



FORUM EUROPÉEN, CŒUR, EXERCICE & PRÉVENTION



Intérêts de l'épreuve d'effort cardio-respiratoire dans l'HTAP

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Conflits d'intérêts

- Liens d'intérêt :

Ferrer, Galenica, MSD, Johnson and Johnson

- Liens d'intérêt en relation avec la présentation :

Aucuns

Déterminants de la dyspnée - HTAP

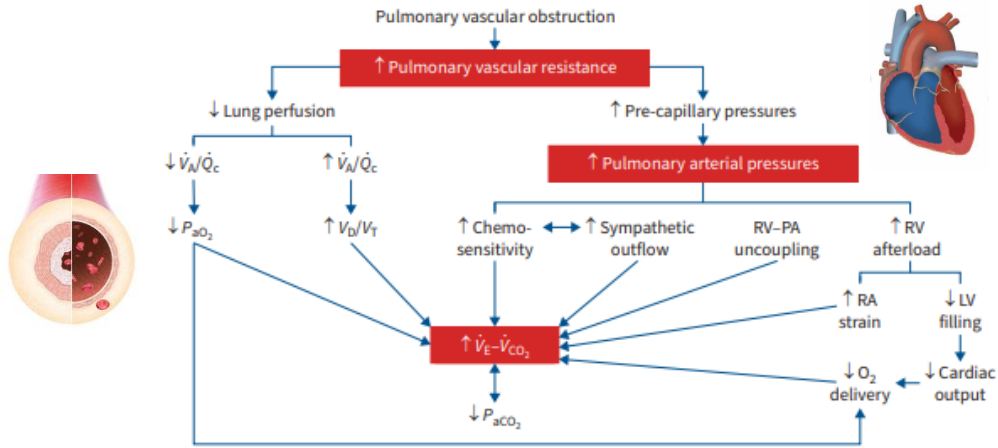


FIGURE 4 The main mechanisms linking the fundamental pathophysiological features of pulmonary hypertension (increased pulmonary vascular resistance and pulmonary arterial pressures) with excess exertional ventilation, i.e. high ventilation (\dot{V}_E)-pulmonary CO₂ output (\dot{V}_{CO_2}) relationship. The relative importance of individual mechanisms varies in pulmonary arterial hypertension and chronic thromboembolic pulmonary hypertension. See text for elaboration. †: increased; ‡: decreased; \dot{V}_A : alveolar ventilation; \dot{Q}_c : capillary perfusion; P_{aCO_2} : arterial CO₂ partial pressure; V_D : dead space volume; V_T : tidal volume; RV: right ventricle; PA: pulmonary artery; LV: left ventricle; RA: right atrium.

