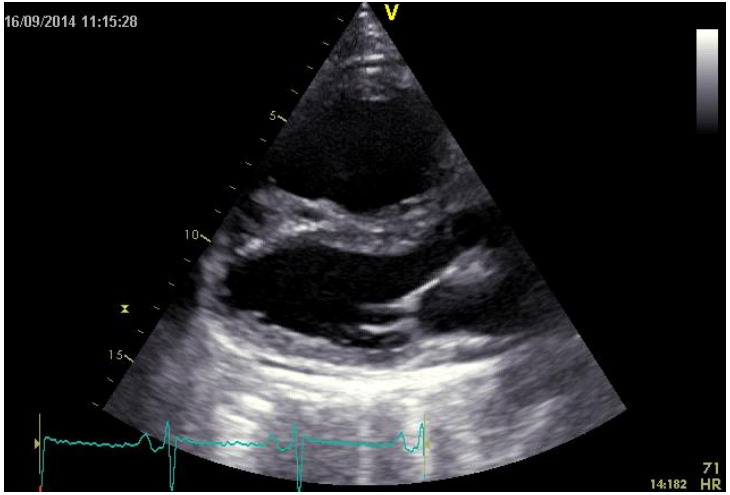
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71
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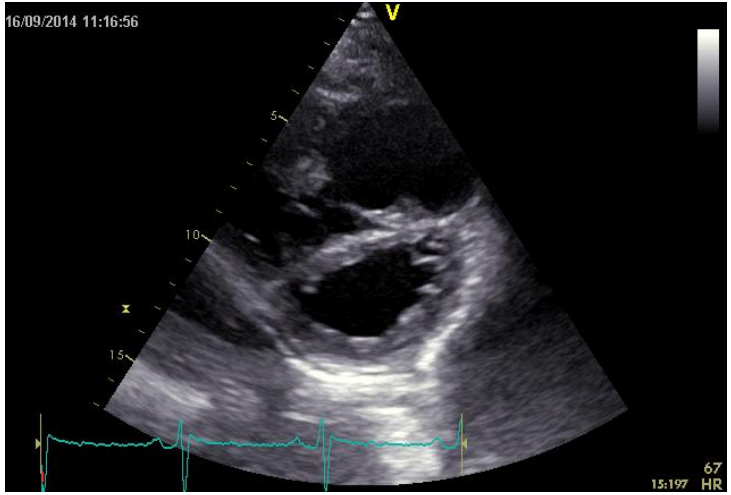
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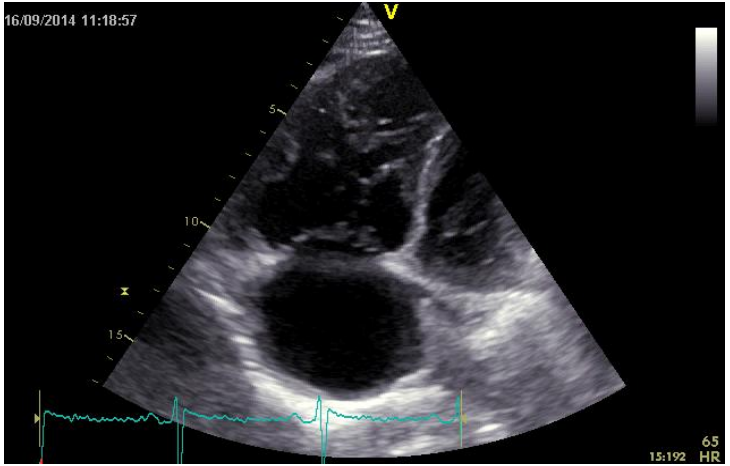
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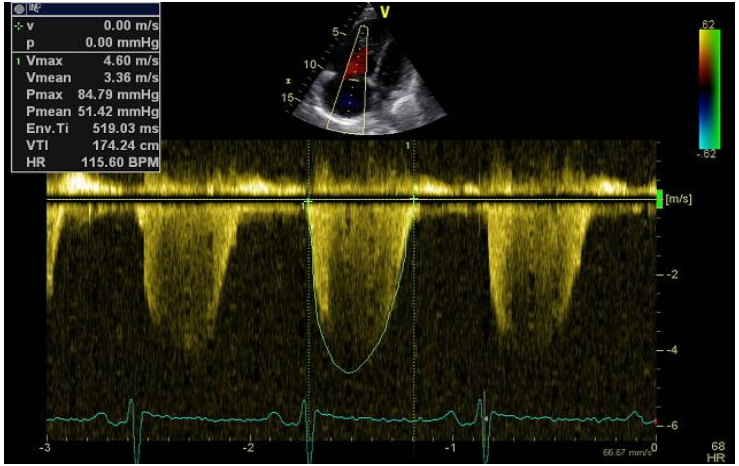
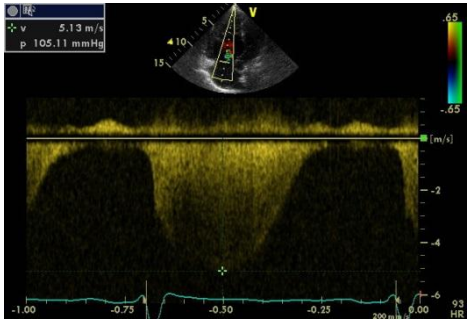

v 0.00 m/s
p 0.00 mmHg
Vmax 4.60 m/s
Vmean 3.36 m/s
Pmax 84.79 mmHg
Pmean 51.42 mmHg
Env.Ti 519.03 ms
VTI 174.24 cm
HR 115.60 BPM
[m/s]
68
HR

