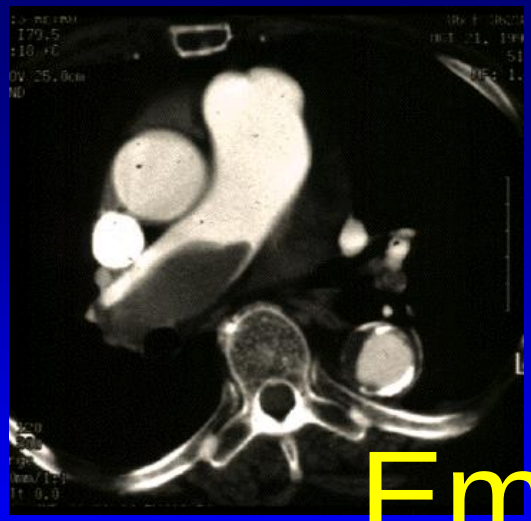

Embolie Pulmonaire Massive

Prat Gwénaël
Réanimation médicale
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Objectifs du cours

1. Définir les critères diagnostiques

2. Illustrer l'apport de l'échocardiographie dans l'aide au traitement et le suivi

Task Force Report
guidelines on diagnosis and management of acute pulmonary embolism
(Europan Heart journal 2000)

Massive

Choc et/ou hypotension

✓ PAS < 90 mmHg

ou

✓ chute PA de + 40mmHg pendant >15 min

> 2 artères lobaires

Non massive

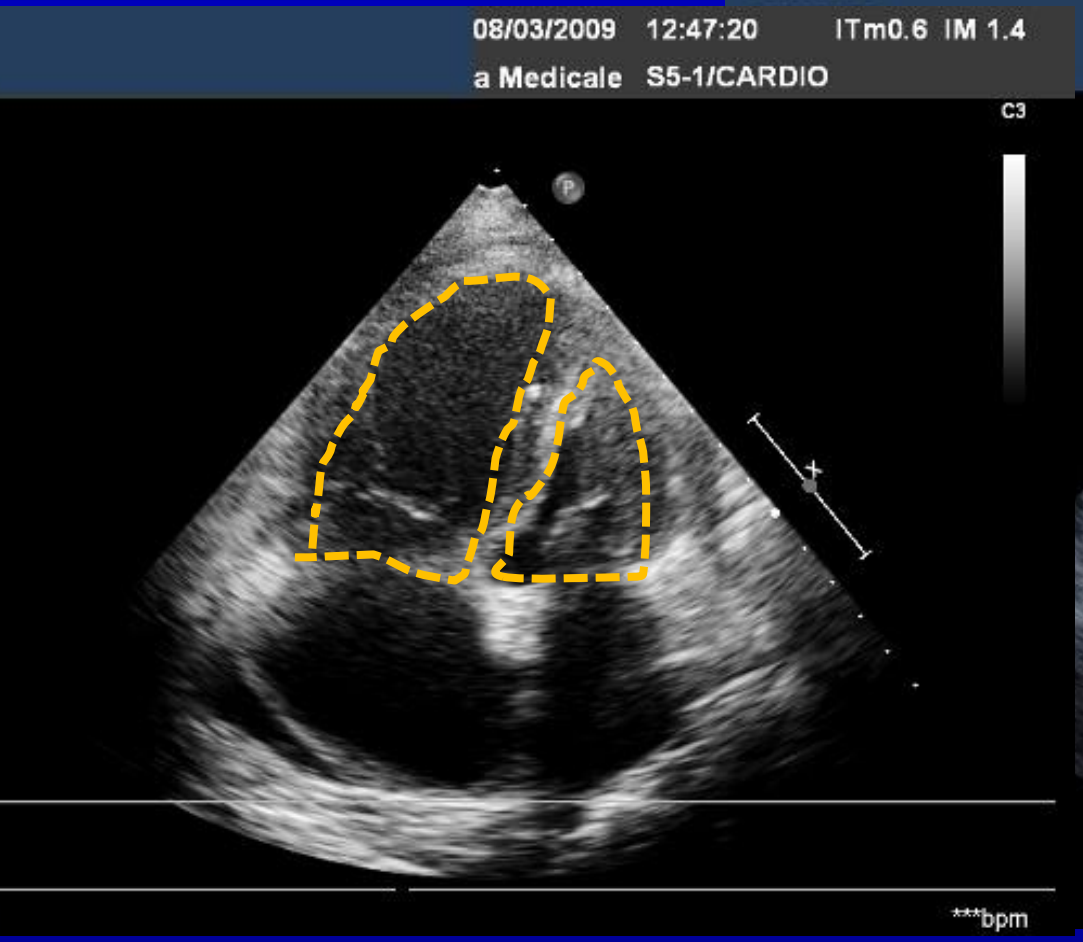
✓ Sans dysfonction VD

✓ Avec dysfonction VD (= sub-massive)

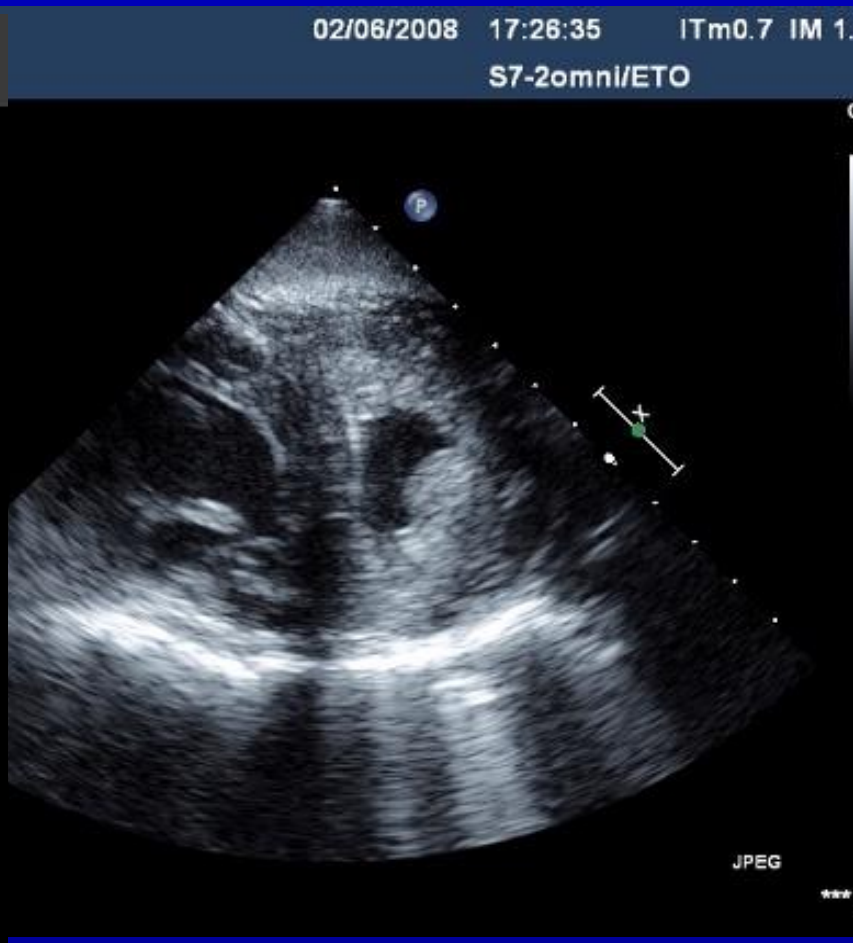
Déjà vu! Cours précédent

- Dysfonction VD : Décrire les signes échographiques
  - Définir les critères diagnostiques de CP

Surcharge diastolique

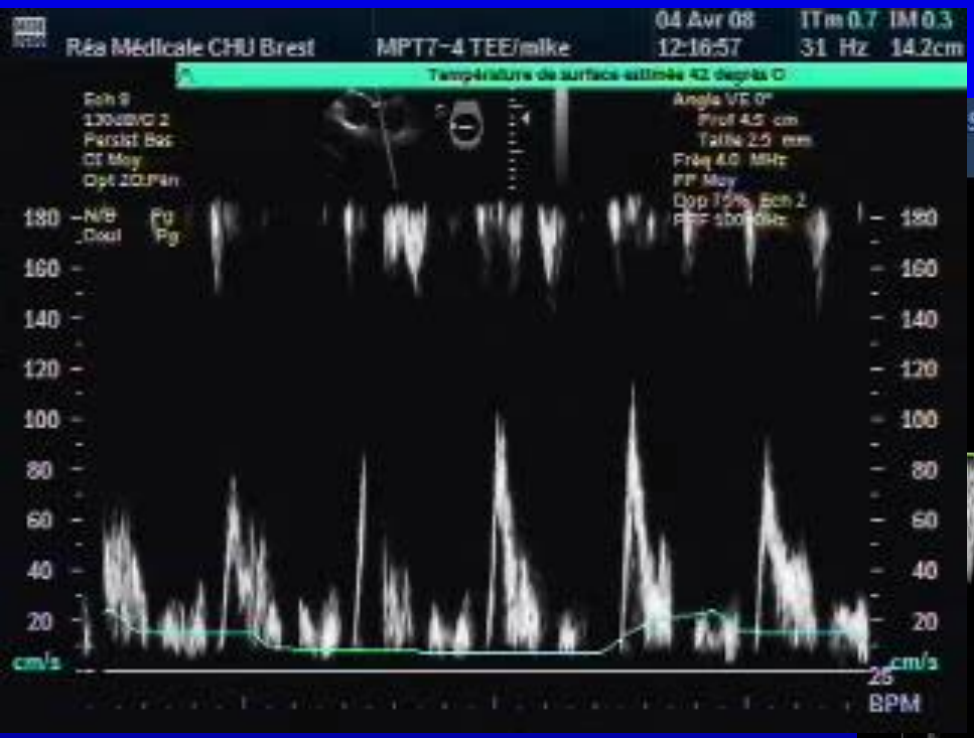


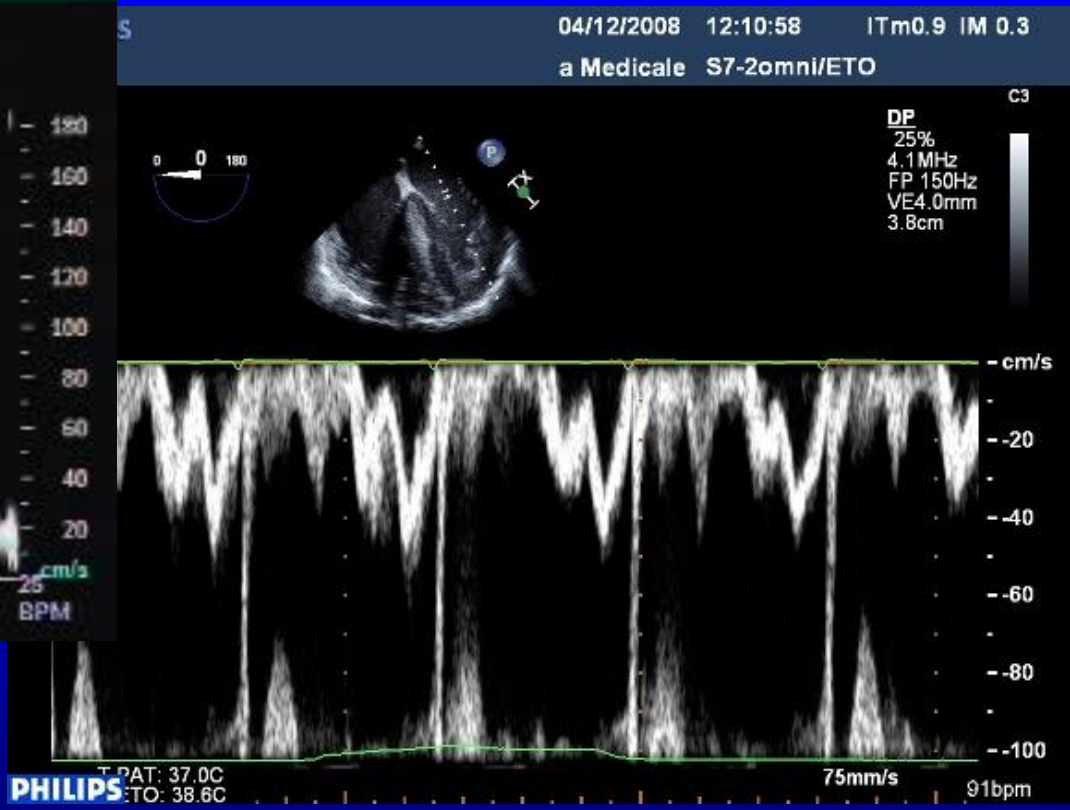
Surcharge systolique



HTAP

Perturbation fonction diastolique VG





HTAP

PAPs = 110mmHG

= 4 Vmax2 + 10

= 4 (5 x 5) + 10

CPA ou CPC?

- Histoire clinique
- Valeur de l'HTAP
  - CPA: PAPs ≈ 40-45mmHg
  - CPC: PAPS + élevée
- Épaisseur paroi du VD
  - Normale < 4mm
  - Hypertrophie modérée 4-7 mm (possible en situation aigue)
  - Hypertrophie importante > 7mm (Chronique en général)

1/ Définir les critères diagnostiques : EP

Comparison of Different Echocardiographic Indexes Secondary to Right Ventricular Obstruction in Acute Pulmonary Embolism

Nicolas Mansencal, MD, Thierry Joseph, MD, Antoine Vieillard-Baron, MD, PhD, Salah D. Qanadli, MD, PhD, Guillaume Jondeau, MD, PhD, Pascal Lacombe, MD, François Jardin, MD, and Olivier Dubourg, MD

THE AMERICAN JOURNAL OF CARDIOLOGY® VOL. 92 JULY 1, 2003

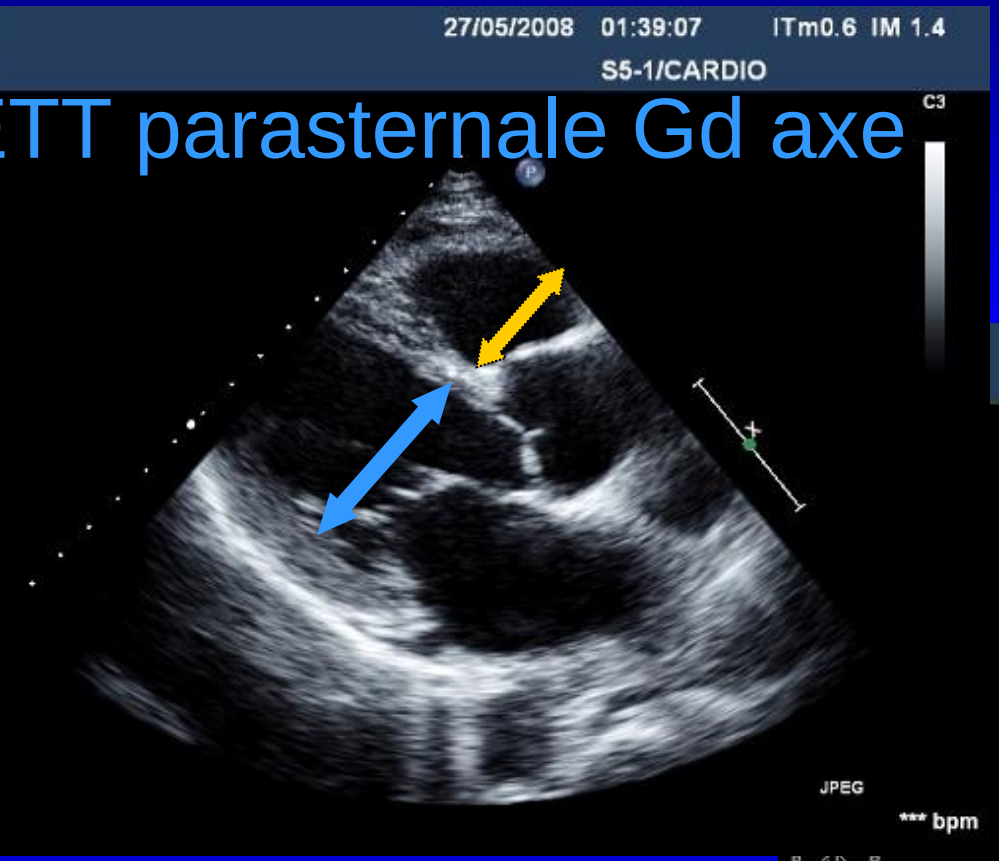
Objectif: Évaluer le caractère discriminant de différents indices échographiques pour diagnostic EP des patients arrivants en réa (ETT)

Comparaison: 1/ diamètre VD *(PSG)*

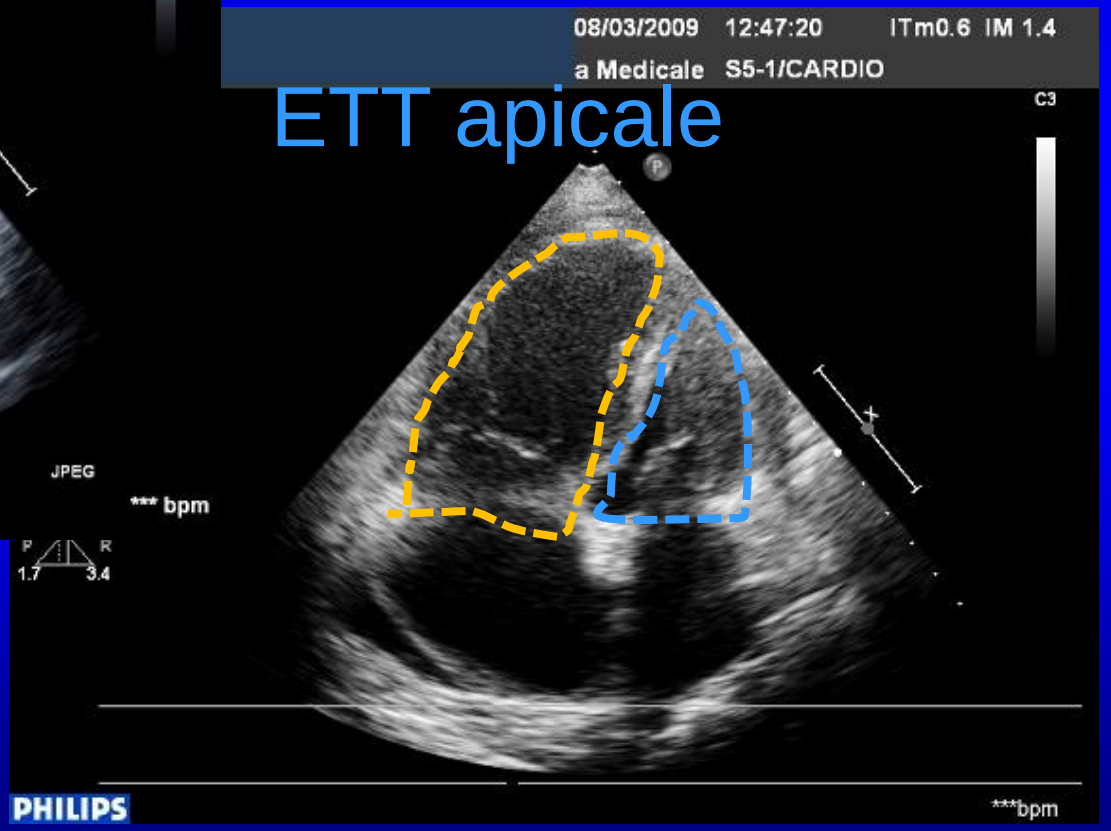
2/ ratio Ø diastolique VD-VG *(PSG)*

3/ ratio surface diastolique VD / VG *(apicale)*

4/ PAPS (artériographie)



ETT parasternale Gd axe



ETT apicale

TABLE 1 Fifty-seven Patients With Hemodynamically Stable Pulmonary Embolism (PE)

Characteristics	n = 57 (%)
Men	32 (56%)
Women	25 (44%)
Age (yrs)	56 ± 17
Previous PE	0 (0%)
Previous pulmonary disease	3 (5%)
Postoperatory	8 (14%)
Puerperium	3 (5%)
Neoplasia	4 (7%)
Symptoms	
Dyspnea	47 (82%)
Chest pain	34 (60%)
Syncope	10 (18%)
Sinus tachycardia (>100 beats/min)	19 (33%)
Systemic arterial pressure (mm Hg)	134 ± 22
Pulmonary embolism localization	
Proximal pulmonary embolism	18 (32%)
Lobar pulmonary embolism	23 (40%)
Segmental pulmonary embolism	12 (21%)
Subsegmental pulmonary embolism	4 (7%)

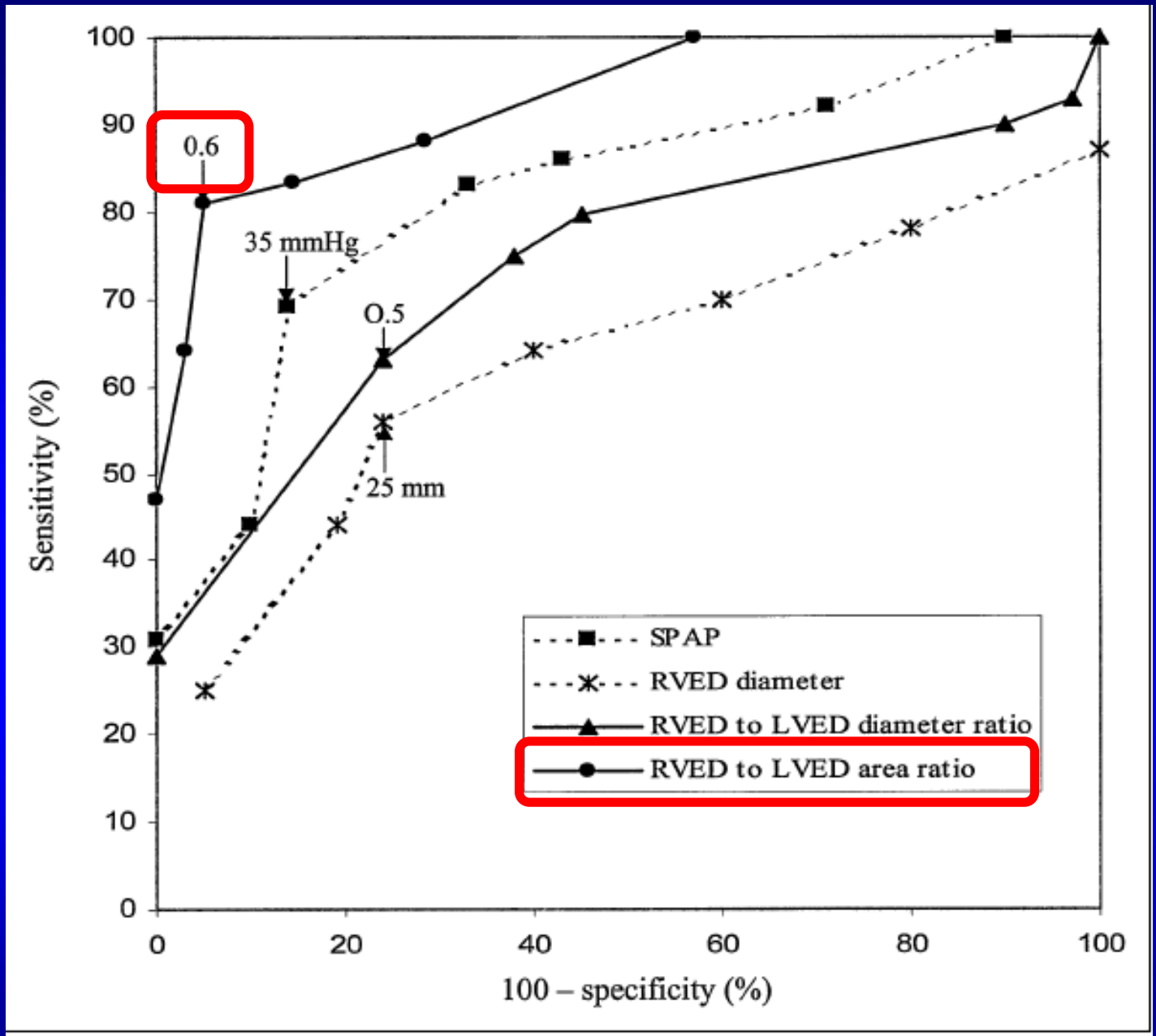


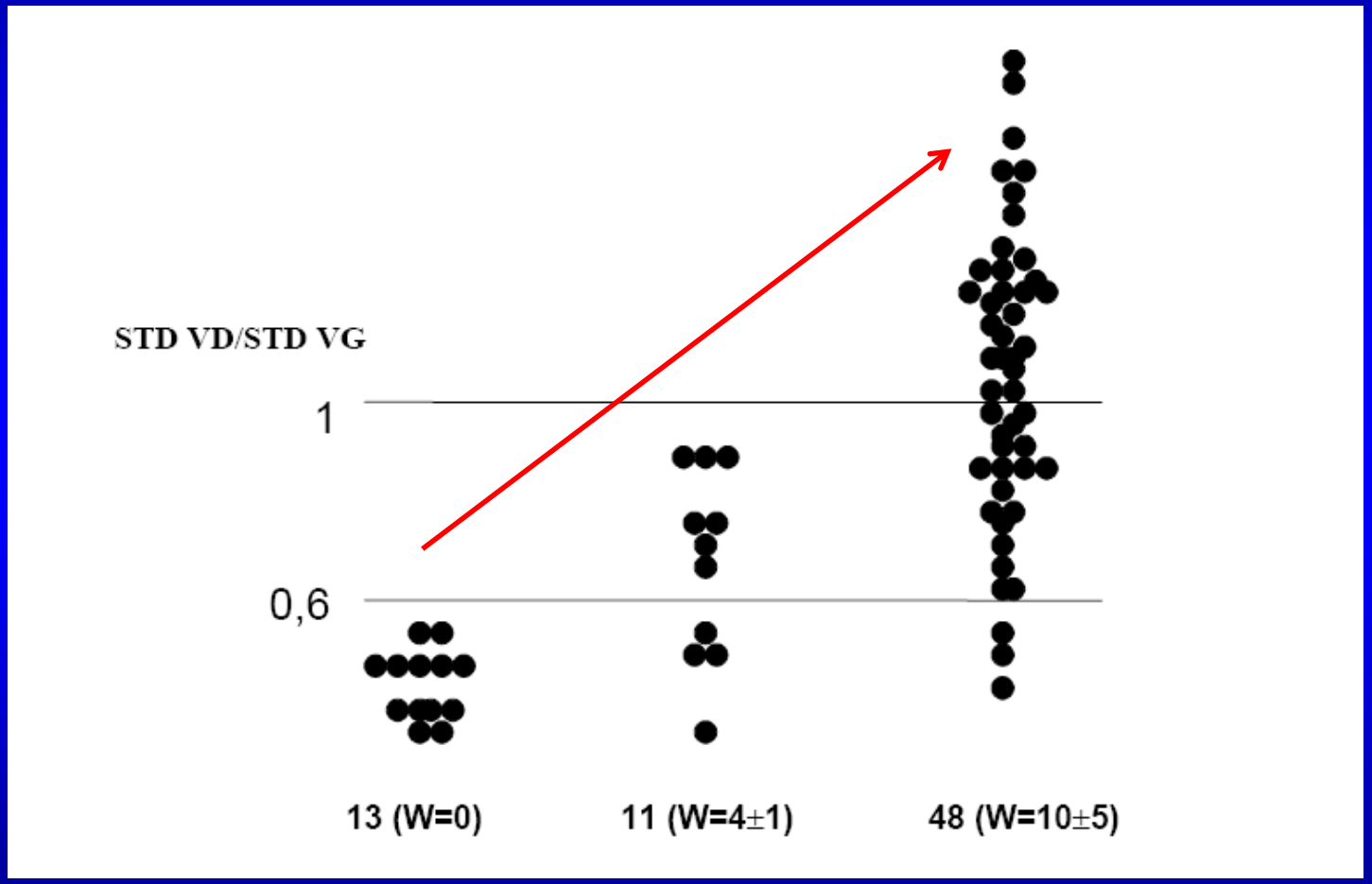
FIGURE 2. ROC curves for the RVED-to-LVED area ratio, RVED-to-LVED diameter ratio, RVED diameter, and SPAP. *Arrows* indicate cut-off values that yield the highest discriminating power.