Neder J.A, et al. ERJ. 2022

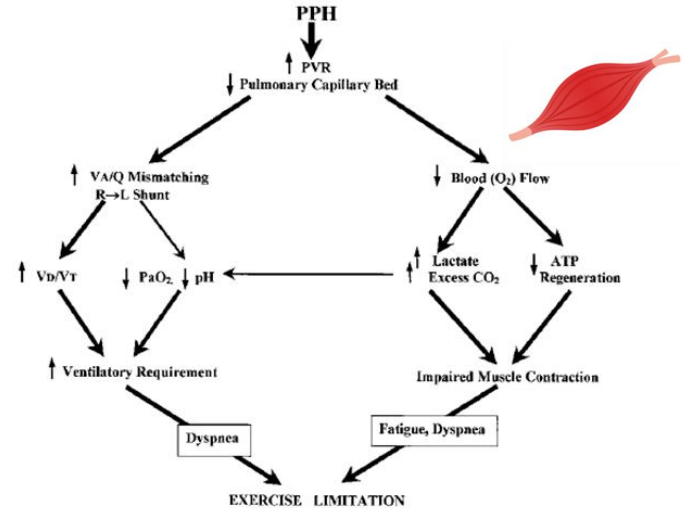


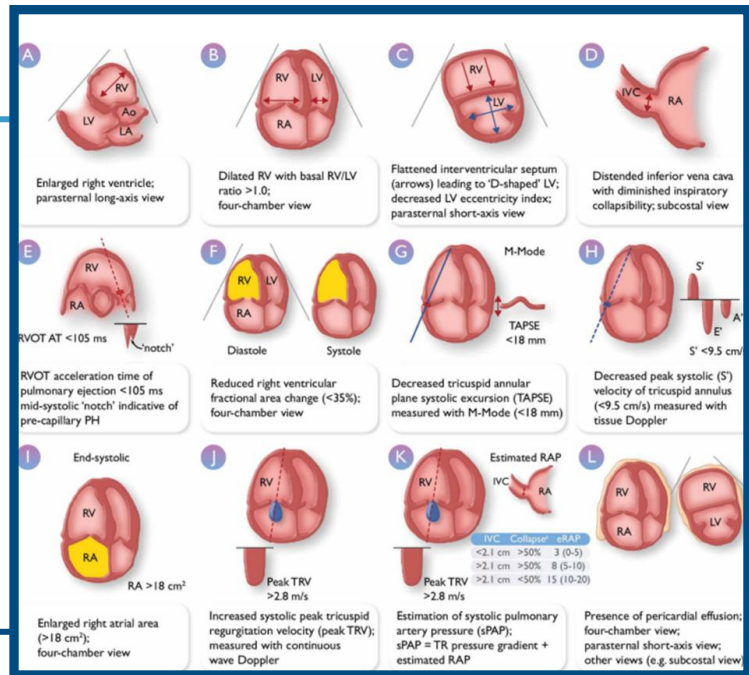
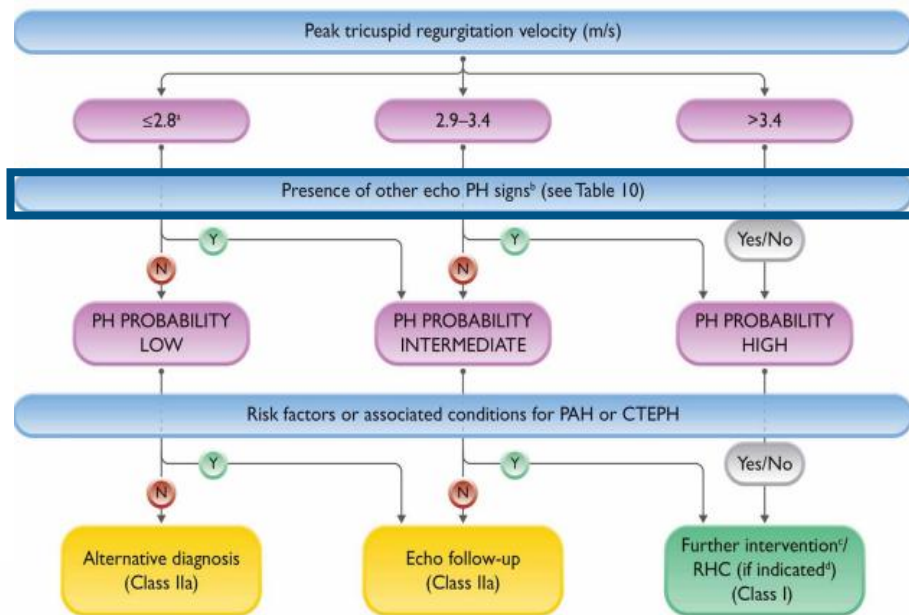
Figure 4. Pathophysiology of exercise limitation of PPH patients. Longer arrows show pathways leading to dyspnea and fatigue with exercise. Shorter arrows indicate how each response differs from normal. PVR indicates pulmonary vascular resistance; V_A/Q , alveolar ventilation/perfusion ratio; R, right; L, left; V_D/V_T , dead space volume/tidal volume ratio; and P_{aO_2} , arterial O₂ pressure.

Sun XG, et al. Circulation 2001

Si IC > 2l/min/m²
GDS

Conséquences physiopathologiques à l'exercice	Epreuve cardio respiratoire d'exercice
Diminution de l'aptitude aérobie	Diminution VO ₂ pic
Espace mort augmenté	Diminution insuffisante voir augmentation V _d /V _t
Hyperventilation	Augmentation VE/VO ₂ , VE/VCO ₂ Augmentation pente VE/VCO ₂
Hypoxémie	Augmentation du gradient alveolo-artériel en O ₂
Augmentation insuffisante du DC	Evolution anormale VO ₂ /FC <i>Puissance circulatoire</i> <i>Temps de ½ récupération VO₂</i> <i>Oscillations ventilatoires</i>
Hyperinflation dynamique (60%des patients)	Diminution de la capacité inspiratoire
Limitation musculaire périphérique	SV1 précoce