ECHOKARDIOGRAFIE



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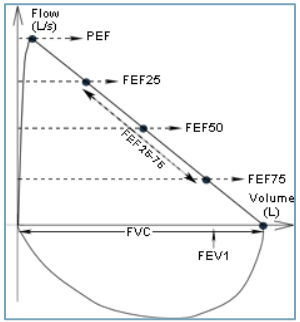

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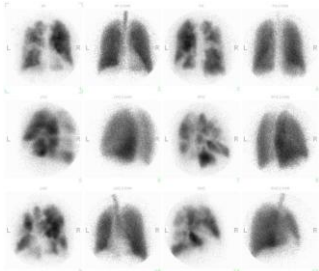
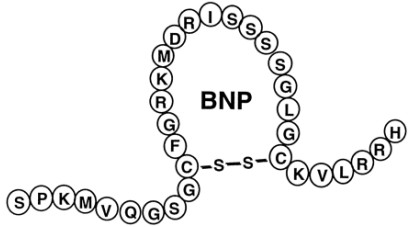
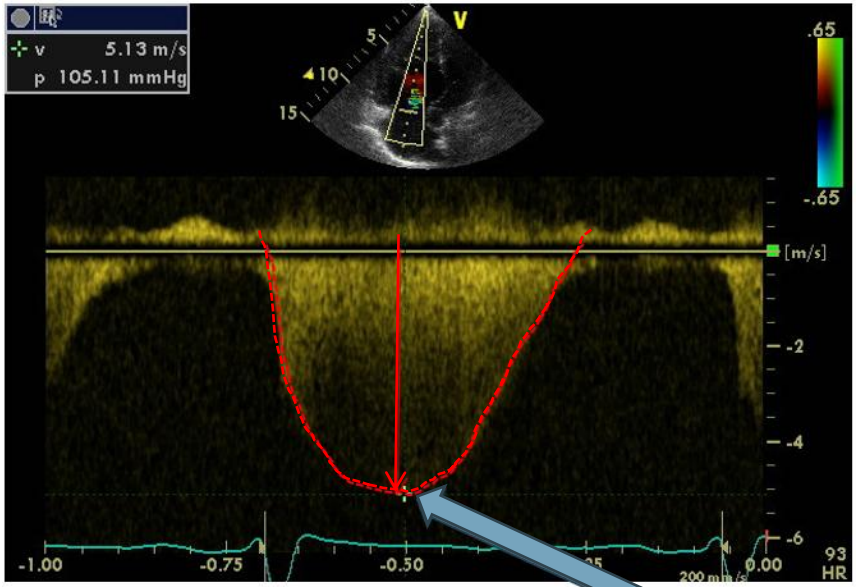
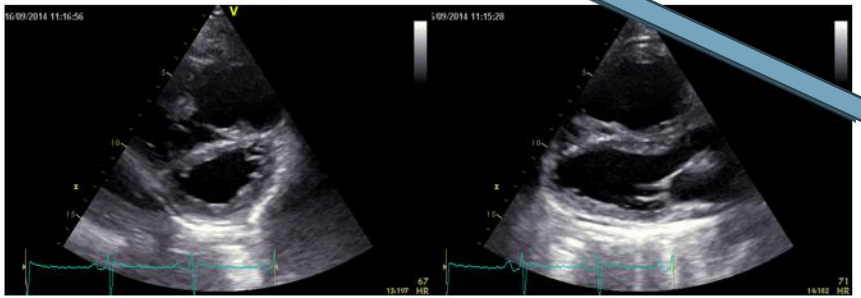


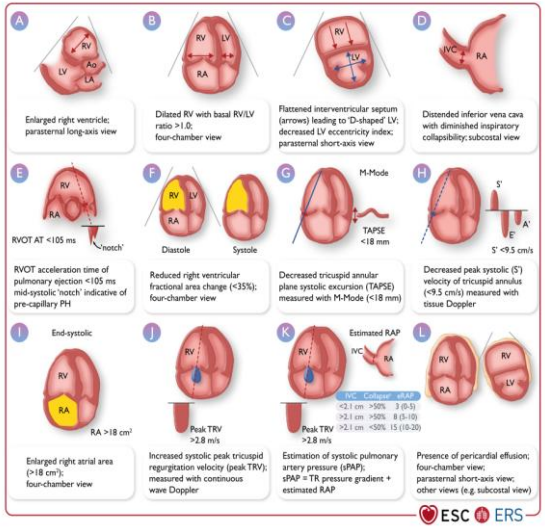
LABORATORNÍ VYŠETŘENÍ



Obrázky poskytnuty laskavostí prof. Jansy







ESC European Society of Cardiology
European Heart Journal (2022) 00, 1–114
https://doi.org/10.1093/eurheartj/ehac237
ESC/ERS GUIDELINES

2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension

Developed by the task force for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS).