Discussion / conclusion

- IT insuffisance tricuspidienne (84%)
- Difficulté: mesure diam VD en PSG

= >Indice le plus performant : STDVD/STDVG en apicale 4 cavités (cut off 0,6)

Jardin F, Lacombe P, Dubourg O, Delorme G, Hardy A, Beauchet A. Échographie bidimensionnelle quantitative au cours de l'embolie pulmonaire aigue. Presse Méd 1991; 20: 2085-9



EP modérée EP importante

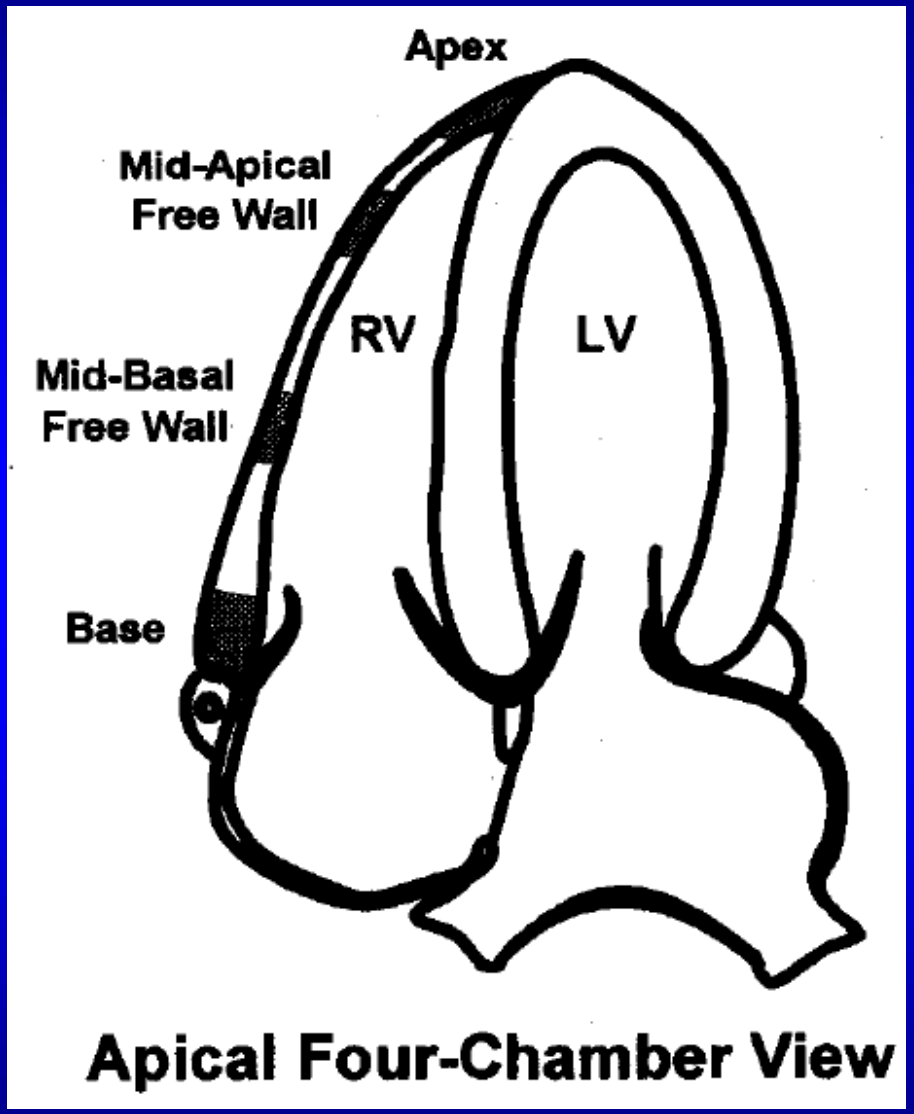
Regional Right Ventricular Dysfunction Detected by Echocardiography in Acute Pulmonary Embolism

Michael V. McConnell, MD, Scott D. Solomon, MD, Mamdouh E. Rayan, MD, Patricia C. Come, MD, Samuel Z. Goldhaber, MD, and Richard T. Lee, MD

- *Objectif:* évaluer dysfonction régionale VD par ETT
- *MM: groupe 1*: 41 patients *(cohorte)*

  14 EP, 9 HTAP primitive, 18 normaux

  groupe 2 : 85 patients *(validation)*



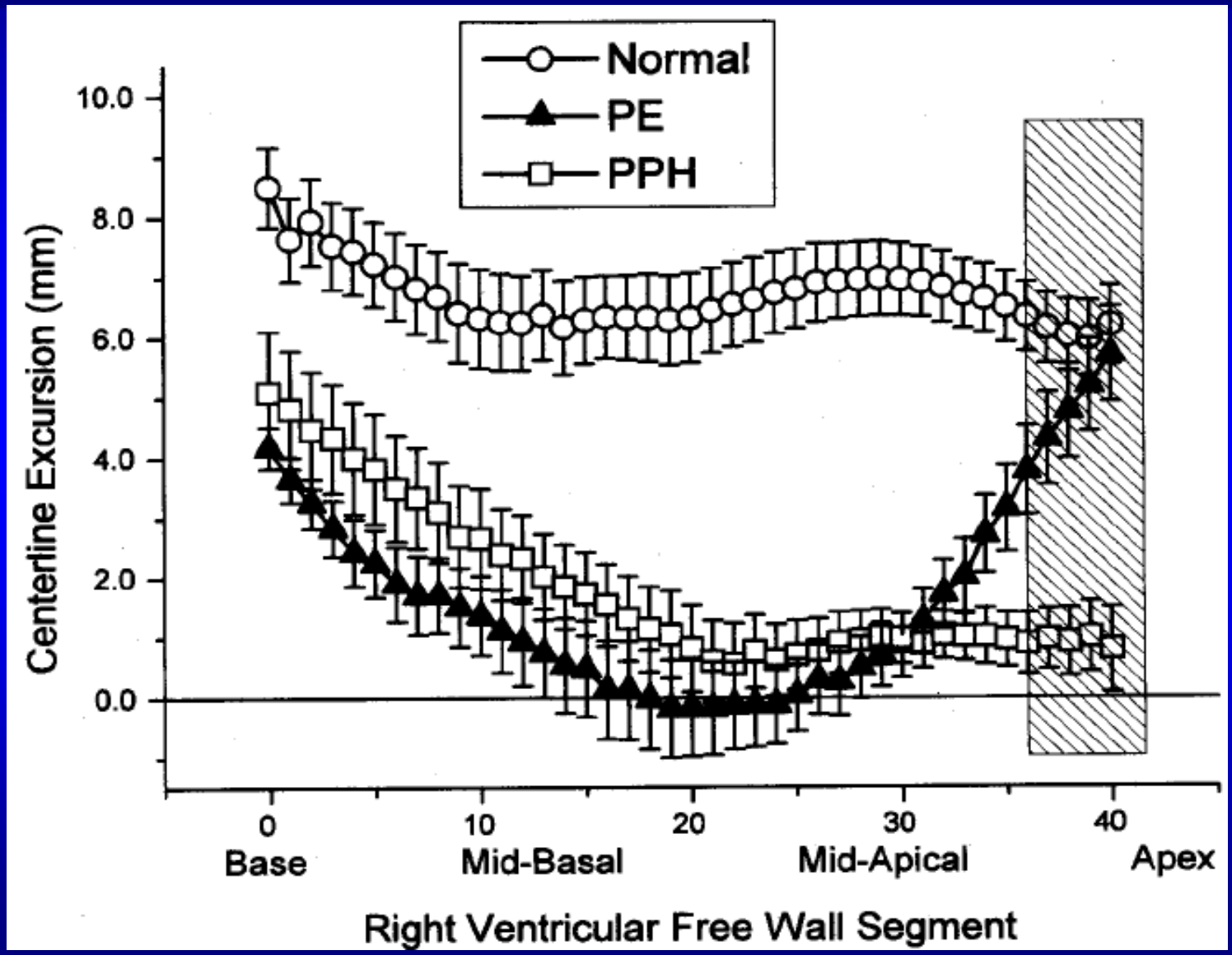
✓ Embolie Pulmonaire

dysfonction partie médiane paroi libre VD,
apex normal

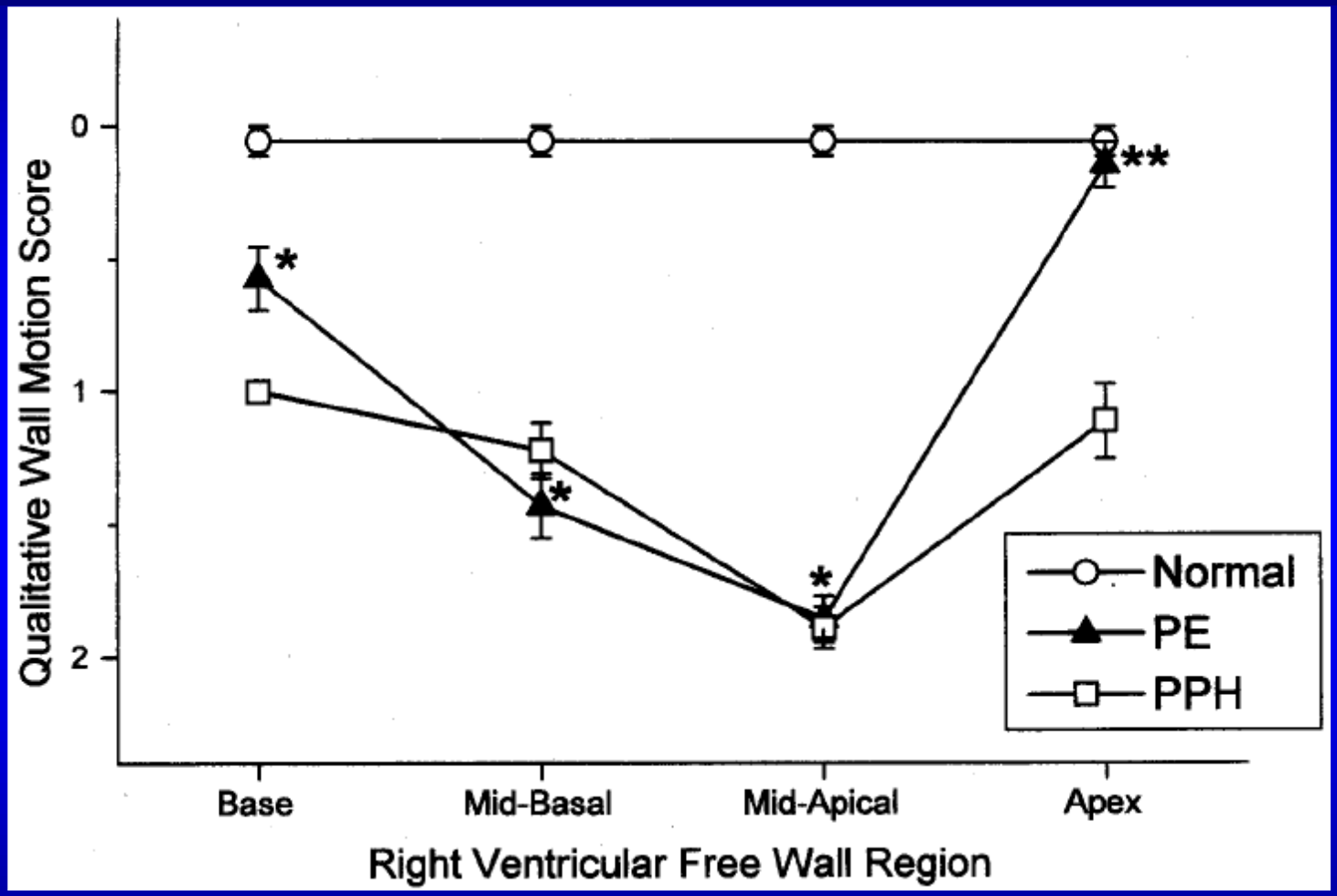
HTAP primitive

dysfonction toute la paroi

Évaluation quantitative



Évaluation qualitative



0: normal ; 1: hypokinésie ; 2: akinésie/ dyskinésie

Étude de validation

- 85 patients dont 13 EP

	EP 13	Autres 72
Echo +	10	4
Echo -	3	68

Se 77%

Sp 94%

VPP 71%

VPN 96%

Hypothèses

- VG hyperdynamique: entraîne pointe VD
- Aspect plus sphérique du VD
- Ischémie localisée

Conclusion

Peut aider au diagnostic quand suspicion d'EP

Disturbed Right Ventricular Ejection Pattern as a New Doppler Echocardiographic Sign of Acute Pulmonary Embolism

(Am J Cardiol 2002;90:507–511

Marcin Kurzyna, MD, Adam Torbicki, MD, Piotr Pruszczyk, MD,
Barbara Burakowska, MD, Anna Fijałkowska, MD, Jarosław Kober, MD,
Karina Oniszh, MD, Paweł Kuca, MD, Witold Tomkowski, MD, Janusz Burakowski, MD,
and Liliana Wawrzyńska, MD

Objectif:

compare en ETT pertinence à diagnostiquer EP entre

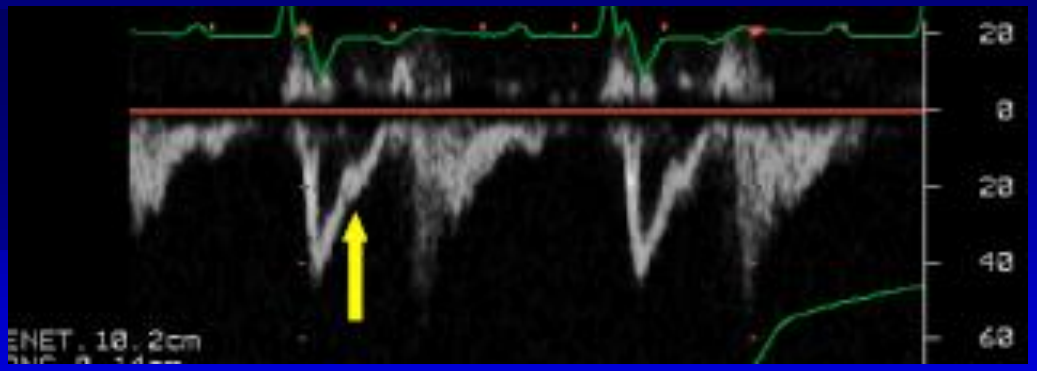
1/ 60/60sign : PAPs < 60mmHg et TA < 60msec

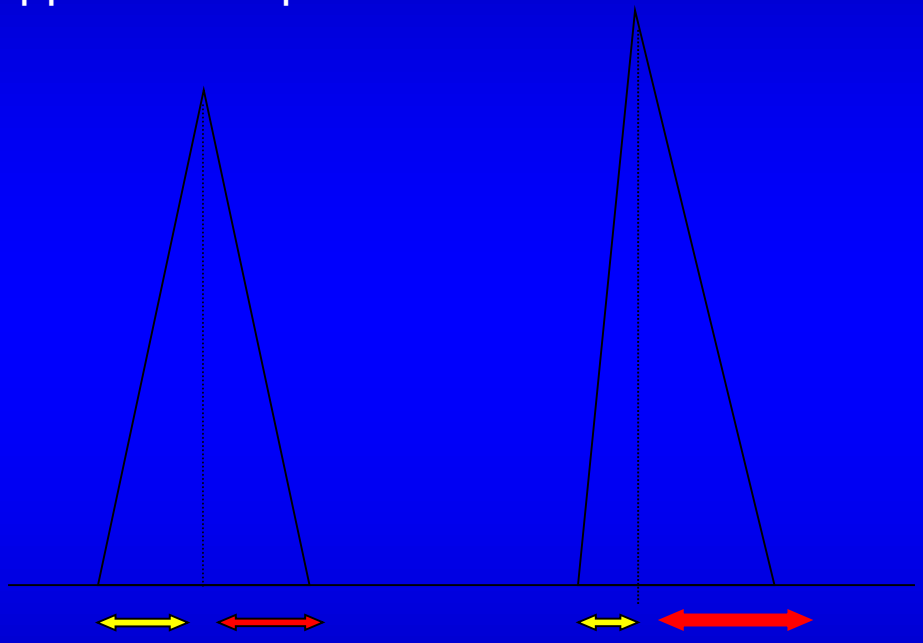
2/ Signe de Mc Connell

3/ Signes surcharges pressions droites (thrombus , ØVD ↑, aplatissement SIV, TA < 90msec et PAPs > 30mmHg

HTAP

Flux doppler Artère pulmonaire





Normal HTAP

Temps d'acceleration (TA)
Temps de décélération (Tdec)

100 patients / 67 EP

Parameter	All Patients (n = 100)	APE (+) (n = 67)	APE (−) (n = 33)	p Value
Right ventricle (mm)	31 ± 5	31 ± 6	28 ± 4	0.01
Right to left ventricle diameter ratio	0.69 ± 0.21	0.72 ± 0.22	0.63 ± 0.18	0.04
Septal flattening (%)	28%	36%	12%	0.02
Tricuspid insufficiency pressure gradient (mm Hg)	39 ± 15 (n = 86)	41 ± 15 (n = 59)	28 ± 4 (n = 27)	0.01
Acceleration time (ms)	80 ± 24	73 ± 20	95 ± 24	<0.0001
Inferior caval vein—expiratory diameter (mm)	17 ± 5	18 ± 5	16 ± 5	0.06
Collapsibility index of inferior caval vein (%)	30 ± 20	28 ± 19	36 ± 21	0.22

	Patients Without Known Previous Cardiorespiratory Diseases		
	RV Pressure Overload Criteria	60/60 Sign	(n = 46) McConnell Sign
Specificity (%)	78	100	100
Sensitivity (%)	81	25	19
Positive predictive value (%)	90	100	100
Negative predictive value (%)	64	37	35

Si TA < 60 msec : en faveur EP

Sans atcd respiratoire

Patients With Known Previous Cardiorespiratory Diseases		
RV Pressure Overload Criteria	60/60 Sign	(n = 54) McConnell Sign
21	89	100
80	26	20
65	82	100
36	40	40

Intensive Care Med (1998) 24: 429–433

ORIGINAL

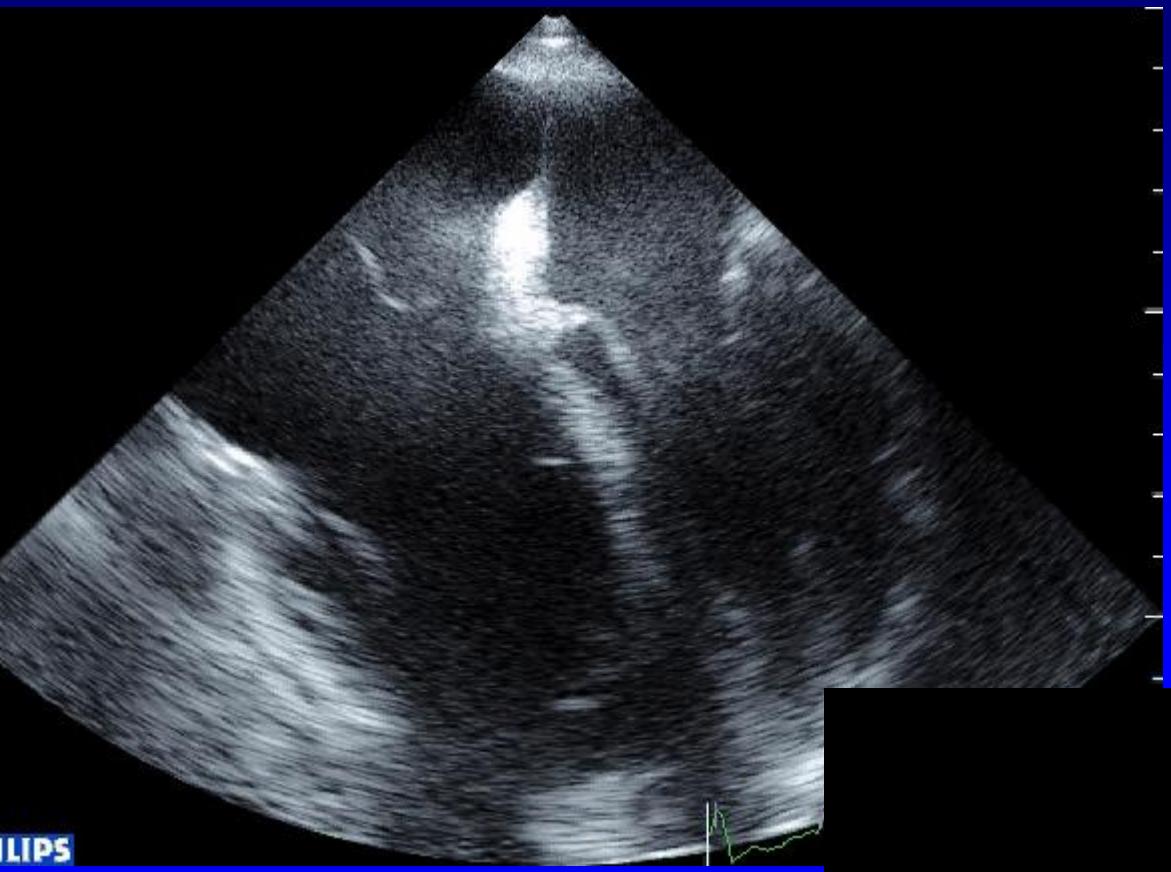
A. Vieillard-Baron
S. D. Qanadli
Y. Antakly
T. Fourme
Y. Loubières
F. Jardin
O. Dubourg