Dépistage échographique



Humbert M. ESC/ERS Guidelines. ERJ. 2022

Cathétérisme cardiaque droit

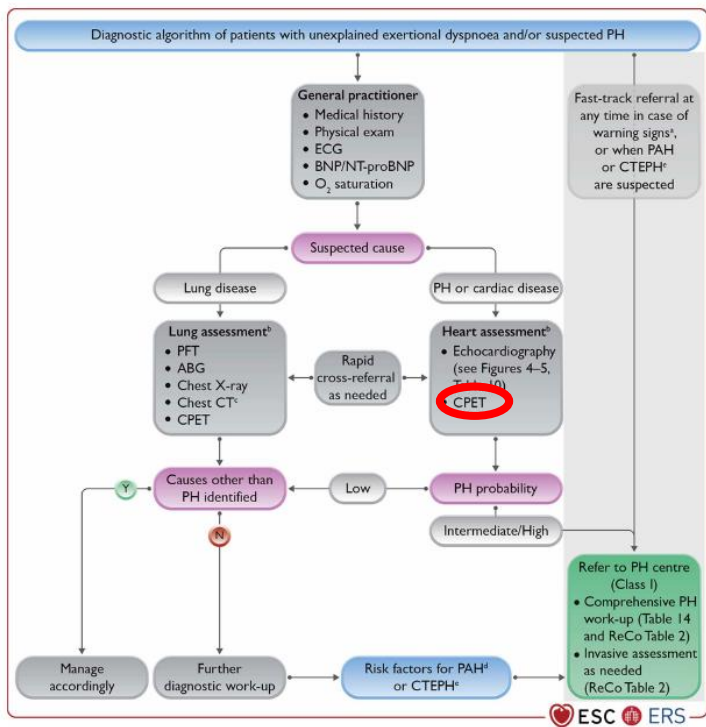


FIGURE 6 Diagnostic algorithm of patients with unexplained dyspnoea and/or suspected pulmonary hypertension. ABG, arterial blood gas analysis; BNP, brain natriuretic peptide; CPET, cardiopulmonary exercise testing; CT, computed tomography; CTEPH, chronic thrombo-embolic pulmonary hypertension; ECG, electrocardiogram; HIV, human immunodeficiency virus; N, no; NT-proBNP, N-terminal pro-brain natriuretic peptide; PAH, pulmonary arterial hypertension; PE, pulmonary embolism; PFT, pulmonary function tests; PH, pulmonary hypertension; ReCo, recommendation; Y, yes. *Warning signs include rapid progression of symptoms, severely reduced exercise capacity, pre-syncope or syncope on mild exertion, signs of right heart failure. ¹Lung and heart assessment by specialist as per local practice. ²As indicated, CT pulmonary angiography recommended if PH suspected. ³Includes connective tissue disease (especially systemic sclerosis), portal hypertension, HIV infection, and family history of PAH. ⁴History of PE, permanent intravascular devices, inflammatory bowel diseases, essential thrombocythemia, splenectomy, high-dose thyroid hormone replacement, and malignancy.

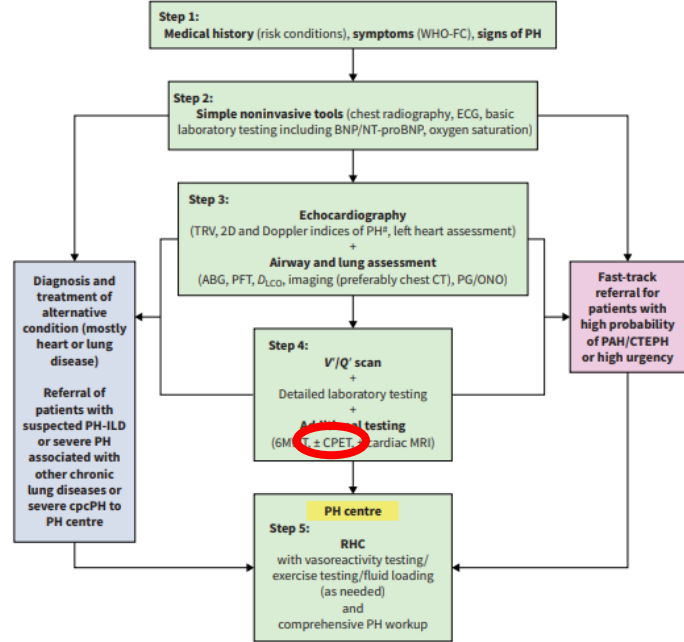


FIGURE 1 Suggested diagnostic approach to pulmonary hypertension (PH). Steps 1–5 represent the most important diagnostic steps of PH from the first presentation of the patient with symptoms or an existing risk condition through final diagnosis with invasive assessment. WHO-FC: World Health Organization functional class; BNP: brain natriuretic peptide; NT-proBNP: N-terminal pro-BNP; TRV: tricuspid regurgitation velocity; 2D: two-dimensional; ABG: arterial blood gases; PFT: pulmonary function testing; D_{LCO} : diffusion capacity of the lung for carbon monoxide; CT: computed tomography; PG: polygraphy; ONO: overnight oximetry; V/Q scan: ventilation/perfusion scan of the lung; 6MWT: 6-min walk test; CPET: cardiopulmonary exercise testing; MRI: magnetic resonance imaging; RHC: right heart catheterisation; PH-ILD: pulmonary hypertension associated with interstitial lung disease; PAH: pulmonary arterial hypertension; CTEPH: chronic thromboembolic pulmonary hypertension; cpcPH: combined post- and pre-capillary PH. ^a: refer to figure 2.