Endorsed by the International Society for Heart and Lung Transplantation (ISHLT) and the European Reference Network on rare respiratory diseases (ERN-LUNG).

Tabulka 4A – Pravděpodobnost zjištění plicní hypertenze echokardiografickým vyšetřením symptomatických pacientů s podezřením na plicní hypertenzi

Maximální rychlost proudu krve při nedomykavosti trojcípé chlopně (m/s)	Přítomnost jiných „známek PH" při echokardiografickém vyšetření[a]	Možnost plicní hypertenze podle echokardiografického vyšetření
≤ 2,8 nebo neměřitelná	Ne	Nízká
≤ 2,8 nebo neměřitelná	Ano	Středně vysoká
2,9–3,4	Ne	
2,9–3,4	Ano	Vysoká
> 3,4	Není nutno provádět	

A: The ventricles	B: Pulmonary artery	C: Inferior vena cava and RA
RV/LV basal diameter/ area ratio >1.0	RVOT AT <105 ms and/or mid-systolic notching	IVC diameter >21 mm with decreased inspiratory collapse (<50% with a sniff or <20% with quiet inspiration)
Flattening of the interventricular septum (LVEI >1.1 in systole and/or diastole)	Early diastolic pulmonary regurgitation velocity >2.2 m/s	RA area (end-systole) >18 cm²
TAPSE/sPAP ratio <0.55 mm/mmHg	PA diameter >AR diameter PA diameter >25 mm	

ESC European Society of Cardiology European Heart Journal (2019) 00, 1–61 doi:10.1093/eurheartj/ehz405
ESC GUIDELINES
2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS)
The Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC)

Key massages

1. In patients presenting with **haemodynamic instability**, perform bedside TTE as a fast, immediate step to differentiate suspected high-risk PE from other acute life-threatening situations.
2. If you suspect acute PE, institute **anticoagulation therapy as soon as possible**, while the diagnostic workup is ongoing, unless the patient is bleeding or has absolute contraindications to this therapy.
3. Use recommended, **validated diagnostic algorithms for PE**, including standardized assessment of (pre-test) clinical probability and D-dimer testing. They help to avoid unnecessary, expensive, and potentially harmful imaging tests and exposure to ionizing radiation.
4. If the CTPA report suggests single subsegmental PE, consider the **possibility of a false-positive finding**. Discuss the findings again with the radiologist and/or seek a second opinion to avoid misdiagnosis, and unnecessary, potentially harmful anticoagulation treatment.
5. Confirmation of PE in a patient, without haemodynamic instability, must be followed by **further risk assessment** involving clinical findings, evaluation of the size and/or function of the RV, and laboratory biomarkers as appropriate. This information will help you to decide on the need for reperfusion treatment or monitoring for patients at elevated risk, or consider the option of early discharge and continuation of anticoagulation on an ambulatory basis for patients at low risk.

6. As soon as you diagnose (or strongly suspect) high-risk PE, select the best **reperfusion option** (systemic thrombolysis, surgical embolectomy, or catheter-directed treatment) considering the patient's risk profile, and the resources and expertise available at your hospital. For patients with intermediate–high-risk PE, reperfusion is not first-line treatment, but you should prospectively plan the management strategy with your team to have a contingency plan ready if the situation deteriorates.

7. **Prefer anticoagulation with a NOAC** over the 'traditional' LMWH–VKA regimen unless the patient has contraindication(s) to this type of drug.

8. Always remember that, with the exception of acute PE provoked by a strong transient/reversible risk factor, there is a lifelong risk of VTE recurrence after a first episode of PE. Consequently, re-examine the patient after the first 3 – 6 months of anticoagulation, weigh the benefits vs. risks of continuing treatment, **and decide on the extension and dose of anticoagulant therapy**, also considering the patient's preference. Remember to recommend regular follow-up examinations, e.g. at yearly intervals.

9. If you suspect PE in a **pregnant patient**, consider diagnostic pathways and algorithms including CTPA or V/Q lung scan, which can be used safely during pregnancy.

10. After acute PE, **patients should not be lost to follow-up**. Apart from checking for possible signs of VTE recurrence, cancer, or bleeding complications of anticoagulation, ask the patient if there is persisting or new-onset dyspnoea or functional limitation. If yes, implement a staged diagnostic workup to exclude CTEPH or chronic thromboembolic disease, and to detect/treat comorbidity or 'simple' deconditioning. Follow-up imaging is not routinely recommended in an asymptomatic patient, but it may be considered in patients with risk factors for development of CTEPH.

DĚKUJEME ZA POZORNOST

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