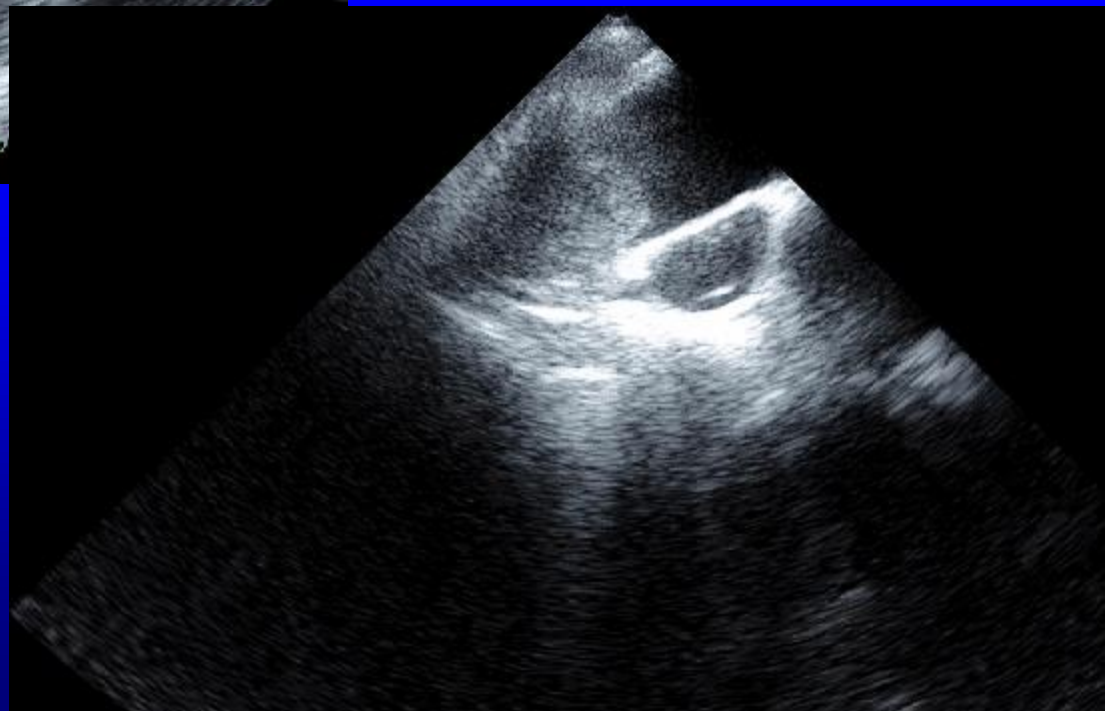
Transesophageal echocardiography for the diagnosis of pulmonary embolism with acute cor pulmonale: a comparison with radiological procedures

Objectif:
ETO permet–elle de dépister embolie / angioTDM

Type étude:
- ✓ *prospective; mai 95 à oct 96*
- ✓ *44 patients: suspicion EP*

Méthode: ETT : si CPA ETO puis TDM


ILIPS



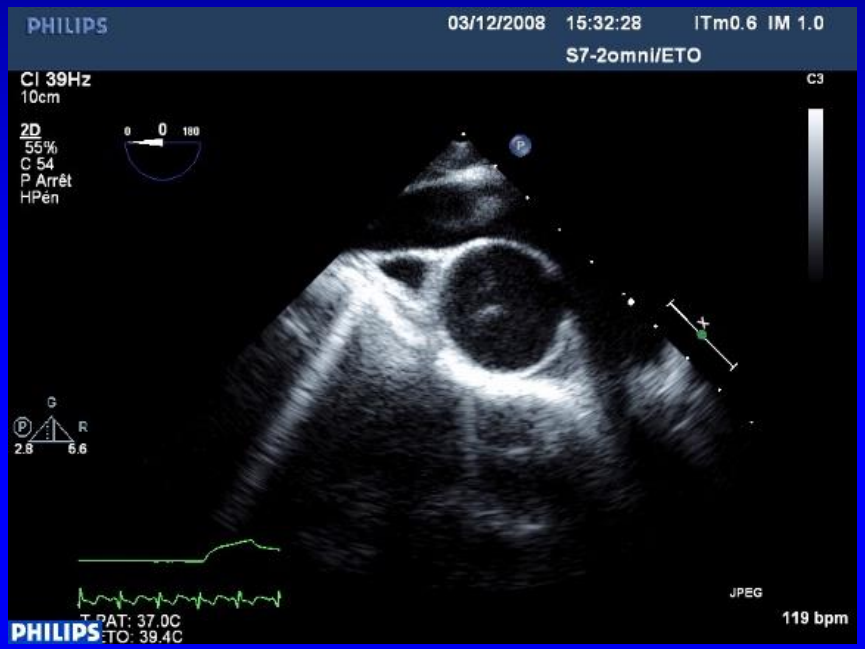
Résultats

- 44 patients
- ETT: 30 CPA (68%)

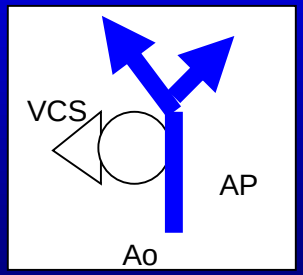
➔1 thrombus OD

- ETO: 19 patients

➔12 thrombus (63%)



- ETO : Se 84%, Sp 84% pour EP proximale
- ETO : Se 92%, Sp86% pour EP AP droite



Conclusion

- ETO: permet diagnostic en qq min
- +++ Intérêt chez patients VM+++
- ETO: si pas d'embol visualisé

  ➔n'exclut pas le diagnostic

Visualisation Thrombus dans l'AP

	Sensibilité	Spécificité
Steiner P Am J Roentgenol 1996; 167: 931-6	82	92
Wittlich N J Am Soc Echocardio 1992; 5: 515-524	97	88
Pruszczyk P Chest 1997; 112: 722-728	80	100
Krivec B Chest 1997 ; 112: 1310-1316	92	100

Triage Patients with Suspected Pulmonary Embolism in the Emergency Department Using a Portable Ultrasound Device

ECHOCARDIOGRAPHY, Volume 25, May 2008)

Nicolas Mansencal, M.D.,* Antoine Vieillard-Baron, M.D., Ph.D.,† Alain Beauchet, M.D.,‡ Jean-Christian Farcot, M.D., F.A.C.C.,* Mostafa El Hajjam, M.D.,§ Ghislaine Dufaitre, M.D.,* Dominique Brun-Ney, M.D.,‖ Pascal Lacombe, M.D.,‡ François Jardin, M.D.,† and Olivier Dubourg, M.D., F.A.C.C., F.E.S.C.*

- Étude prospective / 103 patients
- *Objectif:*

évaluer intérêt écho portable pour diagnostic EP aux urgences

- *Comparaison* :

ETT VS ETT + echodoppler mb infs

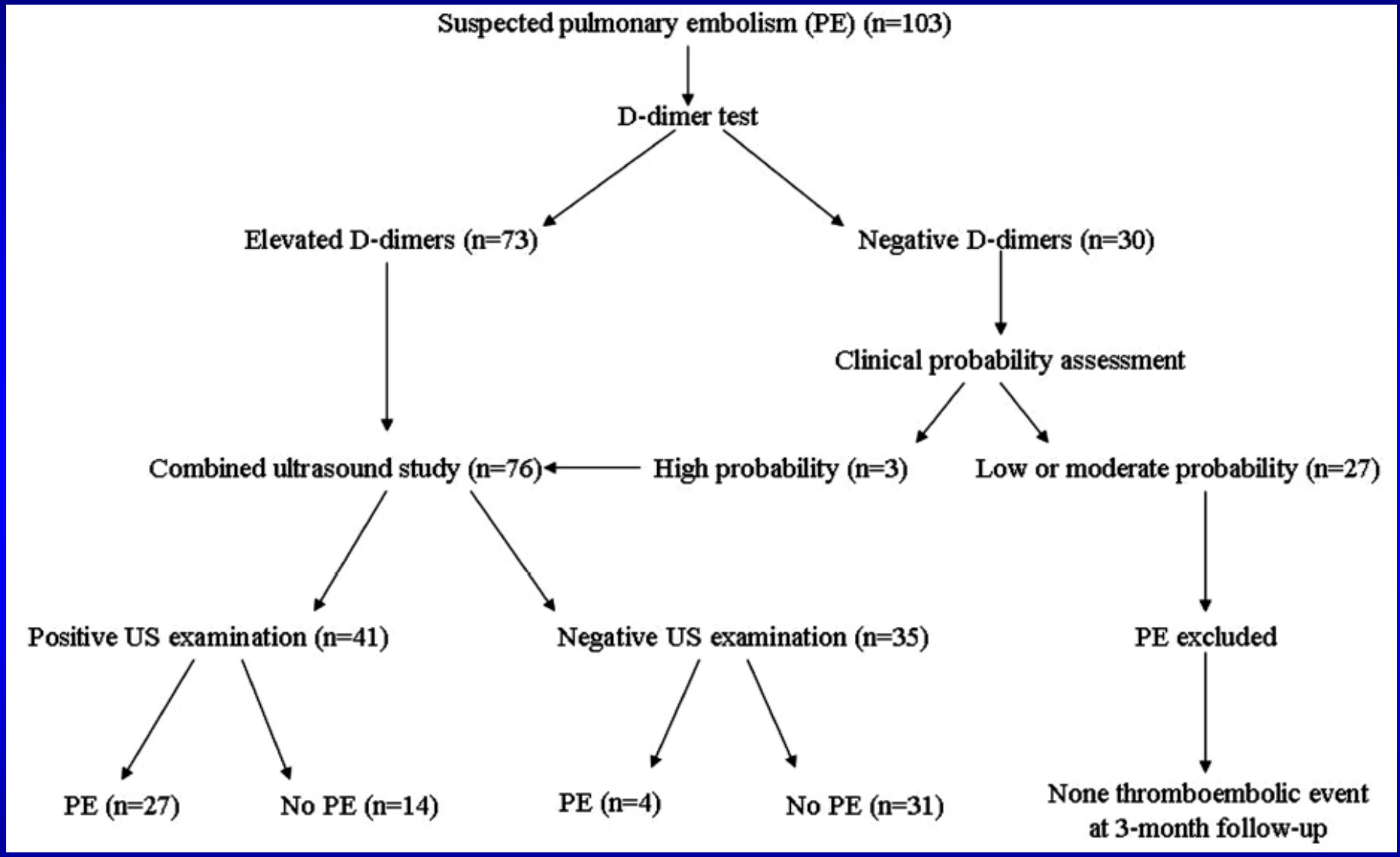

Suspected pulmonary embolism (PE) (n=103)
D-dimer test
Elevated D-dimers (n=73)
Negative D-dimers (n=30)
Clinical probability assessment
Combined ultrasound study (n=76)
High probability (n=3)
Low or moderate probability (n=27)
Positive US examination (n=41)
Negative US examination (n=35)
PE excluded
PE (n=27)
No PE (n=14)
PE (n=4)
No PE (n=31)
None thromboembolic event at 3-month follow-up

TABLE I

Characteristics of Seventy-Six Patients with Suspected Pulmonary Embolism*

	Patients with PE n = 31	Patients without PE n = 45	P-Valu
Women	16 (52)	28 (62)	NS
Age (years)	56 ± 19	68 ± 13	0.03
Previous pulmonary embolism	6 (19)	8 (18)	NS
Previous pulmonary disease	2 (6)	6 (13)	NS
Main symptom			
Dyspnea	18 (58)	27 (60)	NS
Chest pain	13 (42)	18 (40)	NS
Syncope	3 (10)	7 (16)	NS
Pretest clinical probability score			
Low clinical probability	6 (19.3)	23 (51)	0.005
Moderate clinical probability	15 (48.4)	16 (36)	NS
High clinical probability	10 (32.3)	6 (13)	0.04
Systemic arterial pressure (mmHg)	127 ± 16	129 ± 19	NS
D-dimers			
Values (ng/ml)	2366 ± 1284	1307 ± 1447	0.008
Negative level (normal <500 ng/ml)	0 (0)	3 (7)	NS
Troponin I (μg/l)	0.43 ± 0.55	0.57 ± 1.13	0.009
Arterial blood gas			
PaO2 (mmHg)	70 ± 12	71 ± 20	NS
PaCO2 (mmHg)	34 ± 6	32 ± 4	NS
Metabolic acidosis (base deficit >5 mEq/l)	7 (23)	13 (29)	NS
ECG			
Sinus tachycardia (>100 beats/mn)	12 (39)	22 (49)	NS
Normal	11 (35)	8 (18)	NS

TABLE II

Accuracy of Echocardiography, Venous Ultrasonography, and Combined Strategy in Emergency Department

	Sensitivity (% (CI))	Specificity (% (CI))	Positive Predictive Value (% (CI))	Negative Predictive Value (% (CI))
All patients				
Echocardiography alone	55 (36–73)	69 (53–82)	55 (36–73)	69 (53–82)
Venous ultrasonography alone	58 (39–75)	93 (82–99)	86 (64–97)	76 (63–87)
Ultrasound combined strategy	87 (70–96)	69 (53–82)	66 (49–80)	89 (73–97)
Patients with chest pain				
Echocardiography alone	31 (9–61)	72 (47–90)	44 (14–79)	59 (36–79)
Venous ultrasonography alone	54 (25–81)	94 (73–99)	88 (47–99)	74 (52–90)
Ultrasound combined strategy	77 (46–95)	72 (47–90)	67 (38–88)	81 (54–96)
Patients with dyspnea				
Echocardiography alone	72 (47–90)	67 (46–83)	59 (36–79)	78 (56–93)
Venous ultrasonography alone	61 (36–83)	93 (76–99)	85 (55–98)	78 (60–91)
Ultrasound combined strategy	94 (73–99)	67 (46–83)	65 (46–83)	95 (74–99)

Conclusion

« Une stratégie combinée associant

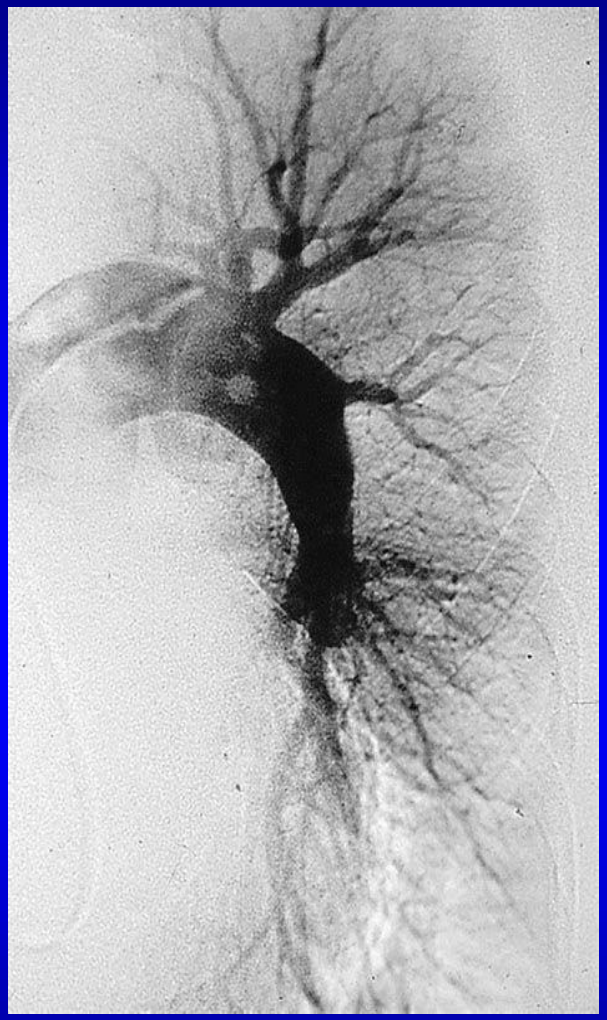
ETT + echodoppler

chez patient dyspnéique aux urgences,

méthode simple et fiable pour dépister ou éliminer EP »

A retenir:
Diagnostic échographique EP

- Rapport STDVD/STDVG >0,6
- Dyskinésie septale
- Épaisseur VD < 5mm
- Signe de Mc Connell
- PAPs < 60mmHg
- TA < 60msec (incisure méso systolique)
- Thrombus échodoppler veineux
- Visualisation d’un thrombus intracardiaque



2/ Illustrer l'apport de l'échocardiographie dans l'aide au traitement et le suivi

Intensive Care Med (2001) 27: 1481–1486
DOI 10.1007/s001340101032

ORIGINAL

Antoine Vieillard-Baron
Bernard Page
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Acute cor pulmonale in massive pulmonary embolism: incidence, echocardiographic pattern, clinical implications and recovery rate

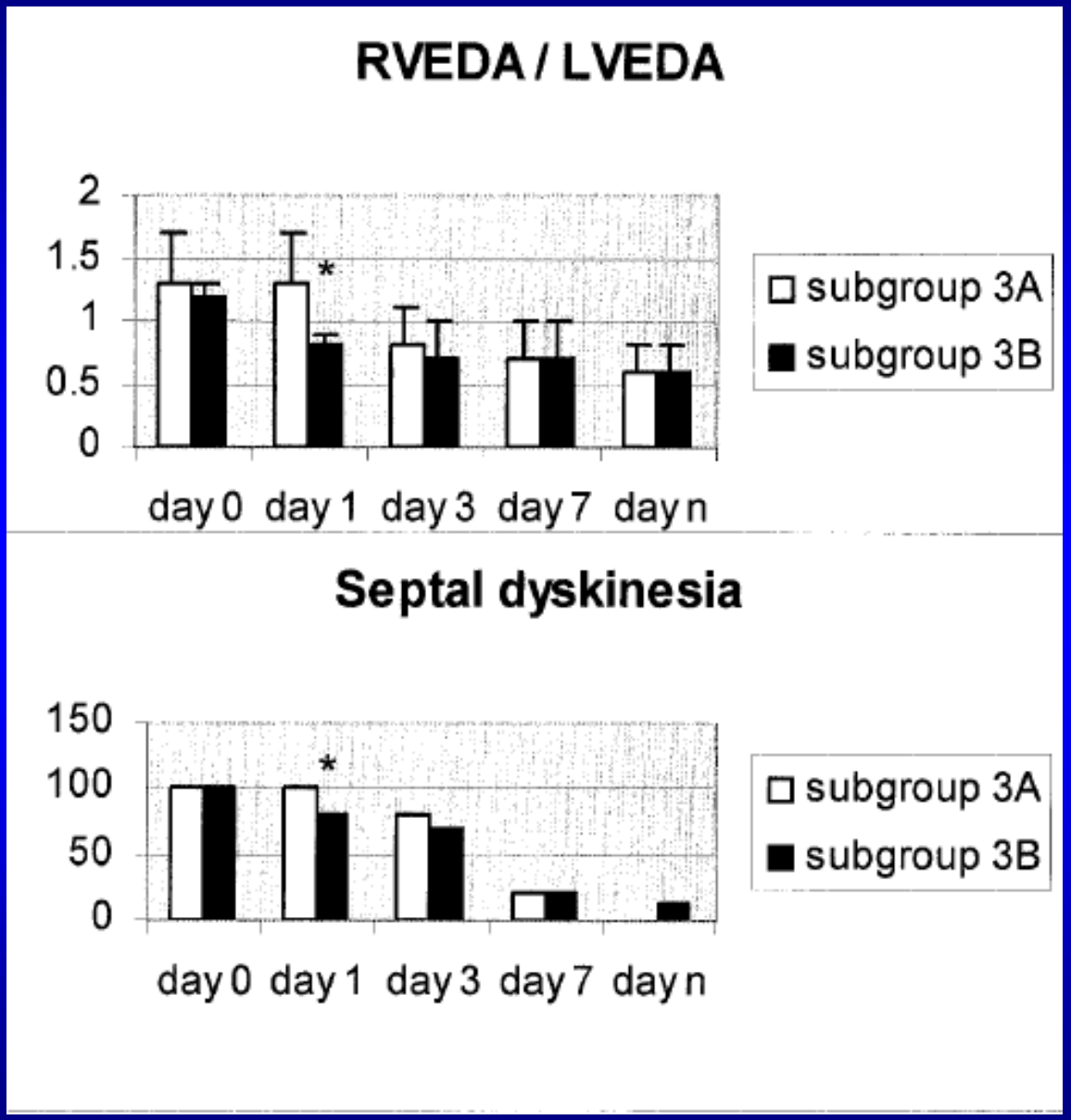
- *Objectif:* incidence CPA si EP Massive (ETT)
- *Type:* rétrospectif sur 10 ans
- *MM:* 161 patients; EP massive (> 2 artères lobaires)
- *ETT:* CPA si ratio VD/VG > 0,6 et dyskinésie septale

CPA 98/161 63%	*Groupe 1*	*Groupe 2*	*Groupe 3*	
			3A	*3B*
Patients	32	32	20	14
Hémodynamique	stable	Inotrope / acidose -	Inotrope / acidose +	
traitement	Héparine	Héparine	Héparine	Héparine + thrombolyse
Mortalité (%)	3	3	60	57

	R ($n = 76$)	Death ($n = 22$)	Univariate p	Multivariate p	OR [CI]
Age	61 ± 15	75 ± 14	0.0002	NS	
HR (beat/min)	100 ± 15	110 ± 20	NS		
CF (%)	59	96	0.001	NS	
RVEDA/LVEDA	1.1 ± 0.3	1.6 ± 0.5	0.0001	NS	
Acidosis (%)	24	91	2^{E-12}	0.001	4.2^{E6} [6^{E-262}/3^{E-274}]

	Group 1	Group 2	Group 3
RVEDA/LVEDA	1 ± 0.2	1.2 ± 0.3	1.4 ± 0.5[a]
HR (beat/min	88 ± 12	104 ± 14[a]	109 ± 18[a]
SI (Doppler, cm^3/m^2)	31 ± 8	22 ± 8[a]	18 ± 8[a]
CI (Doppler, l/min per m^2)	2.7 ± 0.7	2.2 ± 0.7	1.9 ± 0.9[a]
RVEDA (cm^2/m^2)	15.8 ± 4.5	15.7 ± 3.3	15.9 ± 4
RVESA (cm^2/m^2)	11.7 ± 3.8	11.7 ± 3.1	12 ± 3.3
RVFAC (%)	25 ± 15	25 ± 11	23 ± 11
LVEDV (cm^3/m^2)	54.3 ± 11.3	41.3 ± 15.2[a]	37.7 ± 19.7[a]
LVESV (cm^3/m^2)	26.4 ± 13.1	17.4 ± 7.8	19.8 ± 13.4
LVEF (%)	54 ± 15	58 ± 11	49 ± 12
PAP_S (mmHg)	55 ± 18	48 ± 15	48 ± 16
E/A mitral	0.85 ± 0.26	0.80 ± 0.21	0.78 ± 0.20
IVC diam (mm)	16 ± 6	19 ± 6	19 ± 5

[a] $p < 0.05$ versus subgroup 1



Groupe 3A: héparine

Groupe 3B: héparine + thrombolyse

Conclusion

- Aucune mesure discriminante pour décider du type de traitement
- Acidose métabolique meilleur marqueur de sévérité

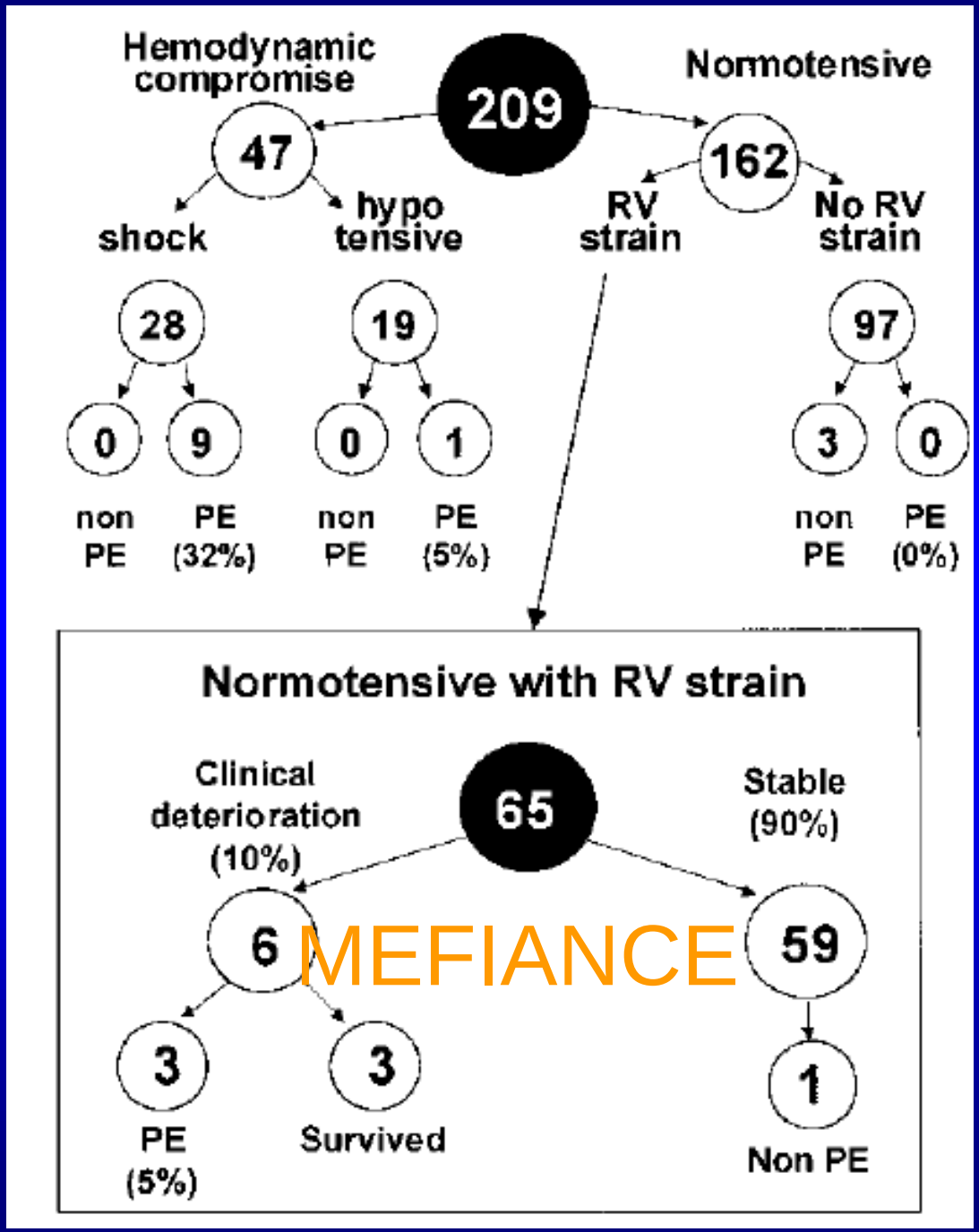
Short-Term Clinical Outcome of Patients With Acute Pulmonary Embolism, Normal Blood Pressure, and Echocardiographic Right Ventricular Dysfunction

Stefano Grifoni, Iacopo Olivotto, Paolo Cecchini, Filippo Pieralli, Alberto Camaiti, Gennaro Santoro, Alberto Conti, Giancarlo Agnelli and Giancarlo Berni

Circulation 2000;101;2817-2822

- Objectif: évaluer devenir des patients EP normotendu mais avec dysfonction VD
- MM: 209 patients EP documentées, dysfonction VD = dilatation VD, septum paradoxal, HTAP

Circulation


Hemodynamic compromise
209
Normotensive
47
162
shock
hypo tensive
RV strain
No RV strain
28
19
97
0
9
0
1
3
0
non PE
PE (32%)
non PE
PE (5%)
non PE
PE (0%)
Normotensive with RV strain
Clinical deterioration (10%)
65
Stable (90%)
6
59
3
3
1
PE (5%)
Survived
Non PE
MEFIANCE

Intérêt de l’échographie

-pour dépister EP sub-massive

- Pour assurer surveillance rapprochée

-quand au traitement?