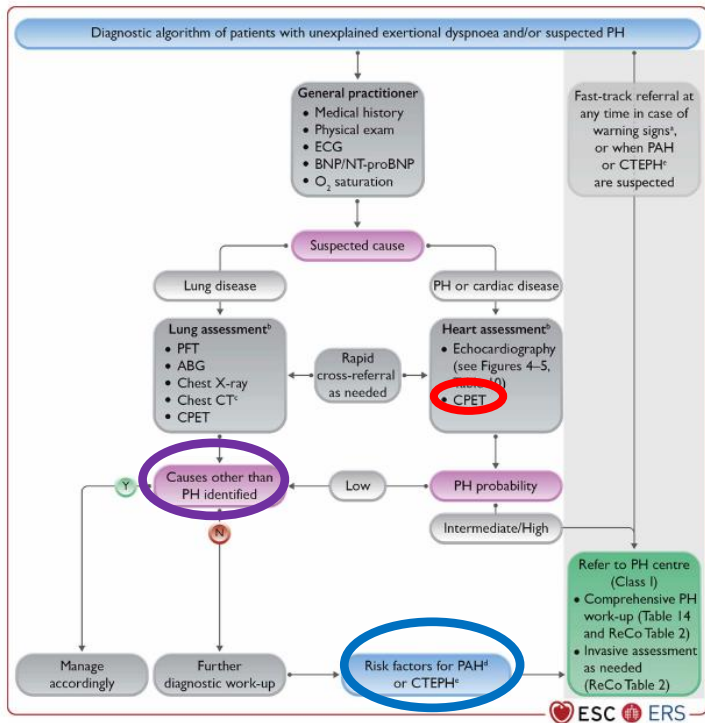


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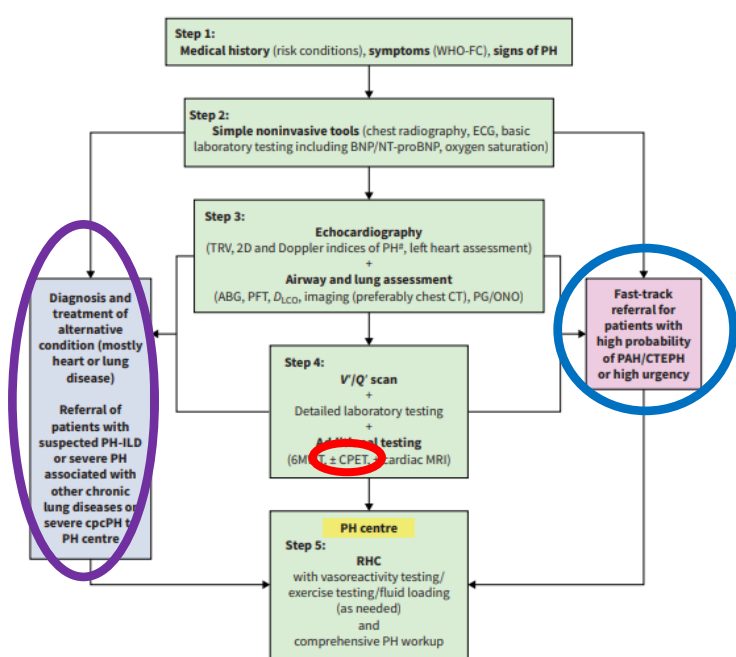


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- **Hyperventilation comme outil diagnostic** (Pente VE/VCO₂ et VE/VCO₂ SV₁)
 - Bonne sensibilité
normaux rendent le diagnostic d'HTP peu probable
 - Peu spécifique
augmentés sont en faveur d'une HTP
en l'absence de PID, Emphysème, Shunt D-G, I. Cardiaque, Sd hyperventilation
Sun XG, et al. Circulation. 2001
Weatherald J, et al. Eur Respir Review. 2020
- « CPET to detect **chronic thromboembolic pulmonary hypertension** in patients with normal echocardiography ». *Held M, et al. Respiration 2014.*
- « CPET for detecting pulmonary arterial hypertension in **systemic sclerosis** ». *Dumitrescu D, et al. Heart 2017*
- « Screening for PAH in **adults carrying a BMPR2 mutation** ». *Montani D, et al. ERJ 2020.*

In symptomatic patients with intermediate echocardiographic probability of PH, CPET may be considered to further determine the likelihood of PH

IIb

In symptomatic patients with SSc, exercise echocardiography or CPET, or CMR may be considered to aid decisions to perform RHC

IIb

In populations at risk of PAH, such as those with SSc, a normal peak VO₂ seems to exclude the diagnosis of PAH .

ESC/ERS Guidelines: Humbert M, et al. ERJ. 2022

In diagnosing PH, we suggest CPET use in selected cases, when no clear decision on whether to perform RHC can be made based on echocardiography and other noninvasive tools.

Proposal from WSPH: Kovacs G, et al. ERJ 2024

- Aide au dépistage dans des populations sélectionnées en l'absence d'atteinte parenchymateuse ou shunt D-G

Evaluation de la sévérité

TABLE 3. Resting and Exercise Values in Normal Subjects and PPH Patients Categorized According to Severity of Reduction in CPET Aerobic Capacity

