



FORUM EUROPÉEN, CŒUR, EXERCICE & PRÉVENTION

Les Épreuves d'Effort



Les autres paramètres: PETO₂ et PETCO₂

Miguel Mendes



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Conflicts of interest

- None



Chers collègues, Chers amis,

Nous avons le plaisir de nous retrouver à l'occasion de cette 18^{ème} édition du « Forum Européen, Cœur, Exercice et Prévention », à Paris, les 20 et 21 mars 2025.

Nous aborderons cette année les nouveautés et les sujets d'actualité en réadaptation, épreuve d'effort, cardiologie du sport et de façon plus générale en prévention.

Les meilleurs experts français et européens présenteront les dernières nouveautés aux cardiologues praticiens, comme à ceux impliqués dans la réadaptation, la prévention et la cardiologie du sport. Nos amis paramédicaux ont également un programme qui leur est dédié auquel ils sont très fidèles. Les ateliers pratiques seront aussi organisés cette année, de même qu'une session posters conclue par la remise d'un prix.

Cette année, nous aurons une session spéciale sur la **cardiomyopathie hypertrophique** pour laquelle les connaissances ont beaucoup évolué. Nous aurons également une session consacrée aux **risques cardiorespiratoires des BPCO**, qui sont mal connus des cardiologues. Nous aborderons des **cas difficiles en réadaptation**, ainsi que des **données récentes sur l'apport de la vaccination en cardiologie**. Nous discuterons également des **situations cardiologiques extrêmes** comme la plongée, l'altitude, l'apesanteur. Nous aurons aussi des ateliers sur l'**épreuve d'effort cardiorespiratoire**, sur la **télesurveillance**. Comme tous les ans, nous mettrons un point d'honneur à discuter avec vous de cas cliniques, qui nous posent tous les jours des problèmes majeurs.

Nous espérons vous retrouver tous nombreux et heureux en mars 2025 au Forum. Nous vous souhaitons un très bon congrès.