Free-Floating Thrombi in the Right Heart : Diagnosis, Management, and Prognostic Indexes in 38 Consecutive Patients

Ludovic Chartier, Jérôme Béra, Maxence Delomez, Philippe Asseman, Jean-Paul Beregi, Jean-Jacques Bauchart, Henri Warembourg and Claude Théry

Circulation 1999;99:2779-2783

- Objectif: évaluer effet de la prise en charge
- MM: 38 patients sur 12 ans
- Résultats: mortalité globale 44%

Chirurgie : 8/17

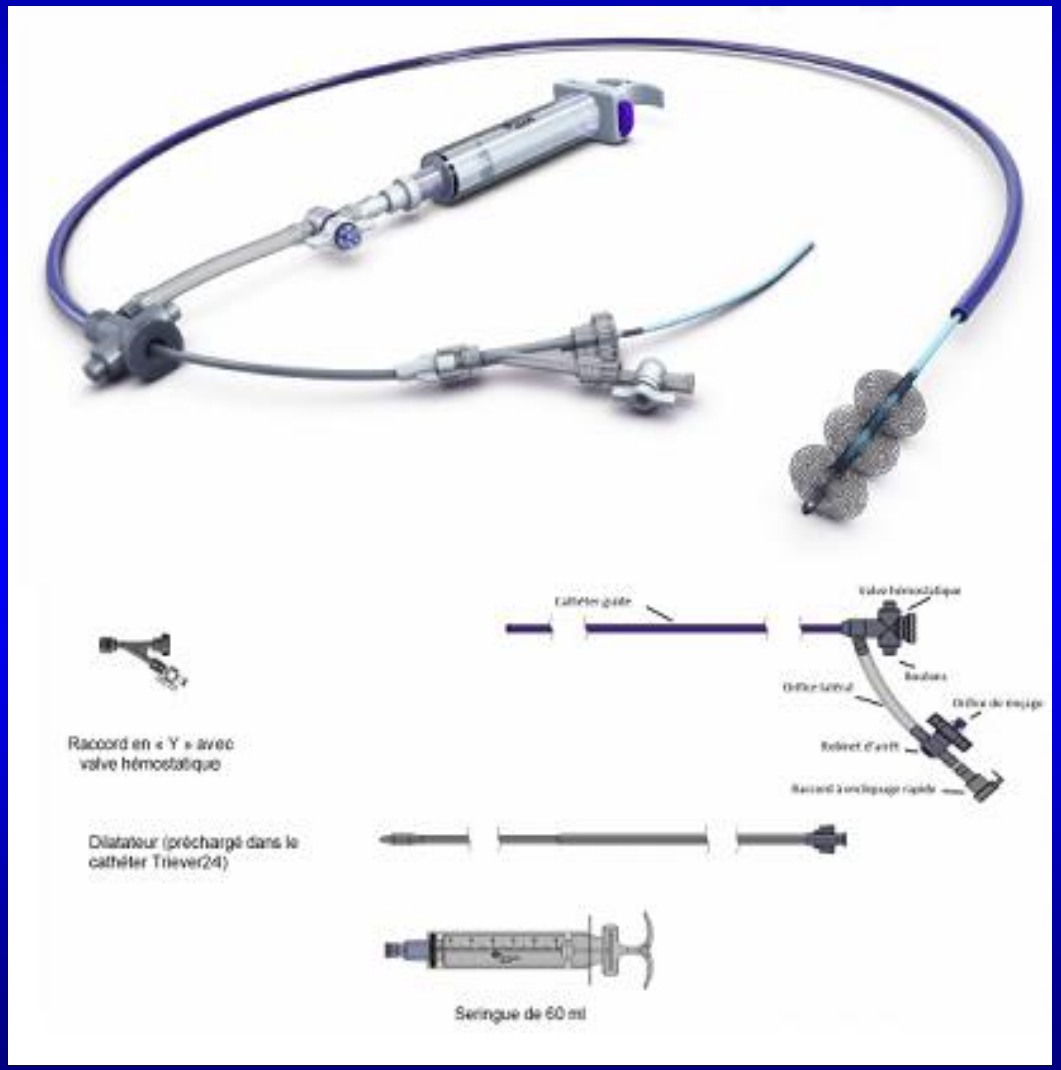
Thrombolyse: 2/17

Héparine: 5/8

Percutané: 2/4

- Conclusion: choix du traitement n'influe pas sur mortalité

Thromboaspiration per cutanée



Flow retriever

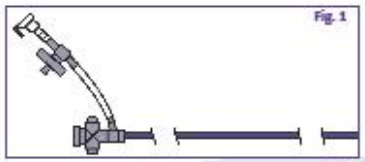
marque INARI

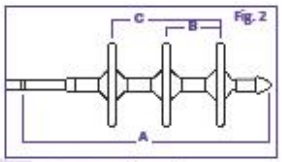
Thromboaspiration per cutanée

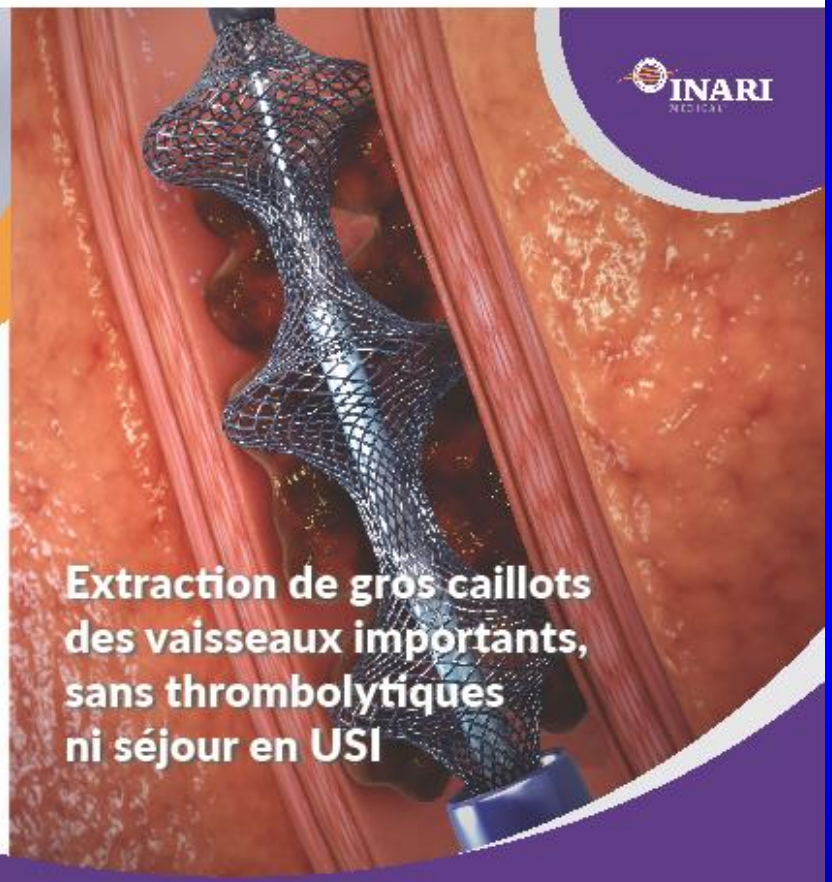
FlowTriever®
CARACTÉRISTIQUES TECHNIQUES

Modèle (Fig. 1)	Longueur utile	Diamètre externe	Diamètre interne	Guide
Triever16® (25-101)	113 cm	16 Fr (5,3 mm)	13 Fr (4,3 mm)	0,035 pouce (0,9 mm)
Triever20® (21-101)	90 cm	20 Fr (6,5 mm)	17 Fr (5,7 mm)	0,035 pouce (0,9 mm)
Triever24® (22-101)	90 cm	24 Fr (7,6 mm)	21 Fr (6,9 mm)	0,035 pouce (0,9 mm)

FlowTriever® Taille de cathéter et modèle (Fig. 2)	Plage de diamètre du vaisseau	Guide	Longueur utile	Diamètre externe	Distance de l'extrémité au repère proximal (A)	Espacement entre les disques (B)	Longueur du segment à disques (C)
S : 10-101	6-10 mm	0,035 pouce (0,9 mm)	115 cm	12 Fr (4 mm)	59 mm	13 mm	26 mm
M : 10-102	11-14 mm				60 mm	12 mm	26 mm
L : 10-103	15-18 mm				62 mm	14 mm	27 mm
XL : 10-104	19-25 mm				75 mm	19 mm	38 mm







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Mise en garde : Selon la loi fédérale américaine, ce dispositif ne peut être vendu que par un médecin ou sur son ordonnance. Se reporter au mode d'emploi pour toutes les indications d'utilisation, contre-indications, avertissements et précautions.

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- Traitement: thrombolyse
- Place de la thromboaspiration ? CI a la thrombolyse
- Chirurgie: c'est compliqué et invasif

EUROPEAN SOCIETY OF CARDIOLOGY®

European Heart Journal (2008) **29**, 2276–2315
doi:10.1093/eurheartj/ehn310

Guidelines on the diagnosis and management of acute pulmonary embolism

The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC)

Table 4 Principal markers useful for risk stratification in acute pulmonary embolism

Clinical markers	Shock
	Hypotension[a]
Markers of RV dysfunction	RV dilatation, hypokinesis or pressure overload on echocardiography
	RV dilatation on spiral computed tomography
	BNP or NT-proBNP elevation
	Elevated right heart pressure at RHC
Markers of myocardial injury	Cardiac troponin T or I positive[b]

BNP = brain natriuretic peptide; NT-proBNP = N-terminal proBNP; RHC = right heart catheterization; RV = right ventricle.

[a]Defined as a systolic blood pressure <90 mmHg or a pressure drop of ≥ 40 mmHg for >15 min if not caused by new-onset arrhythmia, hypovolaemia or sepsis.

[b]Heart-type fatty acid binding protein (H-FABP) is an emerging marker in this category, but still requires confirmation.

Table 5 Risk stratification according to expected pulmonary embolism-related early mortality rate

PE-related early MORTALITY RISK		RISK MARKERS			Potential treatment implications
		CLINICAL (shock or hypotension)	RV dysfunction	Myocardial injury	
HIGH >15%		+	(+)[a]	(+)[a]	Thrombolysis or embolectomy
NON HIGH	Intermediate 3–15%	–	+	+	Hospital admission
			+	–	
			–	+	
	Low <1%	–	–	–	Early discharge or home treatment

[a]In the presence of shock or hypotension it is not necessary to confirm RV dysfunction/injury to classify as high risk of PE-related early mortality.

European Heart Journal (2014) **35**, 3033–3080
doi:10.1093/eurheartj/ehu283

ESC GUIDELINES

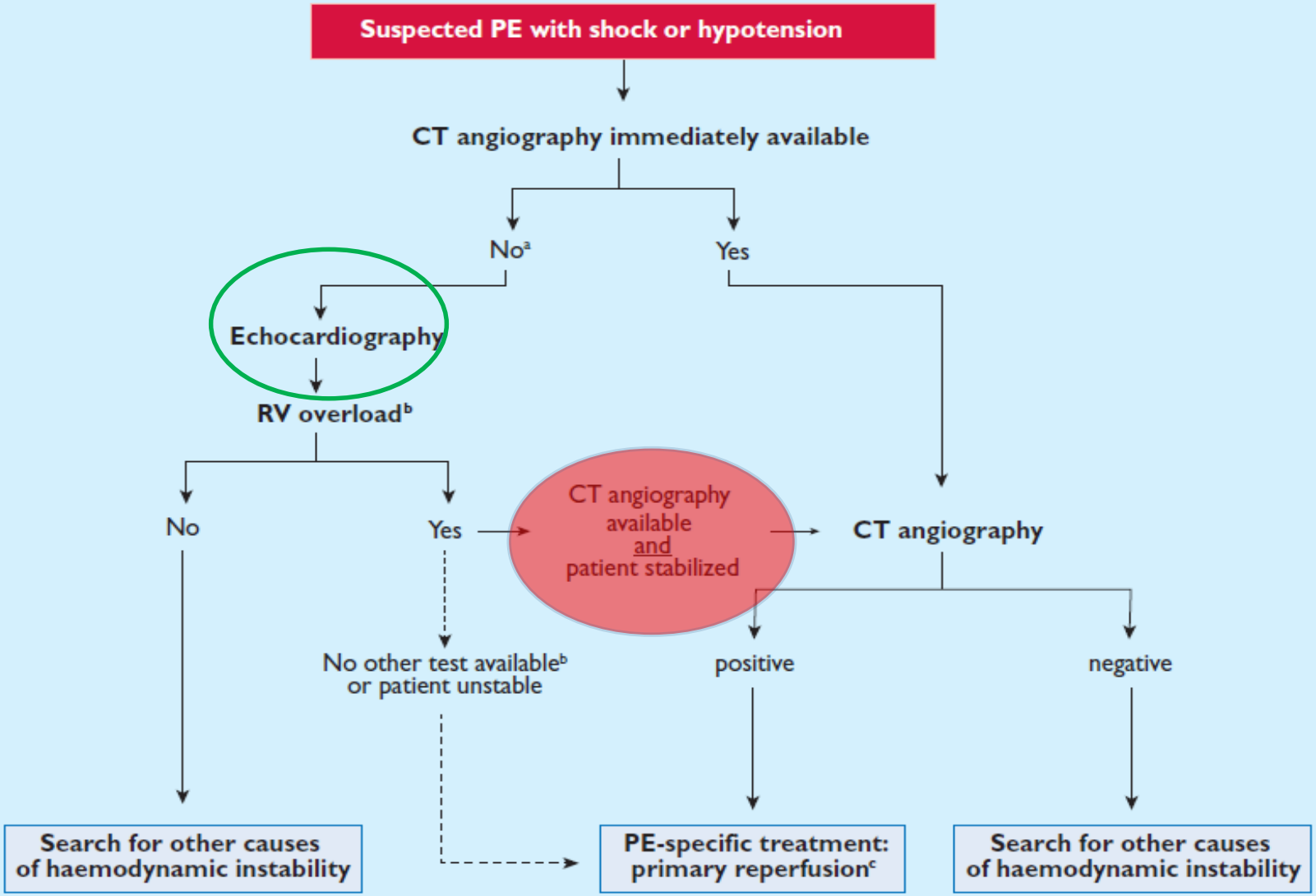
2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism

The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC)

Endorsed by the European Respiratory Society (ERS)

Table 9 Classification of patients with acute PE based on early mortality risk

Early mortality risk		Risk parameters and scores			
		Shock or hypotension	PESI class III-V or sPESI ≥1[a]	Signs of RV dysfunction on an imaging test[b]	Cardiac laboratory biomarkers[c]
High		+	(+)[d]	+	(+)[d]
Intermediate	Intermediate–high	–	+	Both positive	
	Intermediate–low	–	+	Either one (or none) positive[e]	
Low		–	–	Assessment optional; if assessed, both negative[e]	



CT = computed tomographic; PE = pulmonary embolism; RV = right ventricular.

[a]Includes the cases in which the patient's condition is so critical that it only allows bedside diagnostic tests.

[b]Apart from the diagnosis of RV dysfunction, bedside transthoracic echocardiography may, in some cases, directly confirm PE by visualizing mobile thrombi in the right heart chambers. Ancillary bedside imaging tests include transoesophageal echocardiography, which may detect emboli in the pulmonary artery and its main branches, and bilateral compression venous ultrasonography, which may confirm deep vein thrombosis and thus be of help in emergency management decisions.

[c]Thrombolysis; alternatively, surgical embolectomy or catheter-directed treatment (Section 5).

Recommendations for diagnosis

Recommendations	Class[a]	Level[b]	Ref[c]
Suspected PE with shock or hypotension			
In suspected high-risk PE, as indicated by the presence of shock or hypotension, emergency CT angiography or bedside transthoracic echocardiography (depending on availability and clinical circumstances) is recommended for diagnostic purposes.	I	C	182
In patients with suspected high-risk PE and signs of RV dysfunction who are too unstable to undergo confirmatory CT angiography, bedside search for venous and/or pulmonary artery thrombi with CUS and/or TOE may be considered to further support the diagnosis of PE, if immediately available.	IIb	C	188, 189
Pulmonary angiography may			

Table 6 Validated diagnostic criteria (based on non-invasive tests) for diagnosing PE in patients without shock or hypotension according to clinical probability

Diagnostic criterion	Clinical probability of PE				
	Low	Intermediate	High	PE unlikely	PE likely
Exclusion of PE					
D-dimer					
Negative result, highly sensitive assay	+	+	–	+	–
Negative result, moderately sensitive assay	+	±	–	+	–
Chest CT angiography					
Normal multidetector CT alone	+	+	±	+	±
V/Q scan					
Normal perfusion lung scan	+	+	+	+	+
Non-diagnostic lung scan[a] and negative proximal CUS	+	±	–	+	–
Confirmation of PE					
Chest CT angiogram showing at least segmental PE	+	+	+	+	+
High probability V/Q scan	+	+	+	+	+
CUS showing proximal DVT	+	+	+	+	+

Recommendations for acute phase treatment

Recommendations	Class[a]	Level[b]	Ref[c]
PE with shock or hypotension (high-risk)			
It is recommended that intravenous anticoagulation with UFH be initiated without delay in patients with high-risk PE.	I	C	
Thrombolytic therapy is recommended.	I	B	168
Surgical pulmonary embolectomy is recommended for patients in whom thrombolysis is contraindicated or has failed.[d]	I	C	313
Percutaneous catheter-directed treatment should be considered as an alternative to surgical pulmonary embolectomy for patients in whom full-dose systemic thrombolysis is contraindicated or has failed.[d]	IIa	C	

The 'Ten Commandments' for ESC guidelines on pulmonary embolism

(1) Assessment of haemodynamic stability of the patient and clinical probability of pulmonary embolism (PE) is the basis of all diagnostic strategies.

(2) Appropriate use of D-dimer testing may reduce the need for unnecessary imaging and irradiation.

(3) While computed tomographic angiography plays a key role in diagnostic algorithms, ventilation-perfusion (V/Q), compression venous ultrasonography, and emergency echocardiography may be helpful in management decisions.

(4) Clinical assessment may identify patients at high risk (with shock or hypotension) requiring primary revascularization therapy, and patients at low risk of early death despite confirmed PE (Pulmonary Embolism Severity Index classes I or II) who can be considered for early discharge if appropriately anti-coagulated.

(5) Among the remaining patients, those with signs of both right-ventricular overload and positive humoral biomarkers (troponin, BNP) represent an intermediate- to high-risk group which should be monitored, as they may require rescue reperfusion therapy if clinical signs of haemodynamic decompensation appear.

(6) Primary reperfusion treatment, particularly systemic thrombolysis, is the treatment of choice for patients with high-risk PE.

(7) Surgical pulmonary embolectomy and percutaneous catheter-directed treatment are alternative methods of primary and rescue reperfusion treatment.

(8) For most cases of acute PE without haemodynamic compromise, low-molecular-weight heparin or fondaparinux is the initial treatment of choice. Unfractionated heparin should be used in haemodynamically unstable patients and those with severe renal dysfunction.

(9) The non-vitamin-K-dependent oral anticoagulants (NOACs; direct inhibitors of factor Xa or thrombin) are non-inferior in terms of efficacy and possibly safer, particularly in terms of major bleeding, than the standard anticoagulation regimen consisting of heparin followed by a vitamin K antagonist.

(10) Management of PE in patients with cancer and in pregnancy, duration of anti-coagulation after initial episode, and management of patients with persisting symptoms and suspected/confirmed chronic thromboembolic pulmonary hypertension after PE all require specific considerations and may need to follow separate recommendations—see 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)

Authors/Task Force Members: Stavros V. Konstantinides* (Chairperson) (Germany/Greece), Guy Meyer* (Co-Chairperson) (France), Cecilia Becattini (Italy), Héctor Bueno (Spain), Geert-Jan Geersing (Netherlands), Veli-Pekka Harjola (Finland), Menno V. Huisman (Netherlands), Marc Humbert[1] (France), Catriona Sian Jennings (United Kingdom), David Jiménez (Spain), Nils Kucher (Switzerland), Irene Marthe Lang (Austria), Mareike Lankeit (Germany), Roberto Lorusso (Netherlands), Lucia Mazzolai (Switzerland), Nicolas Meneveau (France), Fionnuala Ní Áinle (Ireland), Paolo Prandoni (Italy), Piotr Pruszczyk (Poland), Marc Righini (Switzerland), Adam Torbicki (Poland), Eric Van Belle (France), and José Luis Zamorano (Spain)

2.2 What is new in the 2019 Guidelines?

2.2.1 New/revised concepts in 2019

Diagnosis
D-dimer cut-off values adjusted for age or clinical probability can be used as an alternative to the fixed cut-off value.
Updated information is provided on the radiation dosage when using CTPA and a lung scan to diagnose PE (*Table 6*).
Risk assessment
A clear definition of haemodynamic instability and high-risk PE is provided (*Table 4*).
Assessment of PE severity and early PE-related risk is recommended, in addition to comorbidity/aggravating conditions and overall death risk.
A clear word of caution that RV dysfunction may be present, and affect early outcomes, in patients at 'low risk' based on clinical risk scores.
Treatment in the acute phase
Thoroughly revised section on haemodynamic and respiratory support for high-risk PE (Section 6.1).
A dedicated management algorithm is proposed for high-risk PE (*Supplementary Figure 1*).
NOACs are recommended as the first choice for anticoagulation treatment in a patient eligible for NOACs; VKAs are an alternative to NOACs.
The risk-adjusted management algorithm (*Figure 6*) was revised to take into consideration clinical PE severity, aggravating conditions/comorbidity, and the presence of RV dysfunction.