	Normal* (n=20)	Mild PPH (n=3)	Moderate PPH (n=14)	Severe PPH (n=22)	Very Severe PPH (n=14)
Peak \dot{V}_O range, % pred	82–132	65–79	50–64	35–49	<35
Peak \dot{V}_{O_2} , % pred	101±19	70±4	58±4	42±5	27±4
Peak \dot{V}_{O_2} , mL · min ⁻¹ · kg ⁻¹	29.5±6.6	14.5±3.3	12.5±2.2	11.2±2.6	8.1±1.7
AT, % pred	104±16	85±7	75±10	57±9	41±7
AT, mL · min ⁻¹ · kg ⁻¹	16.3±3.9	10.4±2.3	9.7±1.3	8.7±2.2	6.8±1.3
Peak O ₂ pulse, % pred	108±25	86±11	73±8	56±11	39±5
Peak HR, % pred	96±13	83±12	80±8	77±12	70±13
$\Delta\dot{V}_{O_2}/\Delta WR$, mL · min ⁻¹ · W ⁻¹	9.6±0.9	8.3±0.5	7.0±1.5	6.0±1.0	5.6±1.3
$\dot{V}_E/\dot{V}_{CO_2}@AT$, % pred	99±12	142±22	149±21	161±25	219±76
$\dot{V}_E/\dot{V}_{CO_2}@AT$, absolute	29±4	43±6	45±7	46±8	62±20
\dot{V}_E -vs- \dot{V}_{CO_2} slope, % pred	88±11	164±49	148±27	141±32	215±123
\dot{V}_E -vs- \dot{V}_{CO_2} slope, absolute	25±3	49±14	45±9	40±10	60±32
Peak \dot{V}_E , as % MVV	70±15	63±19	54±9	47±11	43±16
MRT, s	12±10	34±9	37±14	47±13	64±15
mPAP, mm Hg	...	48±17	63±14	70±18	57±17
CO, L · min ⁻¹	...	5.1±1.1	4.4±1.4	3.5±1.0	3.8±1.2
PVR, mm Hg · L ⁻¹ · min ⁻¹	...	8±4	15±8	18±5	14±6
NYHA class	...	2.0±0.4	2.5±0.5	2.8±0.6	3.3±0.4

*Each CPET parameter of all PPH patients is significantly different from that of normal control subjects ($P<0.001$).

20 patients sans HTAP vs 53 HTAP

Sun XG. Exercise Pathophysiology in Patients with Primary Pulmonary Hypertension. Circulation 2001

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20 patients sans HTAP vs 53 HTAP

VO2 Pic

SV1

VO2/FC
FC max

VE/VCO2



Corrélées
à la sévérité

Sun XG. Exercise Pathophysiology in Patients with Primary Pulmonary Hypertension. Circulation 2001

TABLE 16 Comprehensive risk assessment in pulmonary arterial hypertension (three-strata model)

Determinants of prognosis (estimated 1-year mortality)	Low risk (<5%)	Intermediate risk (5–20%)	High risk (>20%)
Clinical observations and modifiable variables			
Signs of right HF	Absent	Absent	Present
Progression of symptoms and clinical manifestations	No	Slow	Rapid
Syncope	No	Occasional syncope ^a	Repeated syncope ^b
WHO-FC	I, II	III	IV
6MWD ^c	>440 m	165–440 m	<165 m
CPET	Peak VO ₂ >15 mL/min/kg (>65% pred.)	Peak VO ₂ 11–15 mL/min/kg (35–65% pred.)	Peak VO ₂ <11 mL/min/kg (<35% pred.)
	VE/VCO ₂ slope <36	VE/VCO ₂ slope 36–44	VE/VCO ₂ slope >44
Biomarkers: BNP or NT-proBNP ^d	BNP <50 ng/L NT-proBNP <300 ng/L	BNP 50–800 ng/L NT-proBNP 300–1100 ng/L	BNP >800 ng/L NT-proBNP >1100 ng/L
Echocardiography	RA area <18 cm ² TAPSE/sPAP >0.32 mm/mmHg No pericardial effusion	RA area 18–26 cm ² TAPSE/sPAP 0.19–0.32 mm/mmHg Minimal pericardial effusion	RA area >26 cm ² TAPSE/sPAP <0.19 mm/mmHg Moderate or large pericardial effusion
cMRI ^e	RVEF >54% SVI >40 mL/m ² RVESVI <42 mL/m ²	RVEF 37–54% SVI 26–40 mL/m ² RVESVI 42–54 mL/m ²	RVEF <37% SVI <26 mL/m ² RVESVI >54 mL/m ²
Haemodynamics	RAP <8 mmHg CI ≥2.5 L/min/m ² SVI >38 mL/m ² SvO ₂ >65%	RAP 8–14 mmHg CI 2.0–2.4 L/min/m ² SVI 31–38 mL/m ² SvO ₂ 60–65%	RAP >14 mmHg CI <2.0 L/min/m ² SVI <31 mL/m ² SvO ₂ <60%

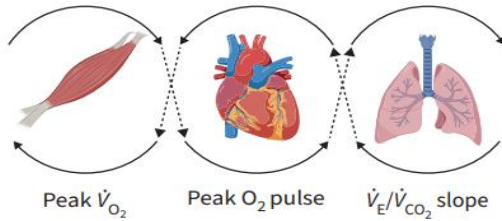
DM6 en m
Pic VO₂ absolue et % théo
Pente VE/VCO₂

6MWD, 6-minute walking distance; BNP, brain natriuretic peptide; CI, cardiac index; cMRI, cardiac magnetic resonance imaging; CPET, cardiopulmonary exercise testing; HF, heart failure; NT-proBNP, N-terminal pro-brain natriuretic peptide; PAH, pulmonary arterial hypertension; pred., predicted; RA, right atrium; RAP, right atrial pressure; sPAP, systolic pulmonary arterial pressure; SvO₂, mixed venous oxygen saturation; RVESVI, right ventricular end-systolic volume index; RVEF, right ventricular ejection fraction; SVI, stroke volume index; TAPSE, tricuspid annular plane systolic excursion; VE/VCO₂, ventilatory equivalents for carbon dioxide; VO₂, oxygen uptake; WHO-FC, World Health Organization functional class. ^aOccasional syncope during heavy exercise or occasional orthostatic syncope in a stable patient. ^bRepeated episodes of syncope even with little or regular physical activity. ^cObserve that 6MWD is dependent upon age, height, and burden of comorbidities. ^dTo harmonize with the four-strata model shown in Table 18, the BNP and NT-proBNP cut-off levels have been updated from the 2015 version based on data from the REVEAL registry, acknowledging that the European validation studies have used the original cut-off levels [274, 292, 293, 295, 296, 302]. ^ecMRI parameters adapted from Section 6.2.2.2.

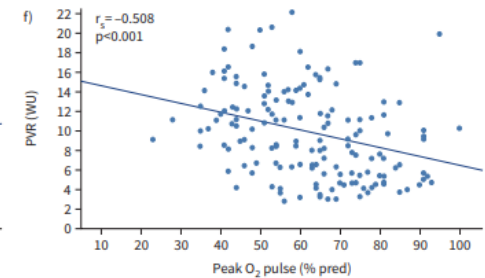
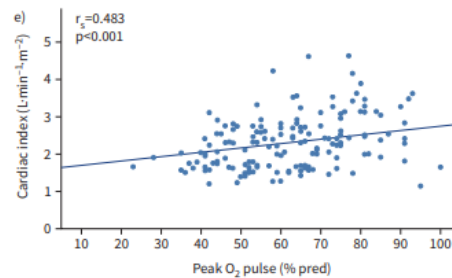
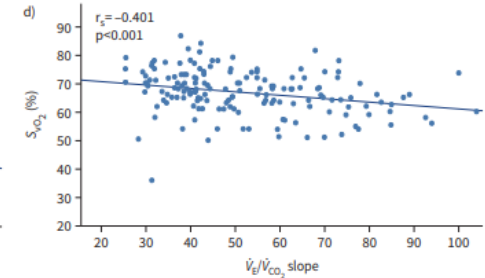
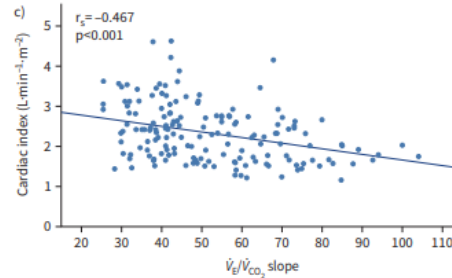
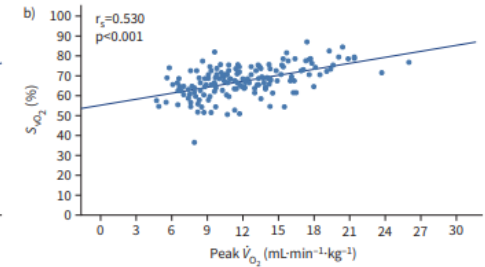
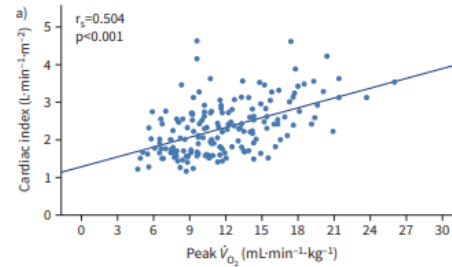
*Humbert M. ESC/ERS Guidelines.
ERJ. 2022*

Score EFX

262 HTAP incidentes



	VO2 pic ml.min.kg	VO2/FC pic % théorique	Pente VE/VCO2
IC	$r=0.504$ $p<0.001$	$r=0.483$ $p<0.001$	$r=-0.467$ $p<0.001$
SVO ₂	$r=0.530$ $p<0.001$		$r=-0.401$ $p<0.001$
RVP		$r=-0.508$ $p<0.001$	