2.2.2 Changes in recommendations 2014–19

Recommendations	2014	2019
Rescue thrombolytic therapy is recommended for patients who deteriorate haemodynamically.	IIa	I
Surgical embolectomy or catheter-directed treatment should be considered as alternatives to rescue thrombolytic therapy for patients who deteriorate haemodynamically.	IIb	IIa
D-dimer measurement and clinical prediction rules should be considered to rule out PE during pregnancy or the post-partum period.	IIb	IIa
Further evaluation may be considered for asymptomatic PE survivors at increased risk for CTEPH.	III	IIb

2.2.3 Main new recommendations 2019

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
Treatment in the acute phase	
When oral anticoagulation is initiated in a patient with PE who is eligible for a NOAC (apixaban, dabigatran, edoxaban, or rivaroxaban), a NOAC is the recommended form of anticoagulant treatment.	I
Set-up of multidisciplinary teams for management of high-risk and selected cases of intermediate-risk PE should be considered, depending on the resources and expertise available in each hospital.	IIa
ECMO may be considered, in combination with surgical embolectomy or catheter-directed treatment, in refractory circulatory collapse or cardiac arrest.	IIb

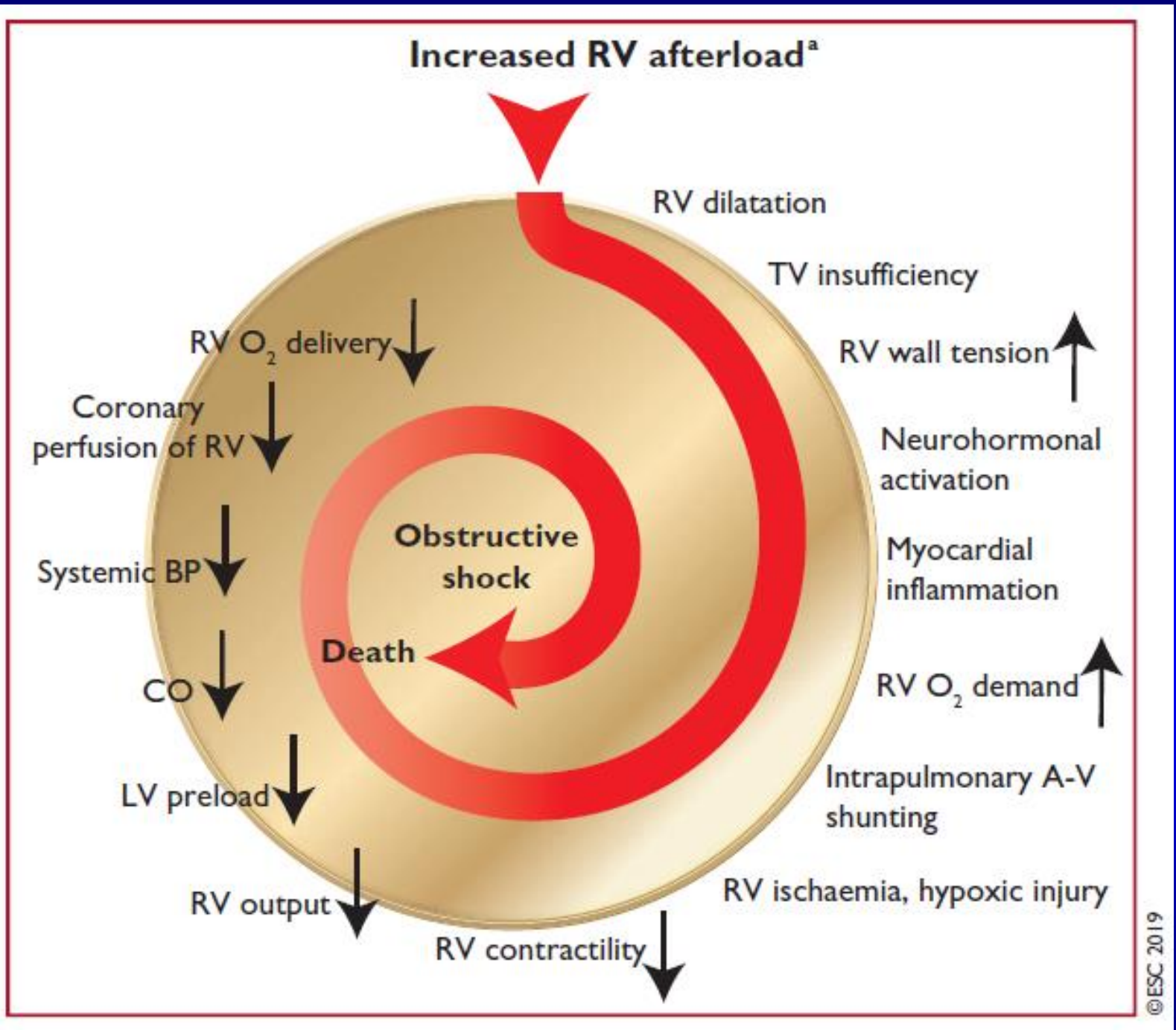

Increased RV afterload[a]
RV dilatation
TV insufficiency
RV wall tension
Neurohormonal activation
Myocardial inflammation
RV O_2 demand
Intrapulmonary A-V shunting
RV ischaemia, hypoxic injury
RV contractility
RV output
LV preload
CO
Systemic BP
Coronary perfusion of RV
RV O_2 delivery
Obstructive shock
Death
©ESC 2019

Table 4 **Definition of haemodynamic instability, which delineates acute high-risk pulmonary embolism (one of the following clinical manifestations at presentation)**

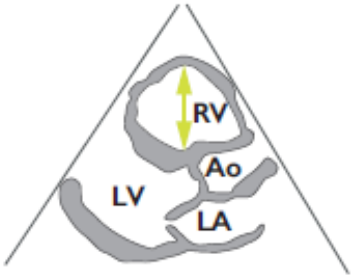
(1) Cardiac arrest	(2) Obstructive shock[68–70]	(3) Persistent hypotension
Need for cardiopulmonary resuscitation	Systolic BP < 90 mmHg or vasopressors required to achieve a BP ≥90 mmHg despite adequate filling status	Systolic BP < 90 mmHg or systolic BP drop ≥40 mmHg, lasting longer than 15 min and not caused by new-onset arrhythmia, hypovolaemia, or sepsis
	And	
	End-organ hypoperfusion (altered mental status; cold, clammy skin; oliguria/anuria; increased serum lactate)	

Table 6 Imaging tests for diagnosis of pulmonary embolism

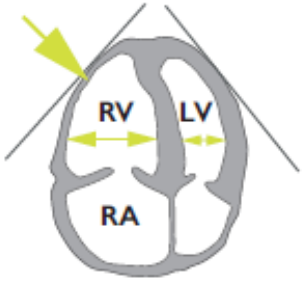
	Strengths	Weaknesses/limitations	Radiation issues[a]

CTPA	• Readily available around the clock in most centres
	• Excellent accuracy
	• Strong validation in prospective management outcome studies
	• Low rate of inconclusive results (3–5%)
	• May provide alternative diagnosis if PE excluded
	• Short acquisition time
		• Radiation exposure
		• Exposure to iodine contrast:
		  ○ limited use in iodine allergy and hyperthyroidism
		  ○ risks in pregnant and breastfeeding women
		  ○ contraindicated in severe renal failure
		• Tendency to overuse because of easy accessibility
		• Clinical relevance of CTPA diagnosis of subsegmental PE unknown
			• Radiation effective dose 3–10 mSv[b]
			• Significant radiation exposure to young female breast tissue

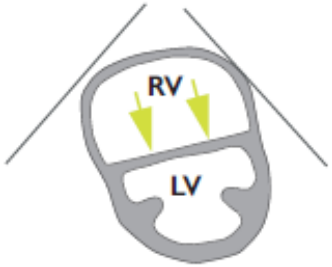
Planar V/Q scan	• Almost no contraindications • Relatively inexpensive • Strong validation in prospective management outcome studies	• Not readily available in all centres • Interobserver variability in interpretation • Results reported as likelihood ratios • Inconclusive in 50% of cases • Cannot provide alternative diagnosis if PE excluded	• Lower radiation than CTPA, effective dose ~2 mSv[b]
V/Q SPECT	• Almost no contraindications • Lowest rate of non-diagnostic tests (<3%) • High accuracy according to available data • Binary interpretation ('PE' vs. 'no PE')	• Variability of techniques • Variability of diagnostic criteria • Cannot provide alternative diagnosis if PE excluded • No validation in prospective management outcome studies	• Lower radiation than CTPA, effective dose ~2 mSv[b]
Pulmonary angiography	• Historical gold standard	• Invasive procedure • Not readily available in all centres	• Highest radiation, effective dose 10–20 mSv[b]



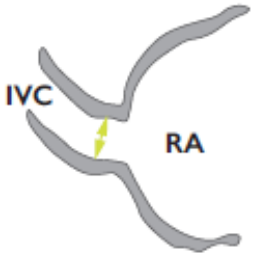
A. Enlarged right ventricle, parasternal long axis view



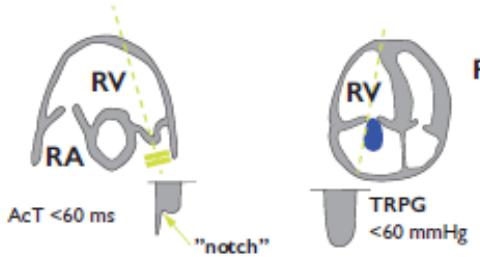
B. Dilated RV with basal RV/LV ratio >1.0, and McConnell sign (arrow), four chamber view



C. Flattened intraventricle septum (arrows) parasternal short axis view

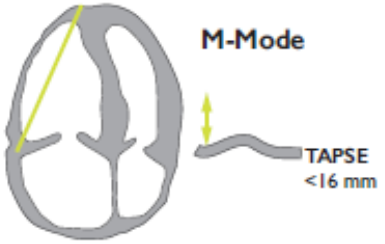


D. Distended inferior vena cava with diminished inspiratory collapsibility, subcostal view

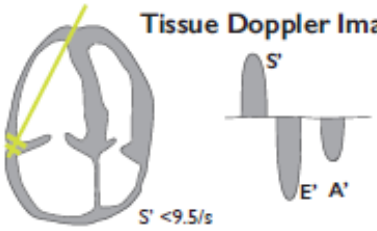


E. 60/60 sign: coexistence of acceleration time of pulmonary ejection <60 ms and midsystolic "notch" with mildy elevated (<60 mmHg) peak systolic gradient at the tricuspic valve

F. Right heart mobile thrombus detected in right heart cavities (arrow)



G. Decreased tricuspid annular plane systolic excursion (TAPSE) measured with M-Mode (<16 mm)



H. Decreased peak systolic (S') velocity of tricuspid annulus (<9.5 cm/s)

4.11 Recommendations for diagnosis

Recommendations	Class[a]	Level[b]
Suspected PE with haemodynamic instability		
In suspected high-risk PE, as indicated by the presence of haemodynamic instability, bedside echocardiography or emergency CTPA (depending on availability and clinical circumstances) is recommended for diagnosis.[169]	I	C
Lower-limb CUS		
It is recommended to accept the diagnosis of VTE (and PE) if a CUS shows a proximal DVT in a patient with clinical suspicion of PE.[164,165]	I	A
If CUS shows only a distal DVT, further testing should be considered to confirm PE.[177]	IIa	B
If a positive proximal CUS is used to confirm PE, assessment of PE severity should be considered to permit risk-adjusted management.[178,179]	IIa	C

nostic values are summarized in Supplementary Data *Table 3*. Of these, an RV/LV diameter ratio ≥1.0 and a TAPSE <16 mm are the findings for which an association with unfavourable prognosis has most frequently been reported.[148]

Table 8 **Classification of pulmonary embolism severity and the risk of early (in-hospital or 30 day) death**

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

Table 9 Treatment of right ventricular failure in acute high-risk pulmonary embolism

Strategy	Properties and use	Caveats
Volume optimization		
Cautious volume loading, saline, or Ringer's lactate, ≤500 mL over 15–30 min	Consider in patients with normal–low central venous pressure (due, for example, to concomitant hypovolaemia)	Volume loading can over-distend the RV, worsen ventricular interdependence, and reduce CO[239]
Vasopressors and inotropes		
Norepinephrine, 0.2–1.0 μg/kg/min[a 240]	Increases RV inotropy and systemic BP, promotes positive ventricular interactions, and restores coronary perfusion gradient	Excessive vasoconstriction may worsen tissue perfusion
Dobutamine, 2–20 μg/kg/min[241]	Increases RV inotropy, lowers filling pressures	May aggravate arterial hypotension if used alone, without a vasopressor; may trigger or aggravate arrhythmias
Mechanical circulatory support		
Veno–arterial ECMO/extracorporeal life support[251,252,258]	Rapid short-term support combined with oxygenator	Complications with use over longer periods (>5–10 days), including bleeding and infections; no clinical benefit unless combined with surgical embolectomy; requires an experienced team

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

N ENGL J MED 370;15 NEJM.ORG APRIL 10, 2014

Fibrinolysis for Patients with Intermediate-Risk Pulmonary Embolism

Guy Meyer, M.D., Eric Vicaut, M.D., Thierry Danays, M.D., Giancarlo Agnelli, M.D.,

CONCLUSIONS

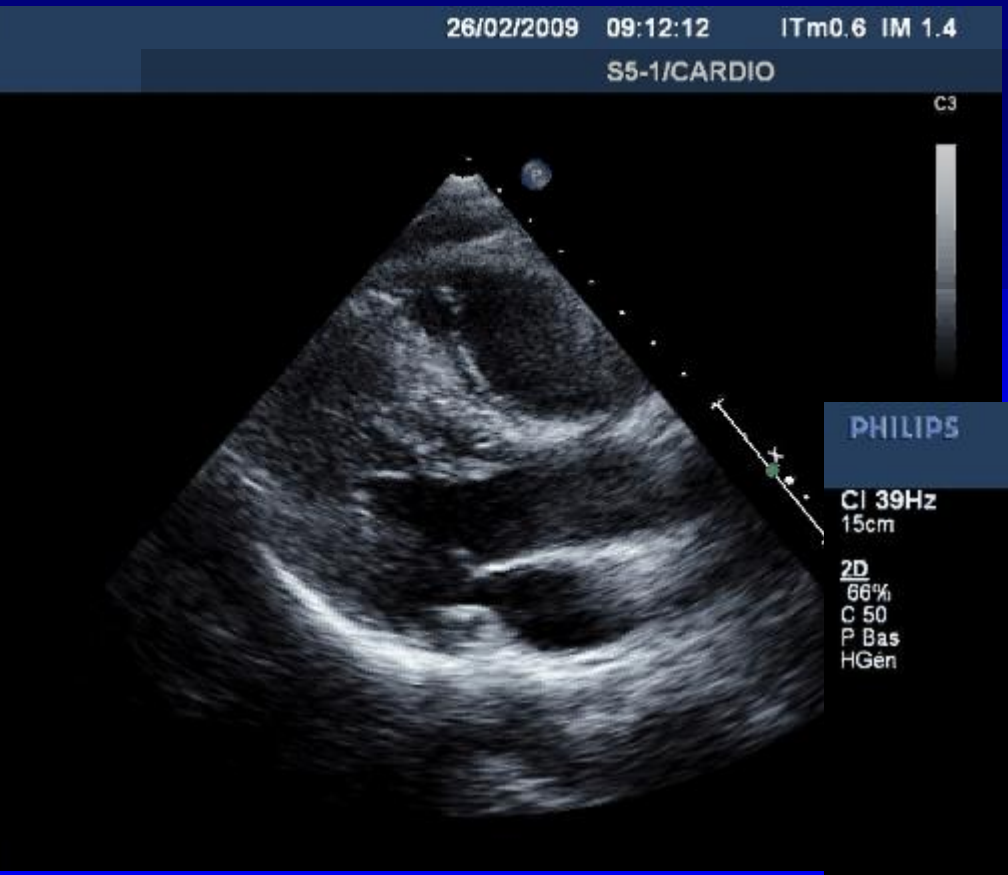
In patients with intermediate-risk pulmonary embolism, fibrinolytic therapy prevented hemodynamic decompensation but increased the risk of major hemorrhage and stroke.

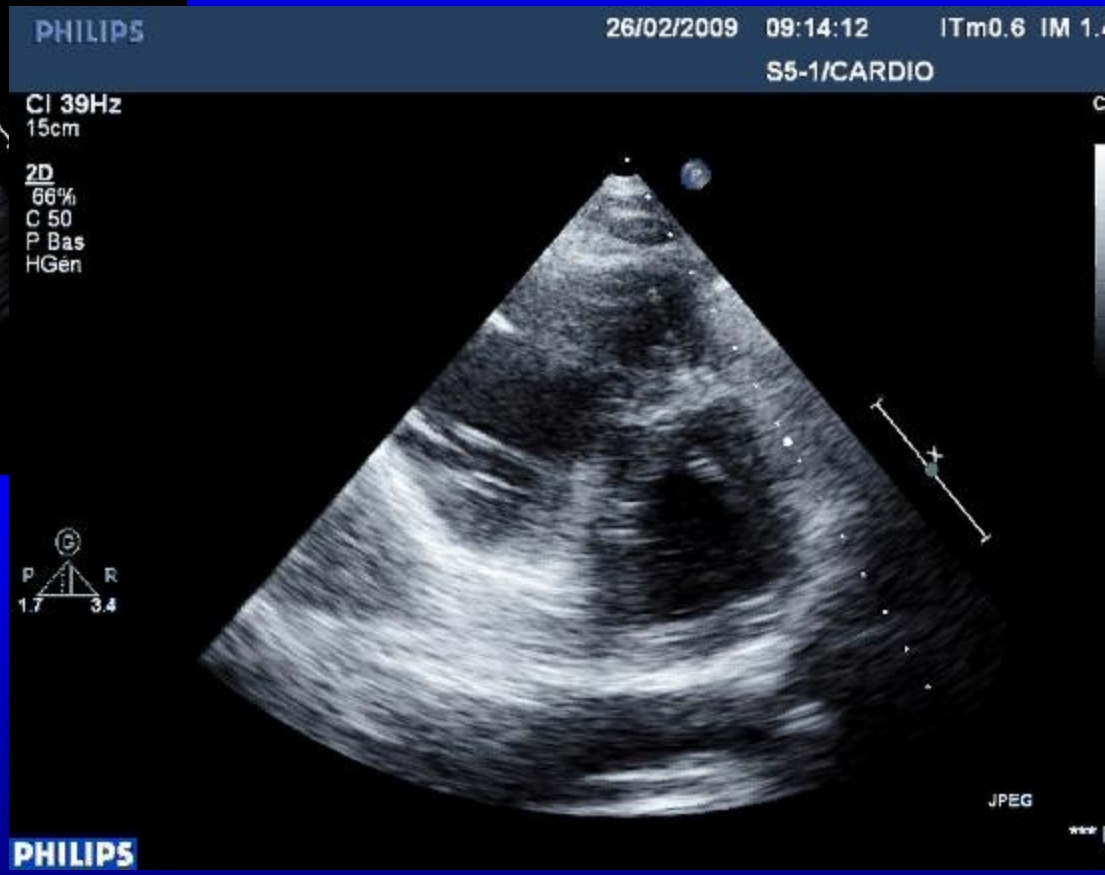
Cinéma

Cas clinique 1

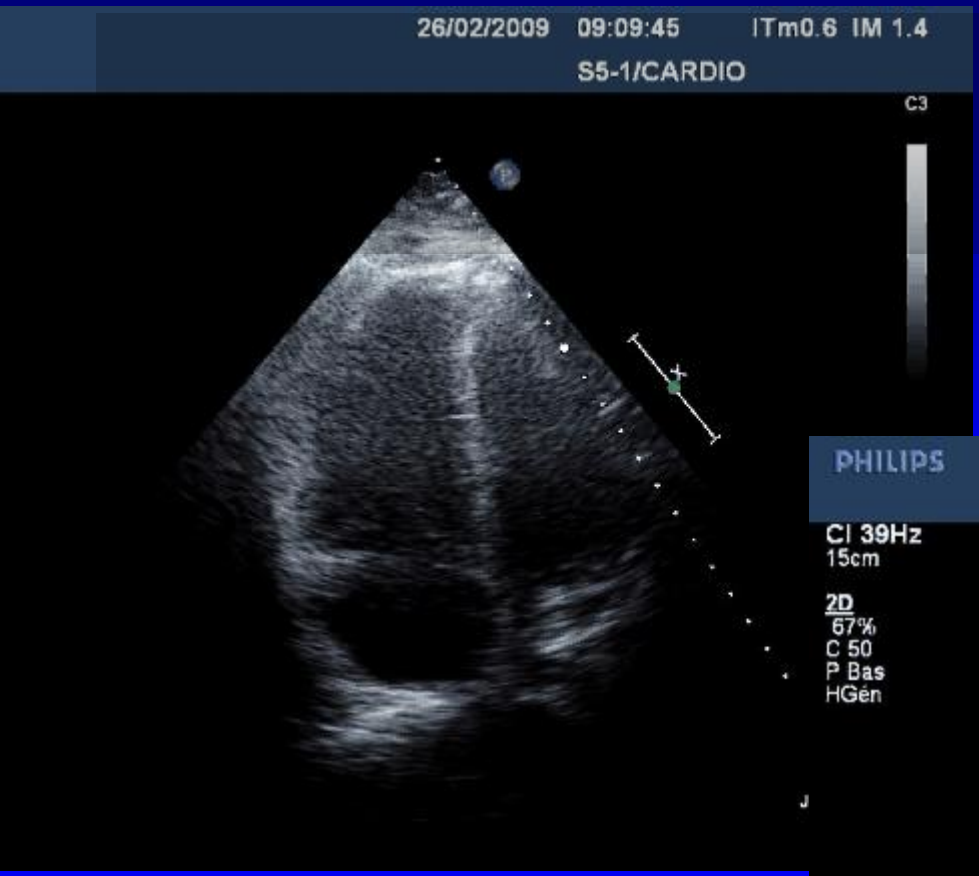
- Patient de 23 ans adressé aux urgences médicales par ambulance après appel au SAMU pour "anxiété".
- A l'accueil des urgences, cyanosé SpO2 60%, agité. ACR à l'arrivée au déchocage.
- MCE 30 min, 29mg adrénaline puis relai IVSE, thrombolyse puis transfert au bloc pour thrombectomie chirurgicale