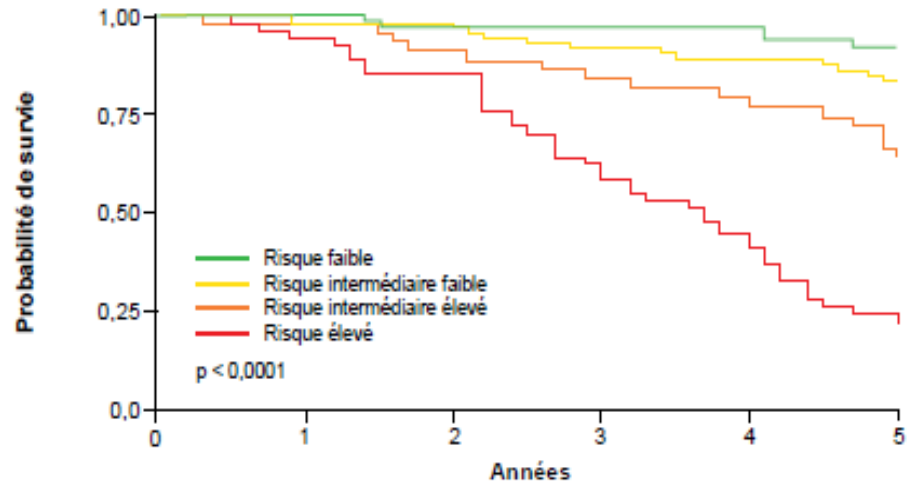


Score EFX seul

EFX à la première réévaluation
Prédiction de la survie à long terme

Catégorie de risque	Risque faible	Risque intermédiaire	Risque élevé
Points attribués	1	2	3
$\dot{V}O_2$ au pic ($\text{mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$)	> 15	11-15	< 11
Pente $\dot{V}E/\dot{V}CO_2$	< 36	36-44	> 44
Pouls d' O_2 au pic (% prédite)	> 65	40-65	< 40

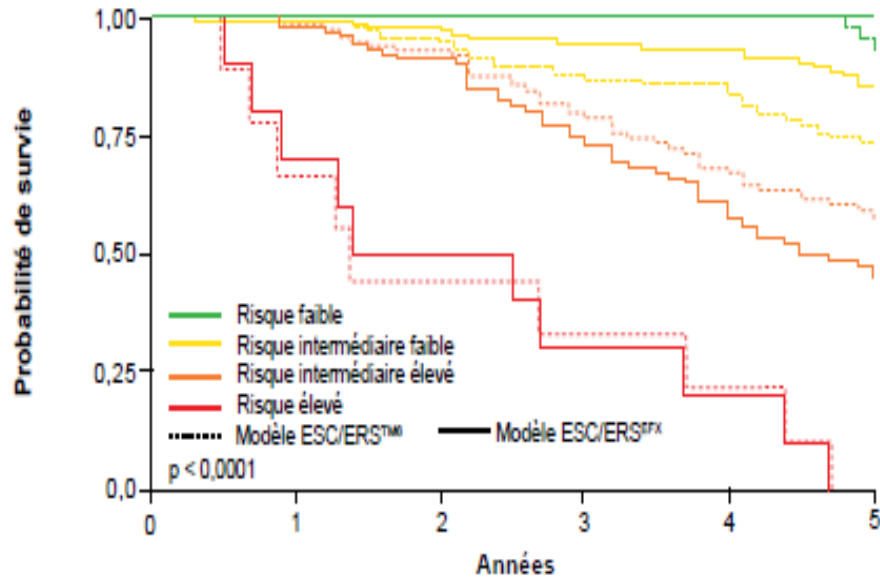
Classe de risque	Risque faible	Risque intermédiaire faible	Risque intermédiaire élevé	Risque élevé
Score EFX	1-1,49	1,5-1,99	2-2,49	2,5-3



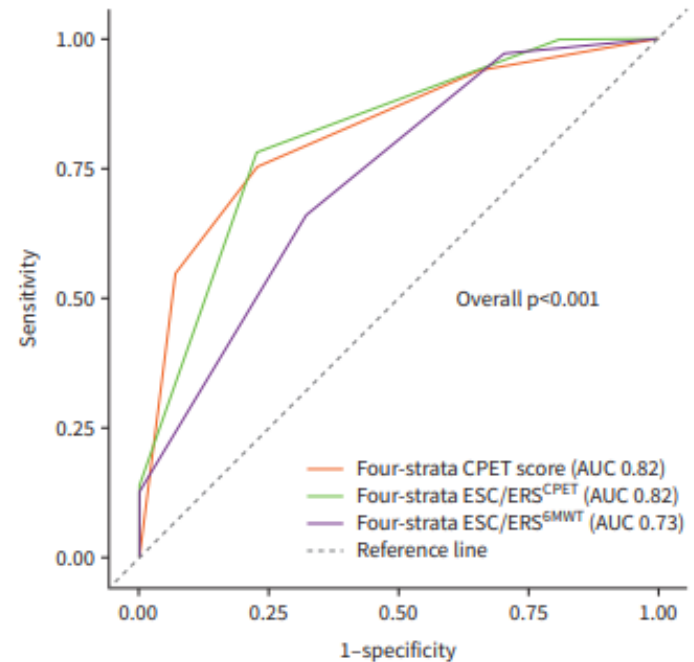
Score composite NYHA - NT pro BNP

+ Score EFX ————

+ DM6 - - - - -



Baccelli A et al, ERJ 2025



Score EFX seul AUC 0.82

Score NYHA-NT pro BNP-EFX AUC 0.82

Score NYHA-NT pro BNP-TM6 AUC 0.73

Facteur limitant à l'exercice

- Dépistage de **comorbidités**: 20% des patients HTAP \geq 2 comorbidités. *Boucly A. ERJ. 2025*
- Diminution de la masse musculaire chez 42% des patients HTAP *Dunefsky Z, et al. AJRCCM. 2025*
Mise en évidence d'une **limitation périphérique**, indication de réhabilitation cardio respiratoire
- Evaluation des **dyspnées post EP**



Patient de 73 ans

EP 2008, 2016, 2023 (mutation hétérozygote du facteur V)

Dyspnée en stade 3 de la NYHA, persistante post EP

Scintigraphie V/Q: 3 défauts sous segmentaires perfusionnels systématisés

ETT: ViT 2.1m/s, PAPS 22mmHg, TAPSE 25mm

Dépense Métabolique	Repos	Seuil 1	%ThéoMax	VO2 Max.	Théo Max	%
Temps [min]	02:16	08:18	-	11:48	-	-
Watt [W]	0	50	48	90	104	86
MET	1.5	3.8	-	5.1	-	-
V'O2 [mL/min]	364	936	57	1270	1635	78
V'O2/kg [(mL/min)/kg]	5.1	13.2	57	17.9	23.0	78
V'CO2 [mL/min]	361	916	-	1408	-	-
VCO2kg [(mL/min)/kg]	5.09	12.91	-	19.83	-	-
QR	0.99	0.98	-	1.11	-	-

Régime Ventilatoire

V'E [L/min]	19	46	51*	76	90*	85*
VTex [L]	0.785	1.606	-	1.996	-	-
FR [1/MIN]	24.2	28.4	-	38.3	41.6	-
EqO2	44.2	45.1	-	56.5	-	-
EqCO2	44.5	46.0	-	51.0	-	-
Réserve Resp. VEMS [%]	79	49	-	15	28	-
SpO2 [%]	100	99	-	98	-	-

Cardio Circulaire

FC [1/MIN]	87	105	71	125	147	85
PoulsO2 [mL]	4.2	8.9	80	10.2	11.1	91
Psys [mmHg]	144	163	-	174	-	-
Pdia [mmHg]	87	85	-	83	-	-
Pcirc [mmHg*L/(kg*min)]	0.738	2.148	-	3.113	-	-

Gaz du sang

	Repos			Fin Effort		
Temps [min]	02:00	-	-	11:30	-	-
PO2 art. [mmHg]	96.80	-	-	79.50	-	-
PCO2 Art. [mmHg]	33.20	-	-	32.40	-	-
pHa	7.41	-	-	7.36	-	-
SaO2 [%]	98	-	-	95	-	-
PAO2 [mmHg]	113.09	-	-	116.25	-	-
AaDO2 [mmHg]	16.29	-	-	36.75	-	-
PETO2 [mmHg]	125.86	-	-	126.71	-	-
PETCO2 [mmHg]	24.88	-	-	22.58	-	-
Pa-ETCO2 [mmHg]	8.32	-	-	9.82	-	-
VDf/VT [%]	33	-	-	42	-	-
Lactates [mmol/L]	0.8	-	-	3.4	-	-

Pentes

		Valeur
Pente VE/VCO2 :	VECO2s	52.92
Pente VO2/Watts :	VO2Ws [(mL/min)/W]	7.33

POD mmHg	5
PAPm mmHg	14
PAPO mmHg	7
IC l/min/m ²	2.8
RVP uw	0.9



Maladie thrombo embolique chronique sans HTP

Proposition d'angioplastie des AP

Conclusion

Guidelines 2022 et World Symposium 2024: Absence de recommandation forte d'utilisation

TABLE 17 Suggested assessment and timing for the follow-up of patients with pulmonary arterial hypertension

	At baseline	3–6 months after changes in therapy ^a	Every 3–6 months in stable patients ^a	In case of clinical worsening
Medical assessment (including WHO-FC)	Green	Green	Green	Green
6MWT	Green	Green	Green	Green
Blood test (including NT-proBNP) ^{b,c}	Green	Green	Green	Green
ECG	Green	Green	Green	Green
Echocardiography or cMRI	Green	Green	Green/Orange	Green
ABG or pulse oximetry ^d	Green	Green	Green	Green
Disease-specific HR-QoL	Orange	Orange	Orange	Orange
CPET	Orange	Orange	Orange	Orange
RHC	Green	Yellow	Orange	Yellow

Class IIb Usefulness/efficacy is less well established by evidence/opinion

May be considered

However, the added value of CPET on top of common clinical and haemodynamic variables remains largely unexplored.

Conclusion

- Nouvelles guidelines attendues en 2026

EFX est un outil sûr et non invasif avec un intérêt pour

- Dépistage de l'HTAP (en l'absence de comorbidités respiratoires)
et des maladies thromboemboliques chroniques (avec ou sans HTP)
- Prise en charge
 - Stratification du risque
 - Suivi des patients
 - Identification des facteurs limitants à l'exercice (comorbidités)
 - Indication et modalités de la réhabilitation cardio-respiratoire