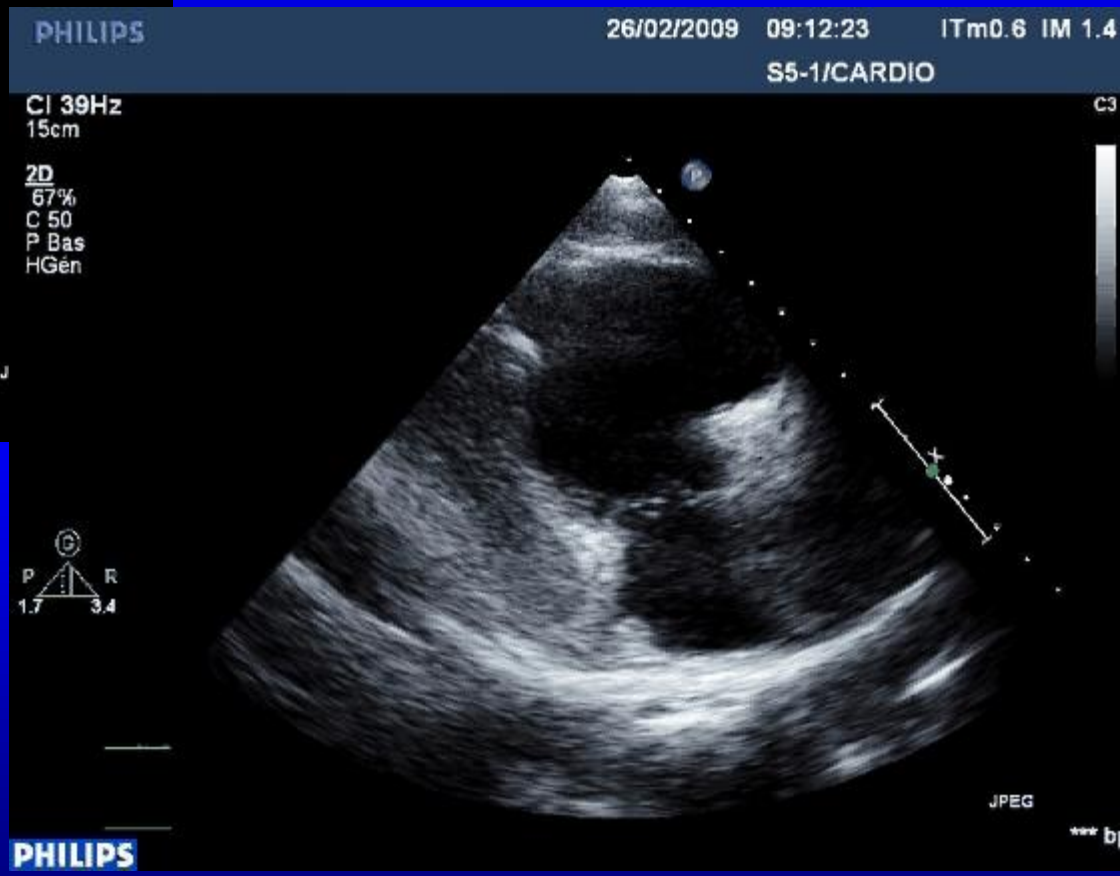
Cas clinique 1



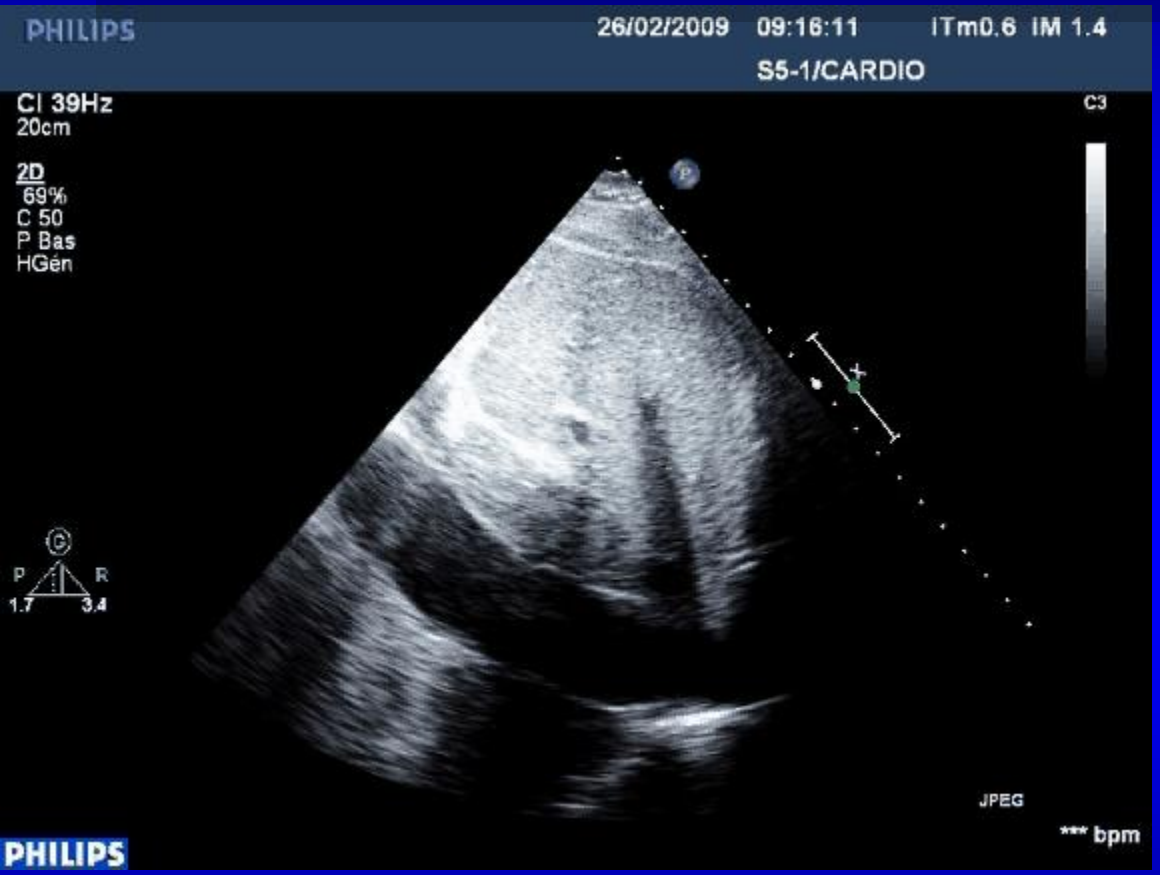


Cas clinique 1

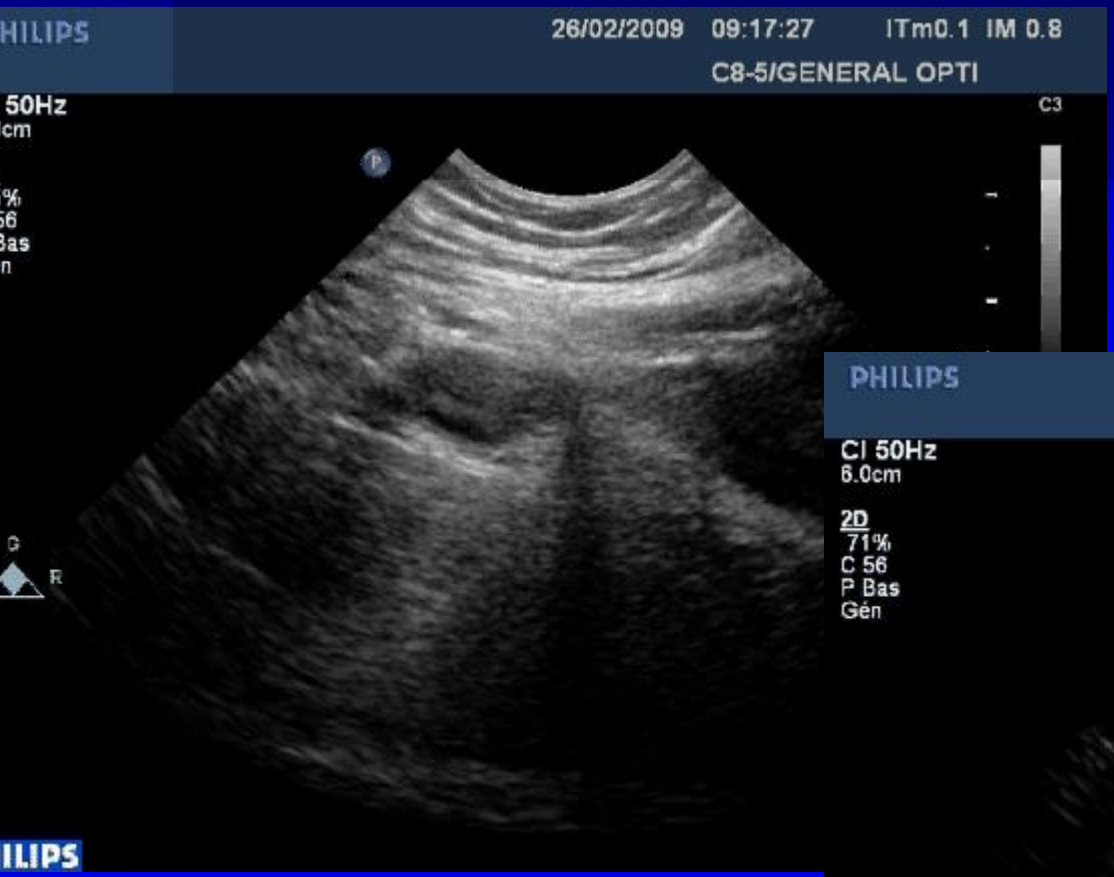


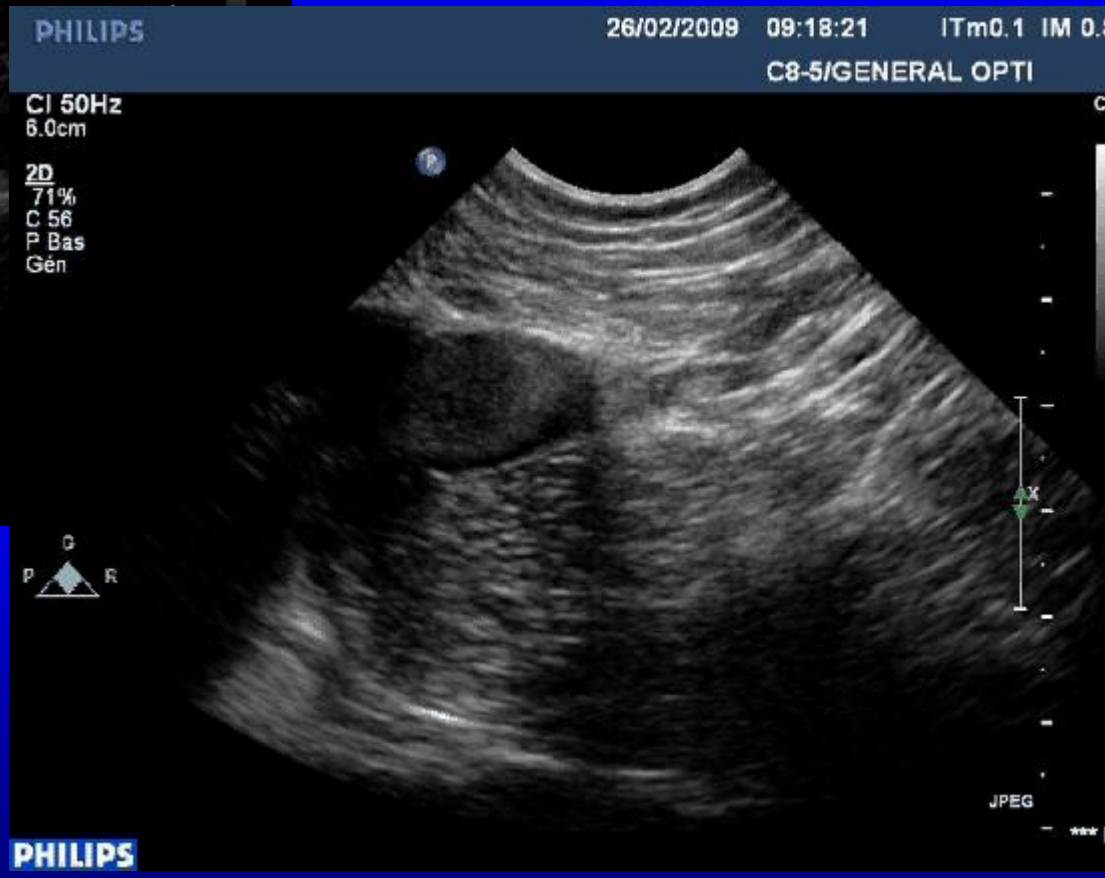


Cas clinique 1



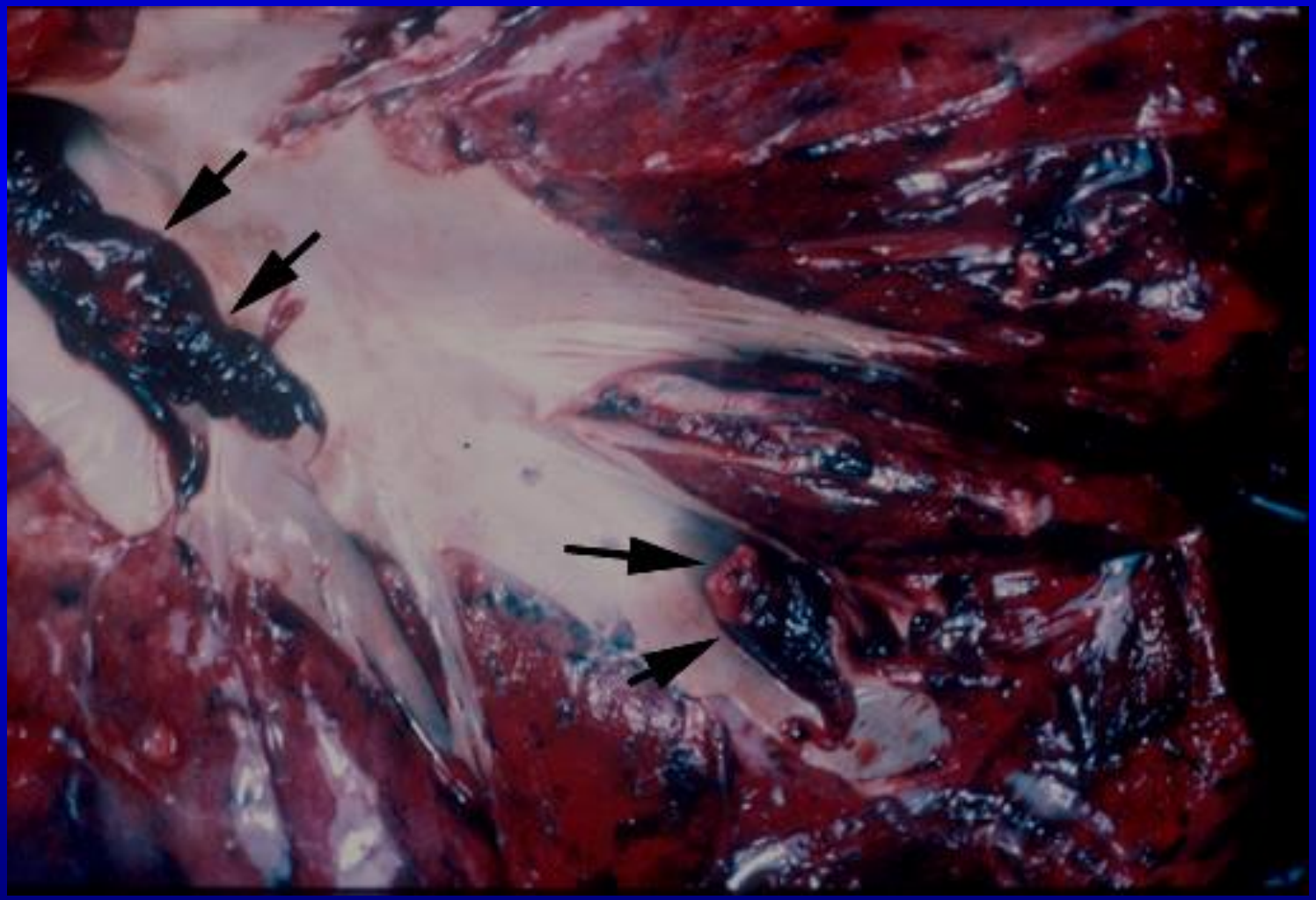
Cas clinique 1





Cas clinique 1

Décès au bloc: caillot envahissant toute la circulation pulmonaire



Cas clinique 2

- Malaise sans Perte de Connaissance Initiale en se rendant chez le kiné.
- Cyanose péribuccale++ et révulsion oculaire transitoire selon les témoins.

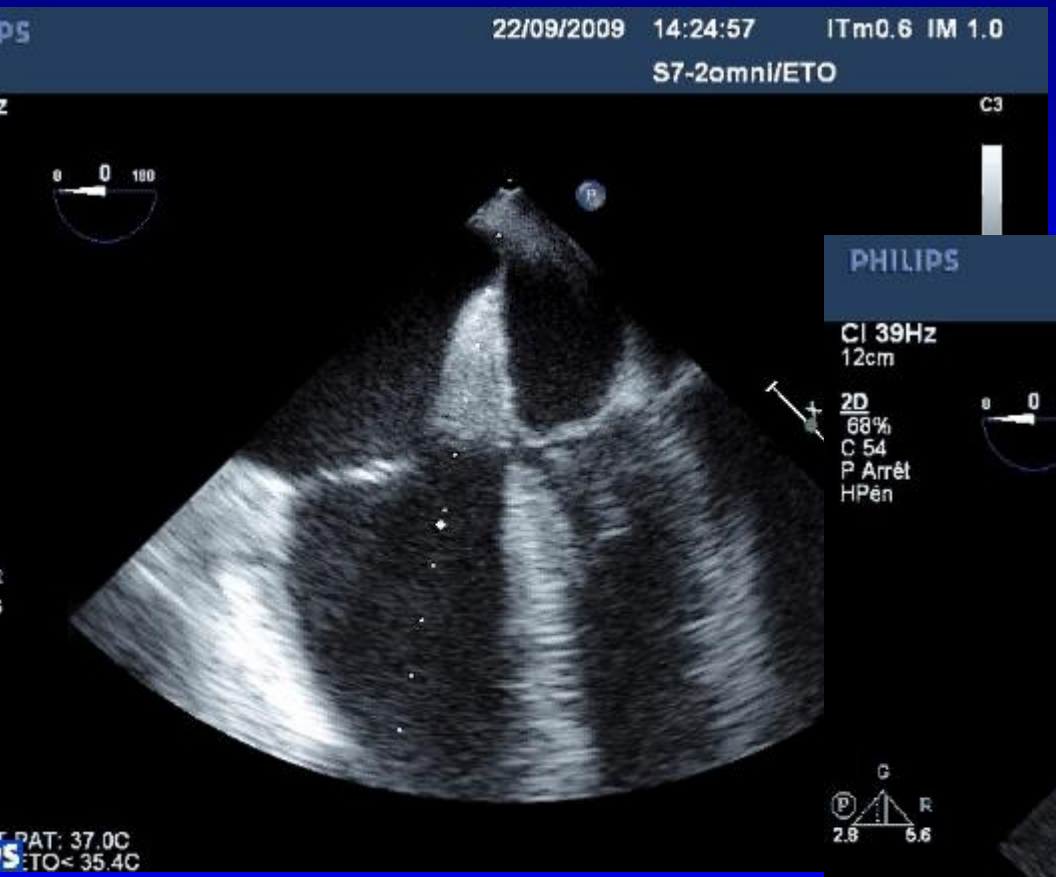
- A l'arrivée du SMUR, patiente très cyanosée, marbrures généralisées. Dyspnée, ne supporte pas le MHC.
- Pas de notion de Sd fébrile récent. Pas de DT.
- Par la suite bradycardie puis asystolie=> MCE+Adré+IOT.

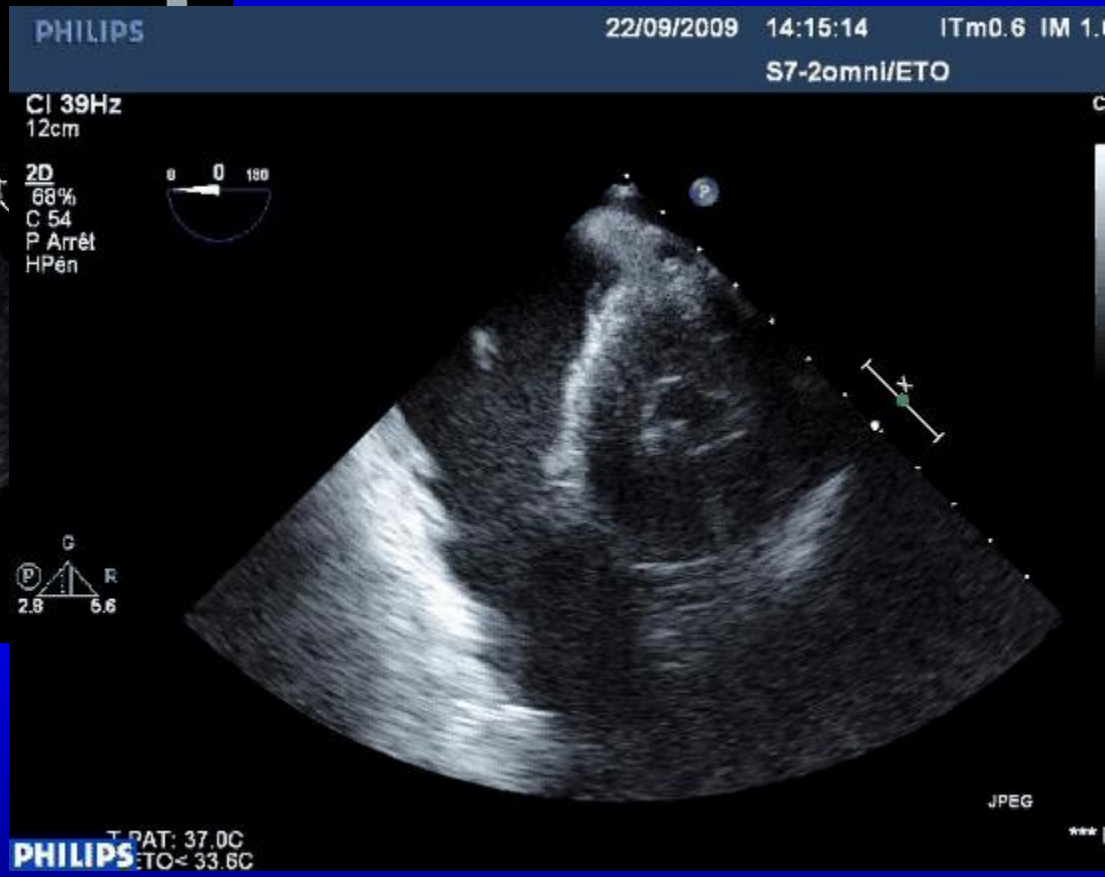
12h45 déchocage : signes de réveil lutte respi
=> Hypno+Suf
A l'ETT: dilatation VD++, embol 0
(conditions d'examen difficile)

13h10 Dégradation hémodynamique PA 70/40,
FC 90 Dobu 5 gamma/kg/mn
13h15 PA 80/30, bradycardie 50
=> Dobu 15 gamma/kg/mn
Protocole thrombolyse EP
Puis asystolie -> MCE+ Adré 1mg X 2
-> Rythme sinusal

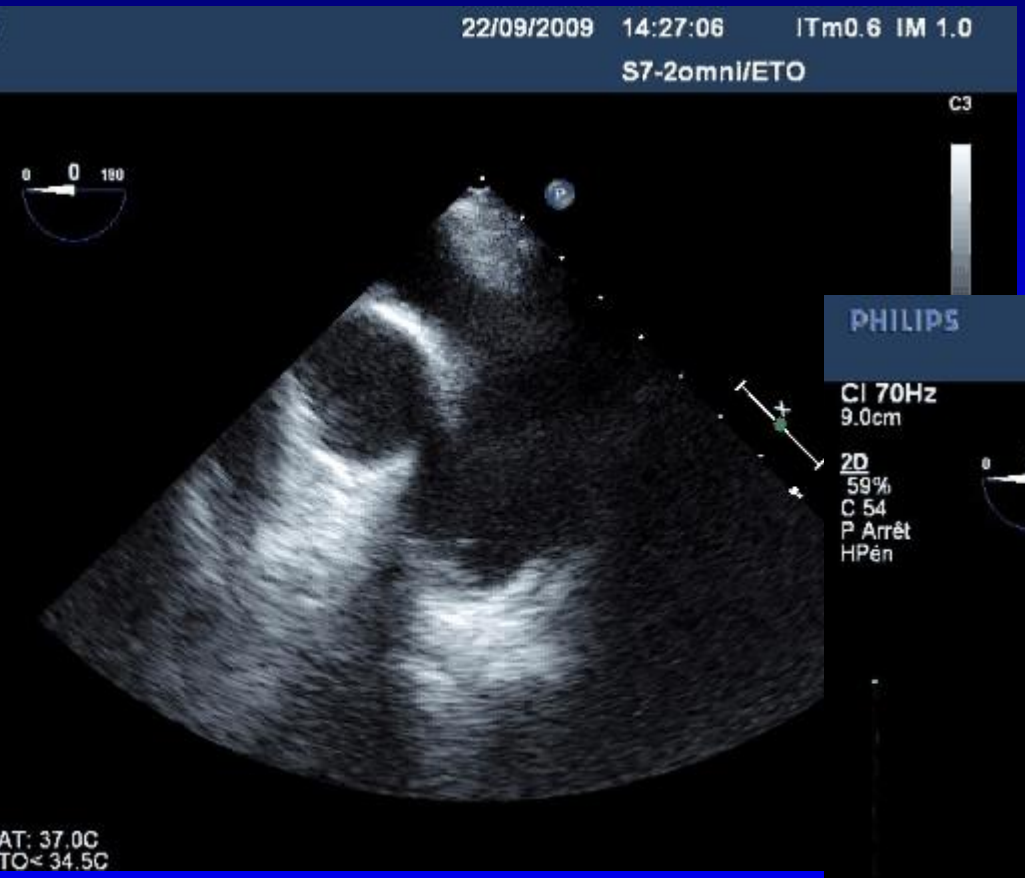
13h40 transfert en réa

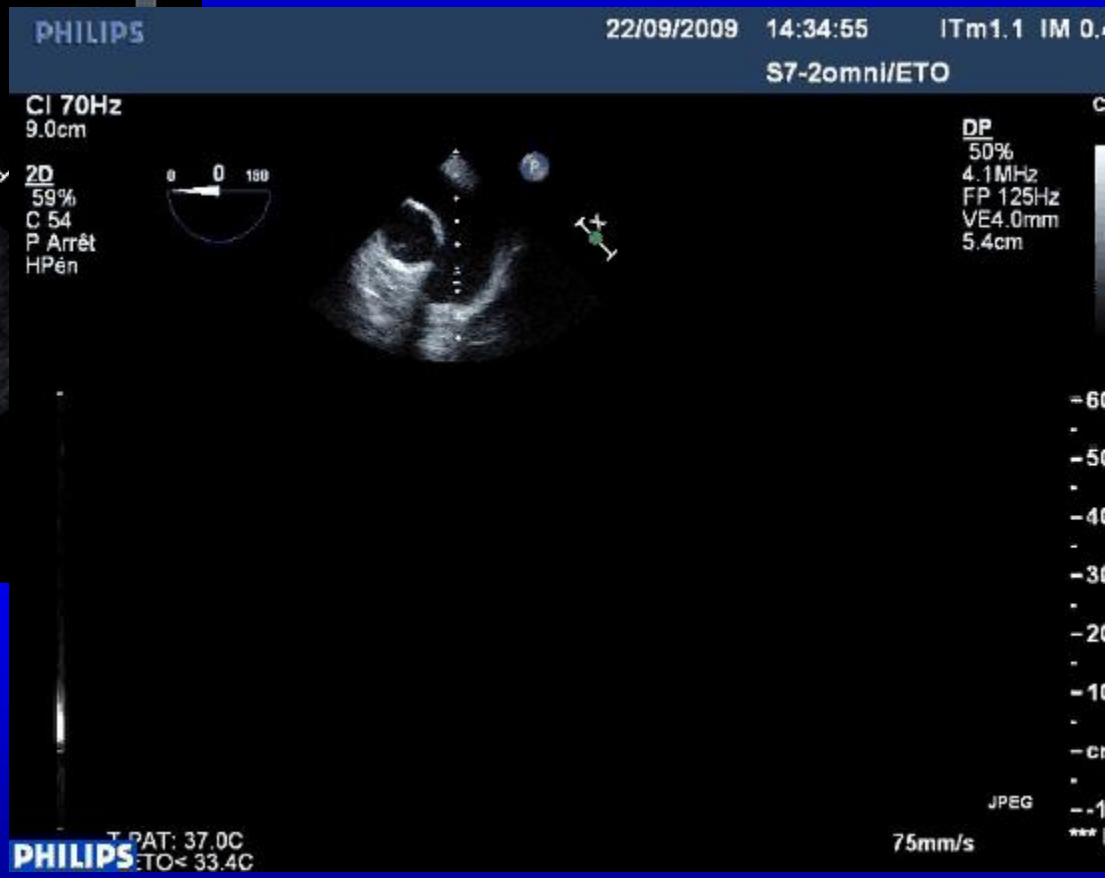
Cas clinique 2



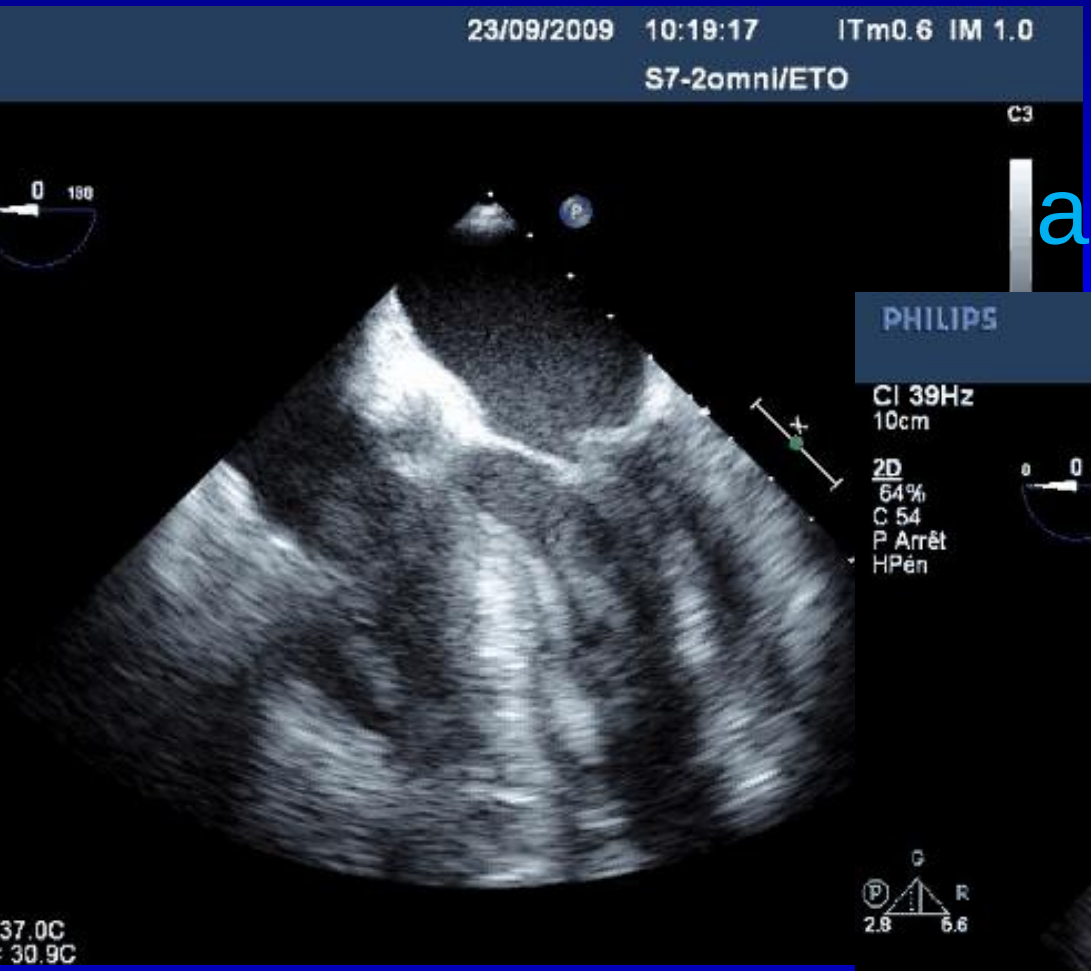


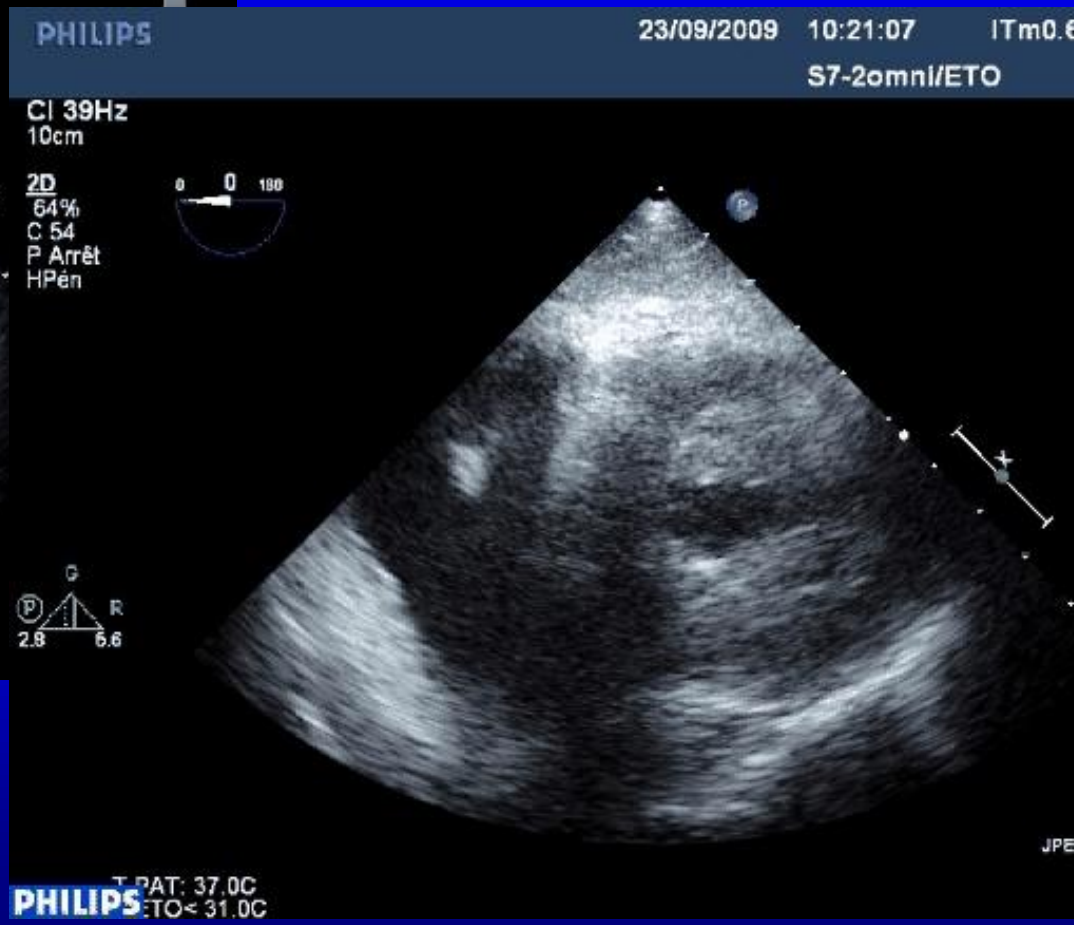
Cas clinique 2





Cas clinique 2
après thrombolyse H6





Cas clinique 3

- Motif d'H: détresse respiratoire aigue suite à une embolisation splénique pour hémoperitoine sur fracture splénique traumatique

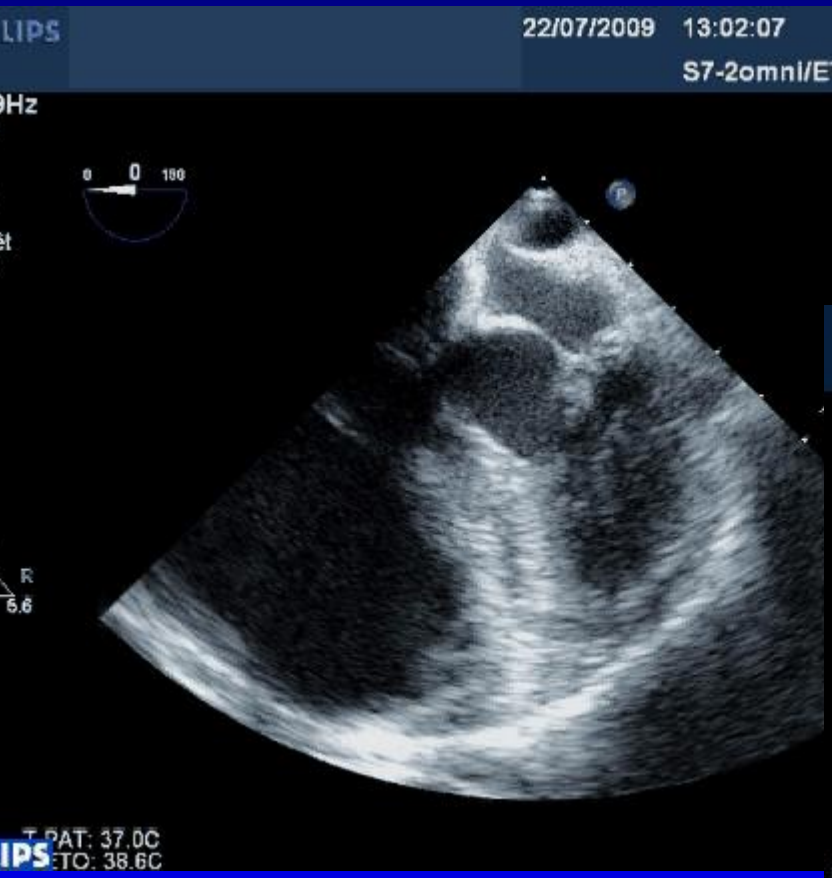
- HDM: *09/07*: chute du toit d'un camion.
- *16/07*: aux urgences pour douleur abdominale: Hb 7,7 g/dl , hémodynamique stable => TDM abdo : épanchement intra-peritonéal dans douglas + image pouvant correspondre à un hématome sous-capsulaire splénique.
- transfusion 3 CG puis embolisation tronc A.splénique au bloc puis transfert au SI de chirurgie viscérale pour surveillance.

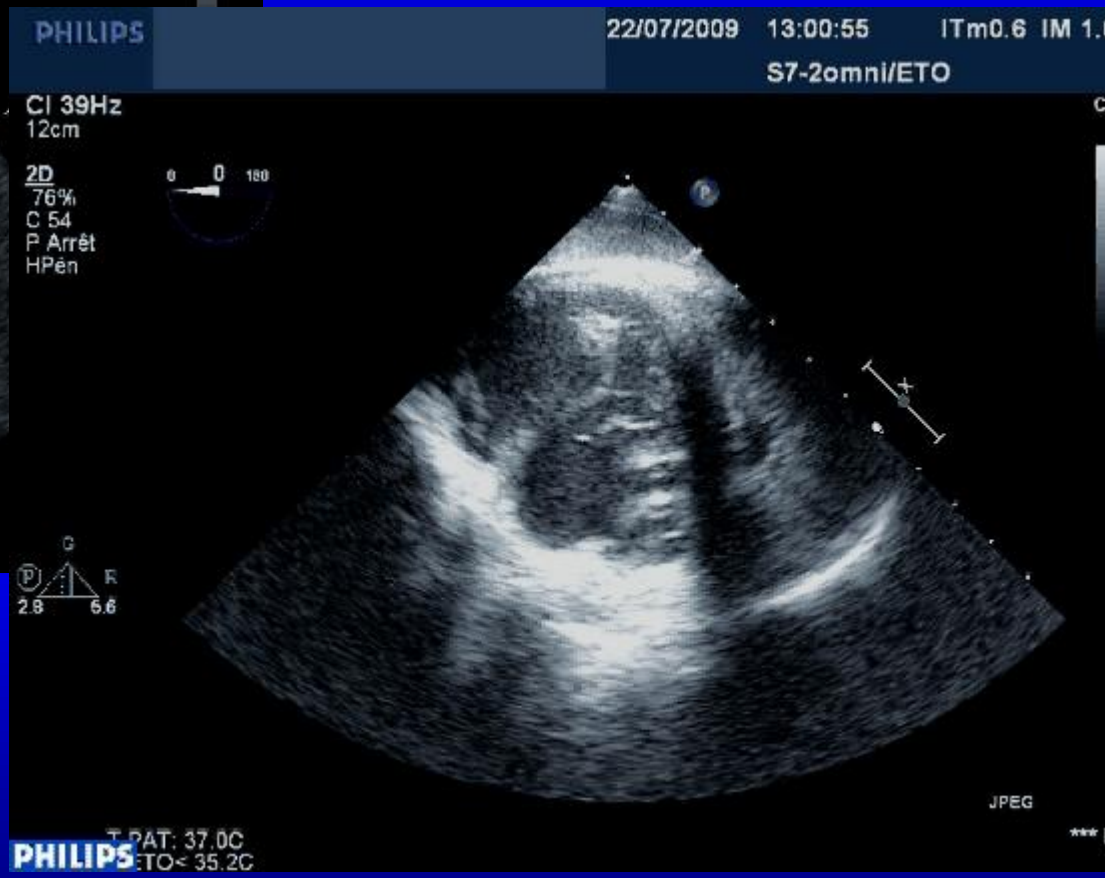
Cas clinique 3

- aux SI de chirurgie viscérale :
- 16 au 21/07 : agitation, trémulation, mis sous loxapac.
-
- le 22/07 : polypnée à 40-45/min désaturation à 60%, tachycardie à 140-145/min, TA 130/80
- => TDM abdo prescrit par interne : pas de saignement splénique.
- => appel réa med pour prise en charge de la détresse respiratoire aigue.

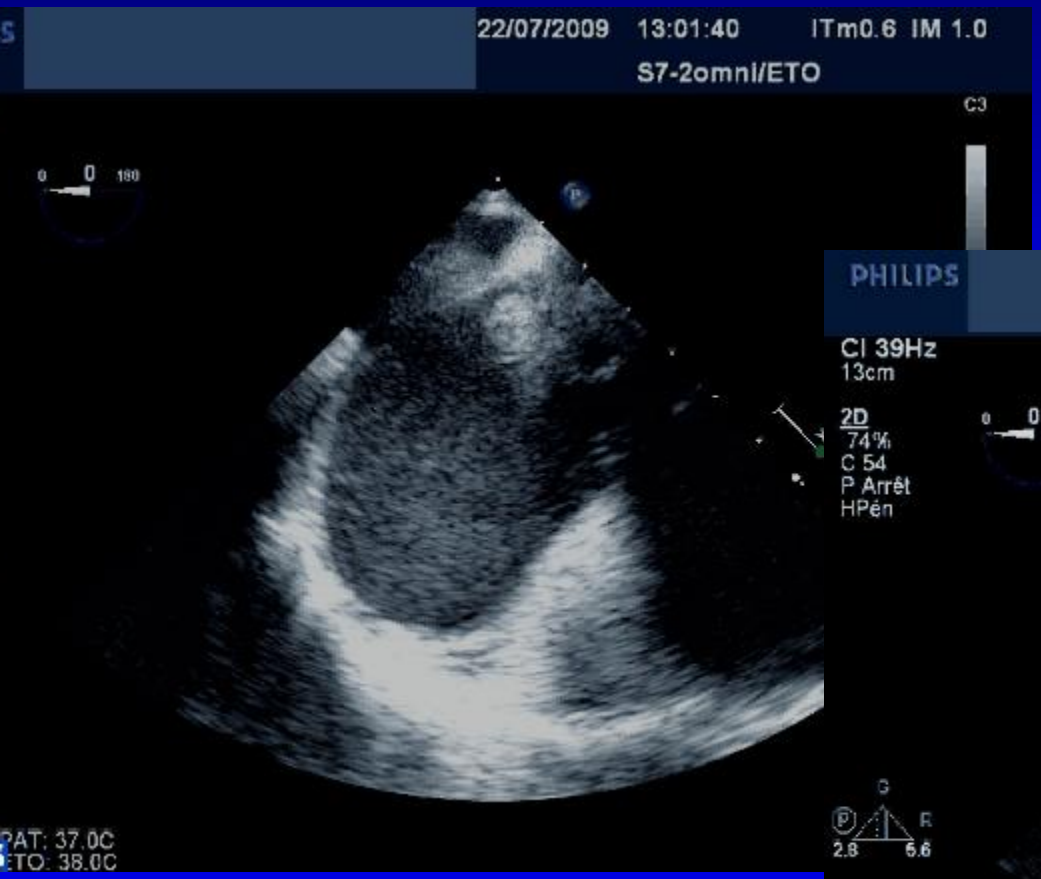
- IOT VM puis échographie

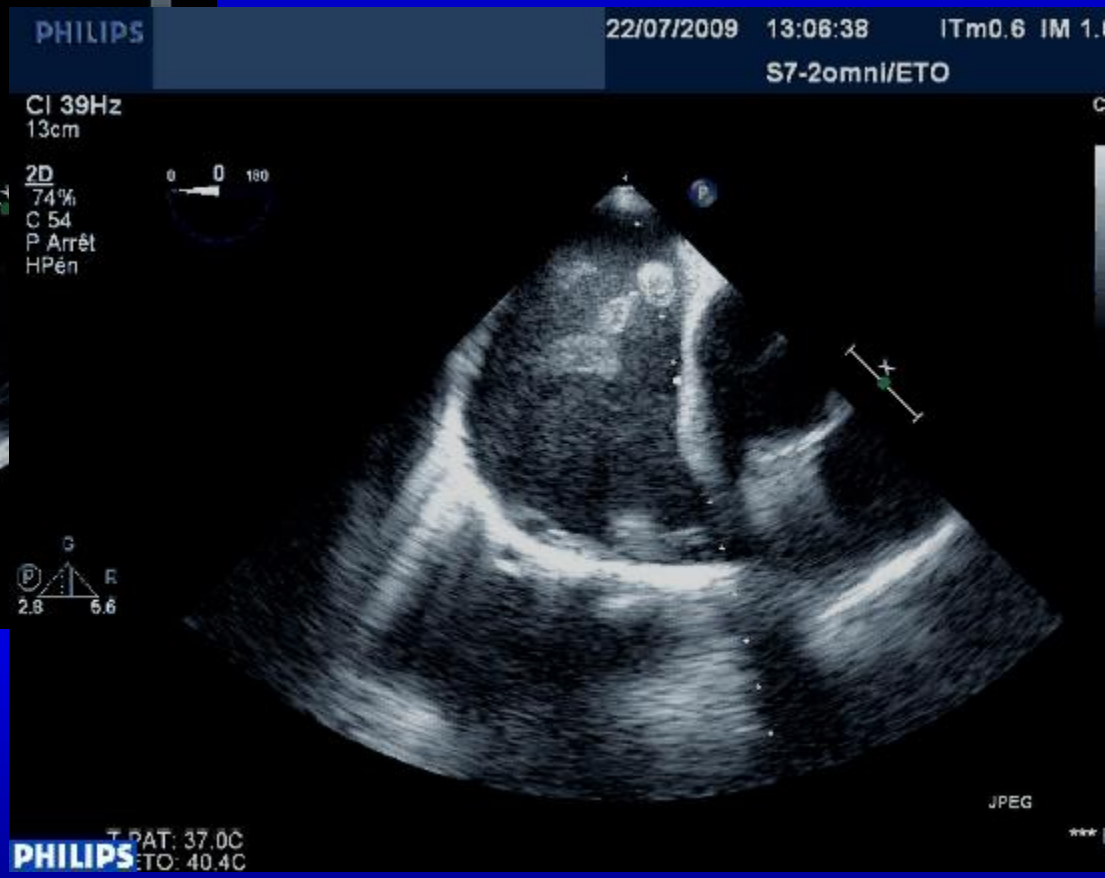
Cas clinique 3

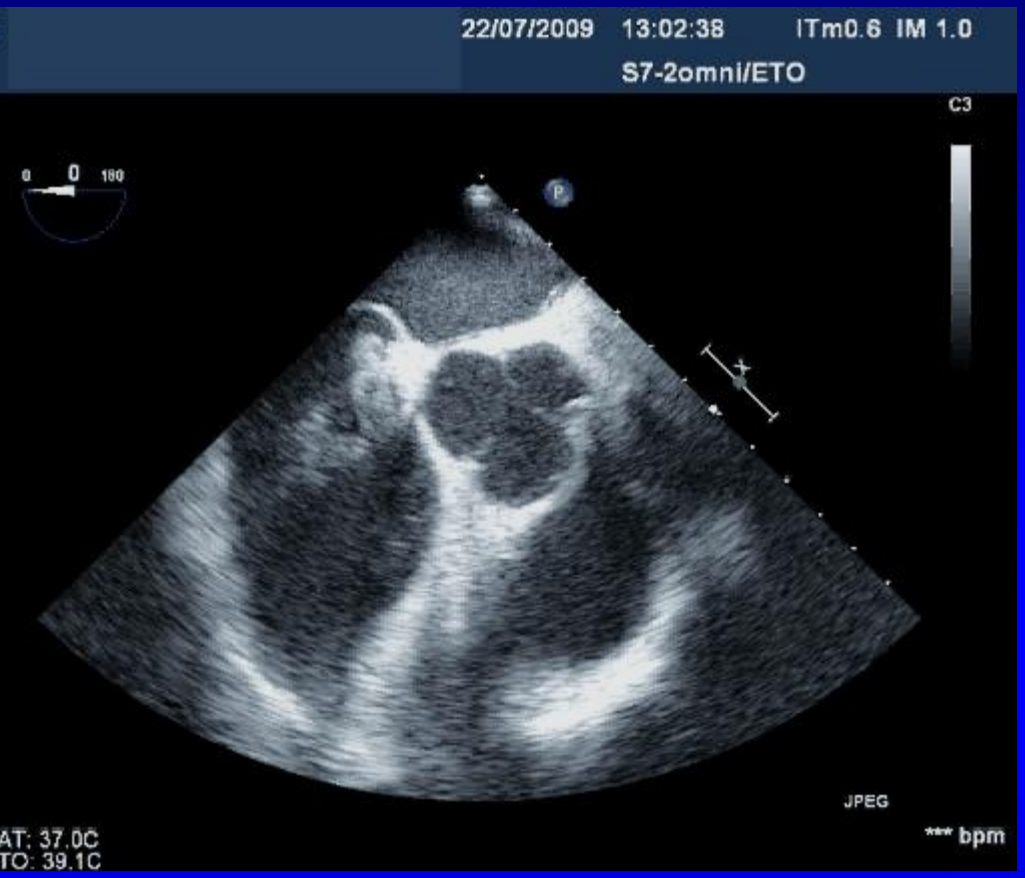




Cas clinique 3



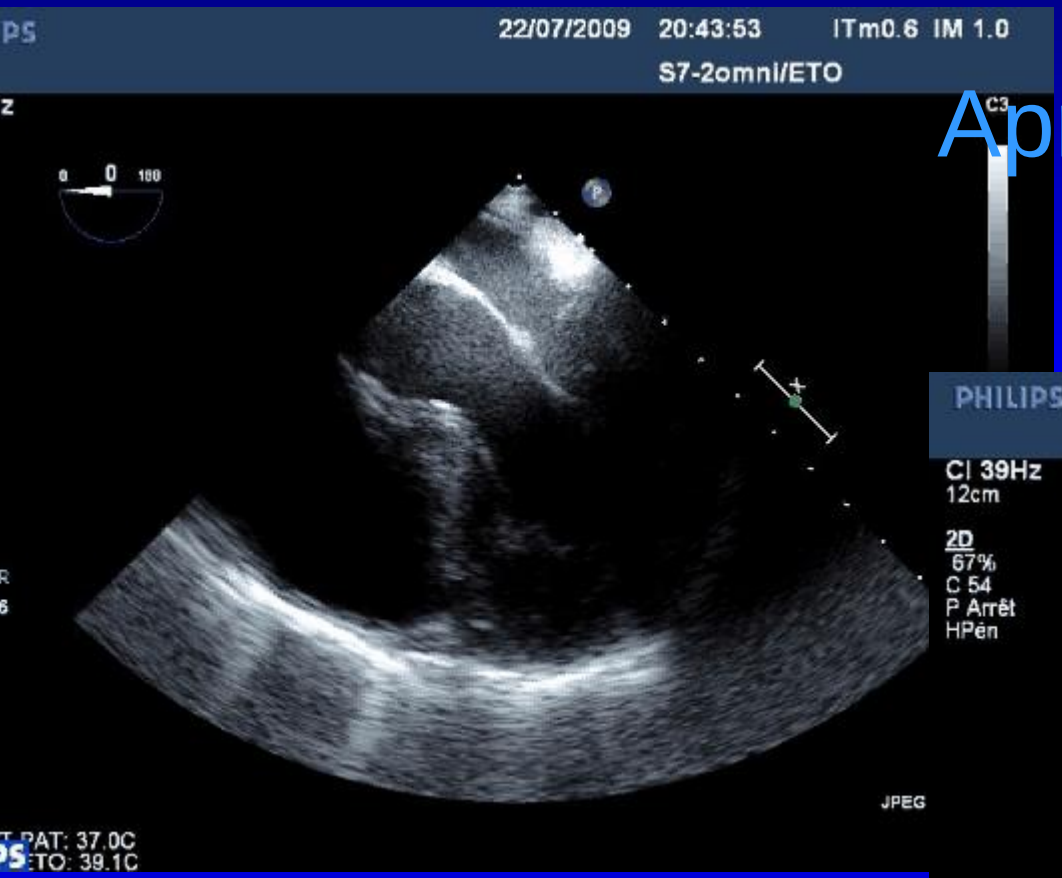


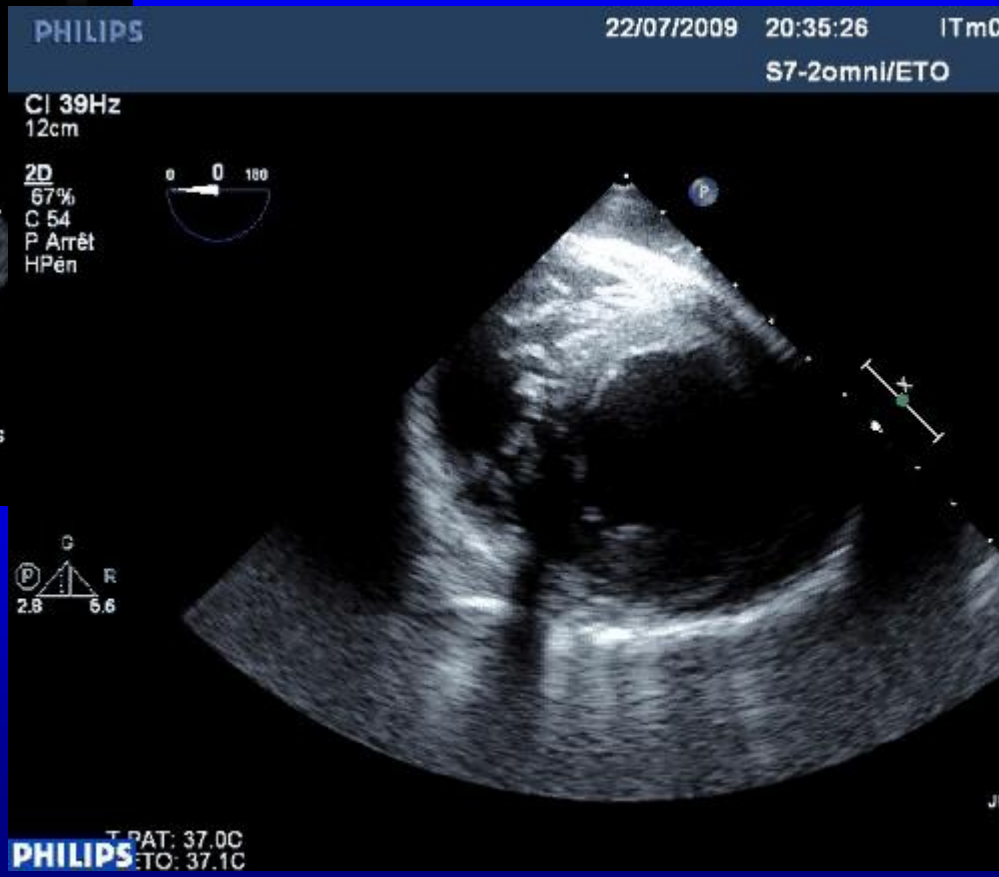


Cas clinique 3

Cas clinique 3

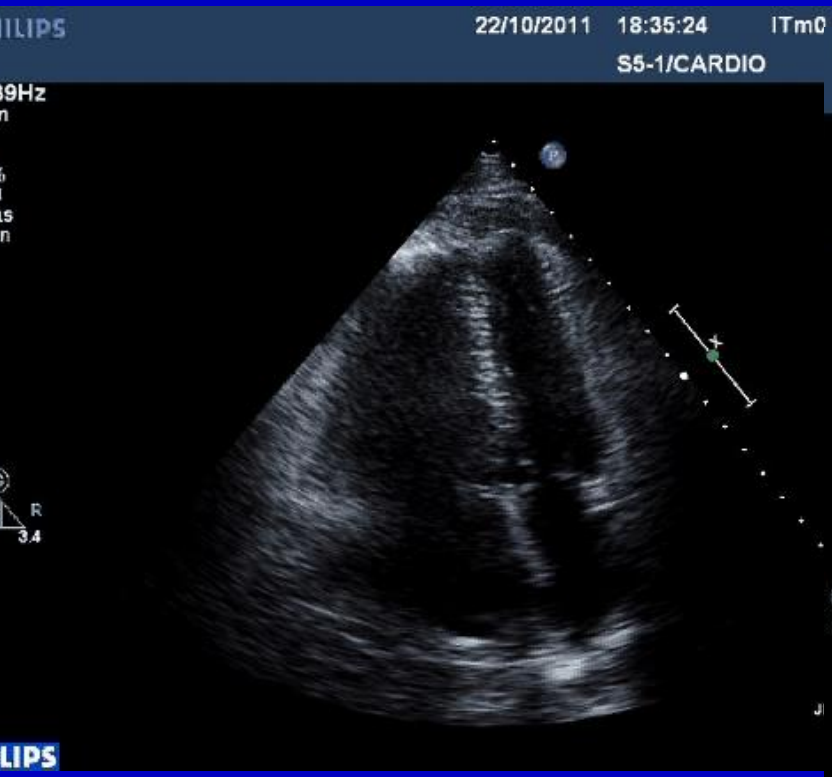
Après thrombectomie





Cas clinique 4

- Homme de 64 ans
- État de mal épileptique
- Hématome sous dural: pas de chir
- J3: état de choc
- ETT+ ETO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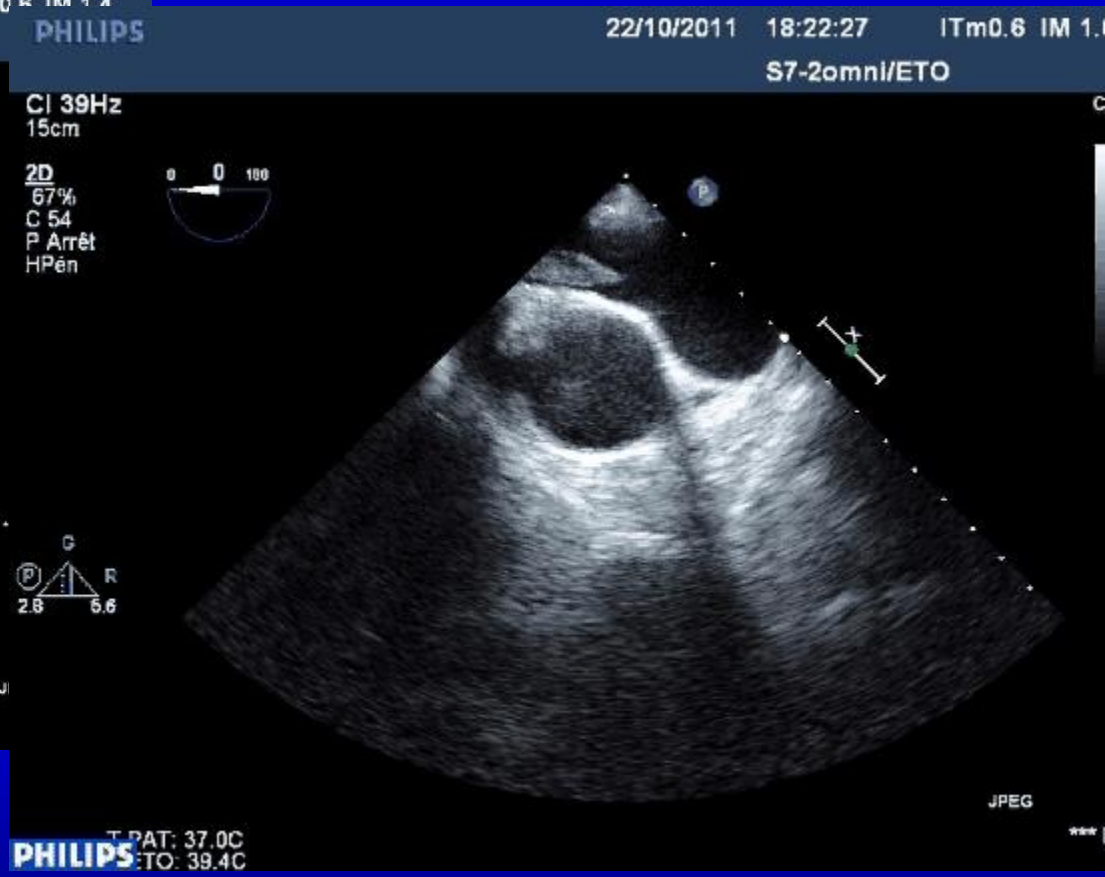
S5-1/CARDIO
39Hz
R
3.4
LIPS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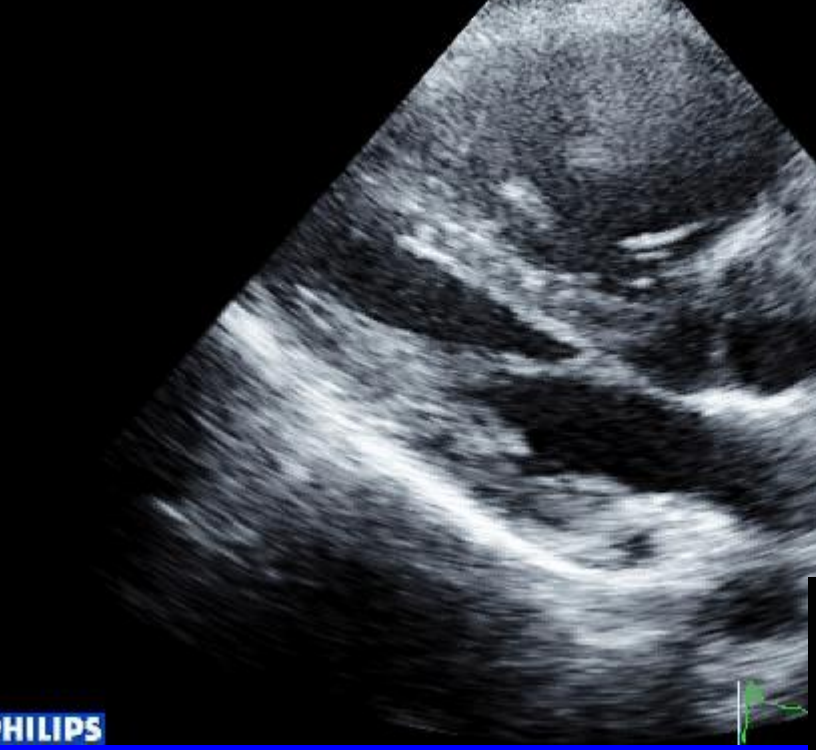
22/10/2011 18:22:27
ITm0.6 IM 1.
S7-2omni/ETO
CI 39Hz
15cm
2D
67%
C 54
P Arrêt
HPén
0 0 100
C
P
R
2.8
5.6
JPEG
PHILIPS
T PAT: 37.0C
ETO: 39.4C

Cas clinique 5

- Femme de 23 ans, 7 SA, terrain de thrombophilie
- 1er séjour en réa: pour EP 06/01/2014 → TAC

- 2ème séjour: tachychardie → suspicion de récidive embolique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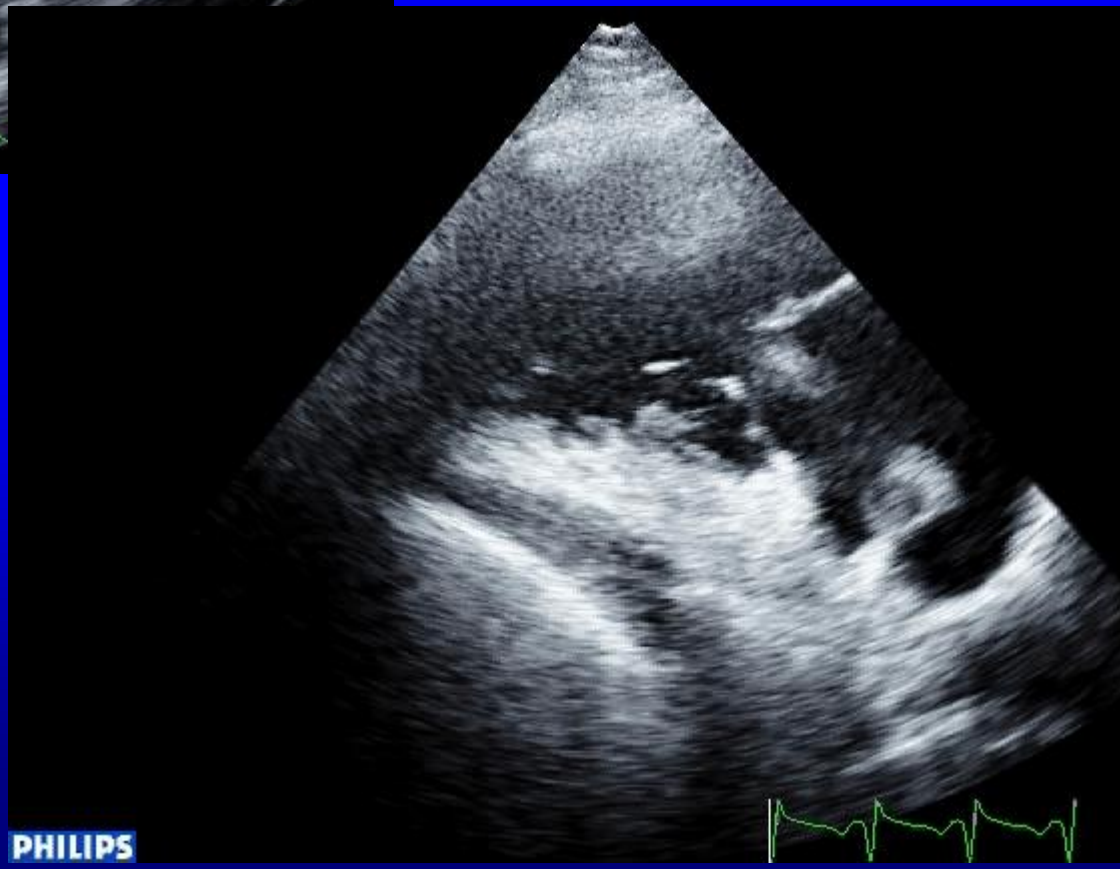
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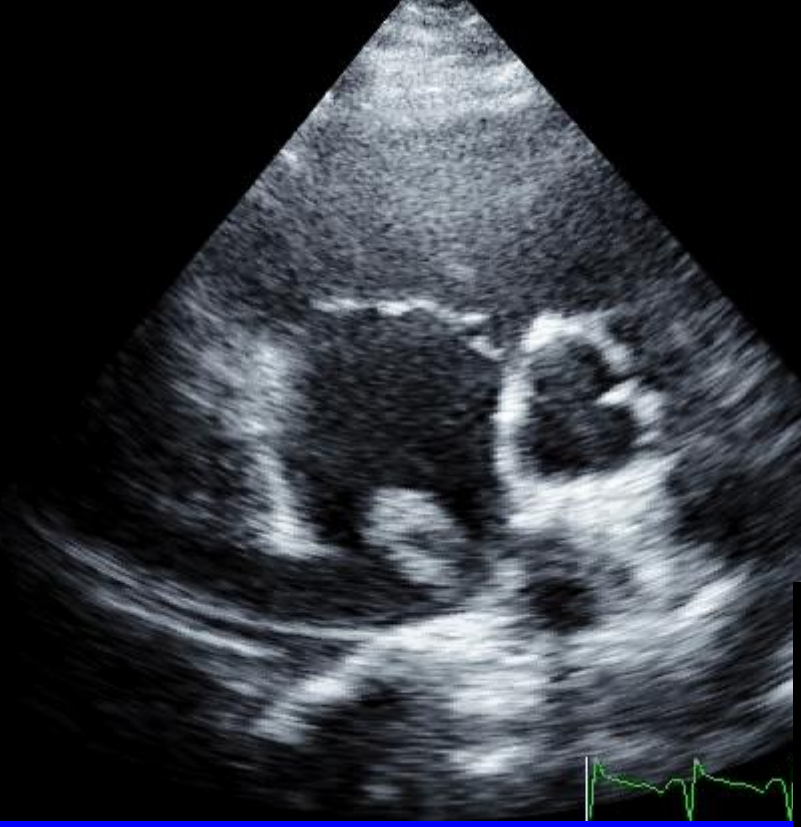

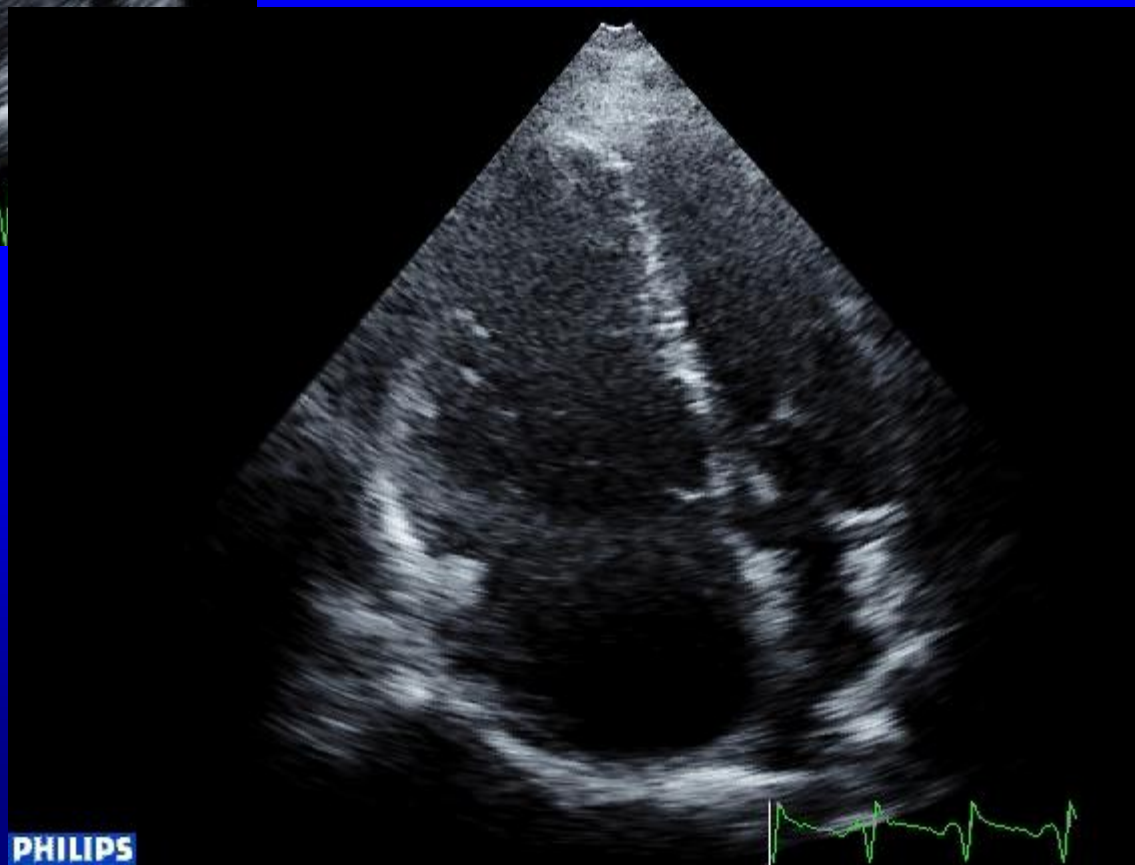
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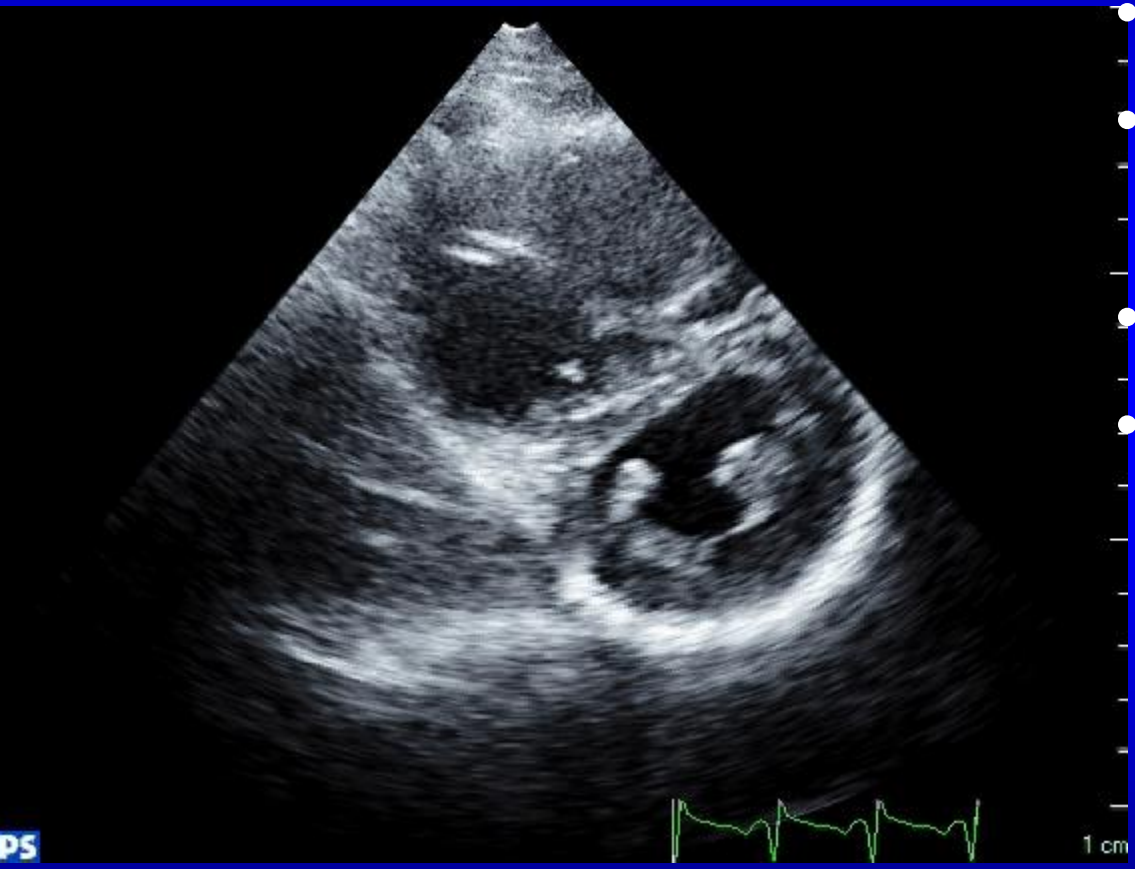

- Thrombolyse
- Disparition du caillot à H12
- Filtre cave à J3
- ITG à J4 avec fenêtre d’arrêt du TAC