Alain Cohen Solal

François Carré

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REVIEW

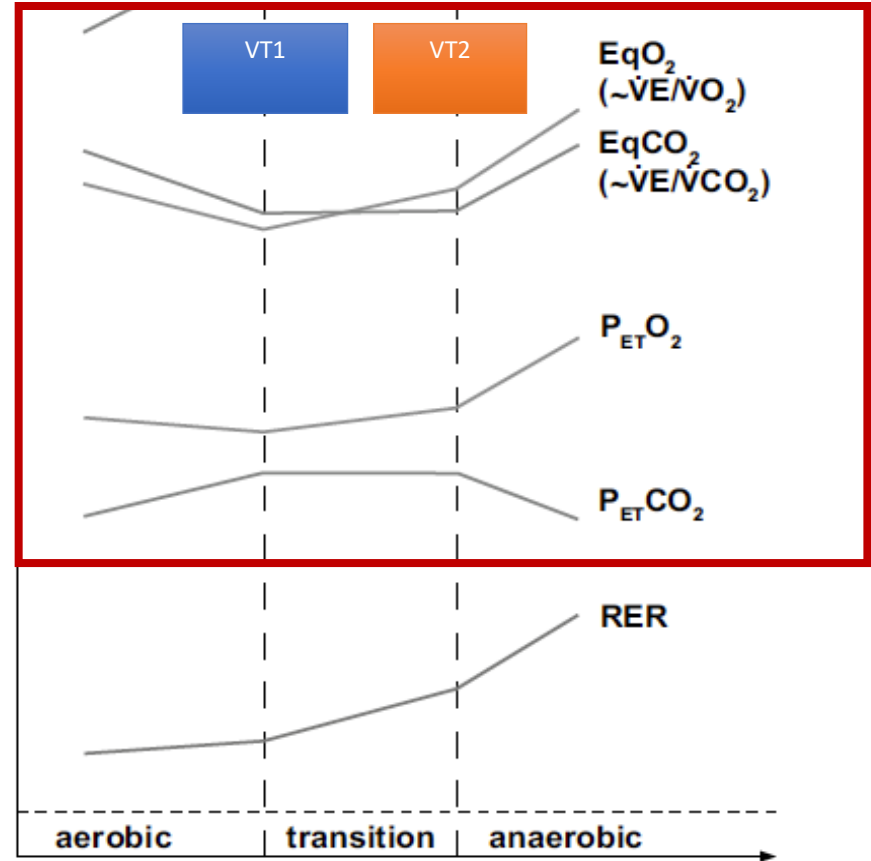
Open Access



Practical guide to cardiopulmonary exercise testing in adults

Thomas Glaab^{1,2*} and Christian Taube³

- Rest to VT1: $P_{ET}CO_2$ increases and $P_{ET}O_2$ decreases, similarly to VE/VO_2 .
- VT1 to VT2: $P_{ET}O_2$ typically begins to increase frankly in concert with the increase in VE/VO_2 . $P_{ET}CO_2$ increases slightly and reaches its highest value at VT2, inversely to VE/VCO_2 which reaches its lowest value at VT₂.
- VT2 to the end of exercise: $P_{ET}CO_2$ drops and $P_{ET}O_2$ rises frankly.



THE PRESENT AND FUTURE

STATE-OF-THE-ART REVIEW

Cardiopulmonary Exercise Testing

What Is Its Value?

Marco Guazzi, MD, PhD,¹ Francesco Bandera, MD, PhD,² Cemal Ozemek, PhD,³ David Systrom, MD,^{4,5} Ross Arena, PhD⁶



TABLE 1 Clinical Stratification for Patients With HF

Primary CPET Variables			
VE/Vco ₂ Slope	Peak V _{O₂}	EOV	P _{ET} CO ₂
Ventilatory Class I VE/Vco ₂ slope <30.0	Ventilatory Class A Peak V _{O₂} >20.0 ml·kg ⁻¹ ·min ⁻¹	Not Present	Resting P _{ET} CO ₂ ≥33.0 mm Hg 3–8 mm Hg increase during ET
Ventilatory Class II VE/Vco ₂ slope 30.0–35.9	Ventilatory Class B Peak V _{O₂} = 16.0–20.0 ml·kg ⁻¹ ·min ⁻¹		
Ventilatory Class III VE/Vco ₂ slope 36.0–44.9	Ventilatory Class C Peak V _{O₂} = 10.0–15.9 ml·kg ⁻¹ ·min ⁻¹	Present	Resting P _{ET} CO ₂ <33.0 mm Hg <3 mm Hg increase during ET
Ventilatory Class IV VE/Vco ₂ slope ≥45.0	Ventilatory Class D Peak <10.0 ml·kg ⁻¹ ·min ⁻¹		
Standard ET Variables			
Hemodynamics	ECG	HRR	
Rise in systolic BP during ET	No sustained arrhythmias, ectopic foci, and/or ST-segment changes during ET and/or in recovery	>12 beats at 1 min recovery	
Flat systolic BP response during ET	Altered rhythm, ectopic foci, and/or ST-segment changes during ET and/or in recovery: did not lead to test termination	≤12 beats at 1 min recovery	
Drop in systolic BP during ET	Altered rhythm, ectopic foci, and/or ST-segment changes during ET and/or in recovery: led to test termination		
Patient Reason for Test Termination			
Lower extremity muscle fatigue	Angina	Dyspnea	

Santa Cruz's CPET report

PROVA DE ESFORÇO EM TAPETE ROLANTE

Estadio	Tempo	Vel.	Incli.	METS	FC	TA Sist.	TA Diast.
Repouso	0	0	0	0	80	140	80
I	3	3,9	11,5	4,8	107	140	80
II	3	5,3	13,5	6,1	133	150	70
III	3	6,6	15,5	6,9	142	160	60

Estadio	FC	TA Sist.	TA Diast.
Rec.1'	131	160	60
Rec.3'	100	180	70

RESULTADOS E COMENTÁRIOS

PE clássica	
Protocolo	Bruce
Tempo	9:04
Suspensão por	fadiga
PAS repouso	140
PAS esforço	180
Extrassístoles SV	ausentes
Extrassístoles V	simples
Angina	ausente
Alt. Seg. ST	ausente

PECR	
VO ₂ (mL/kg/min)	
Pico	24,6
% previsto	105
Repouso	5
VT1	13,1
VT1%pVO ₂	55,9
VT2	20,4
Quociente Resp.	1,27
O ₂ pulso % prev.	109

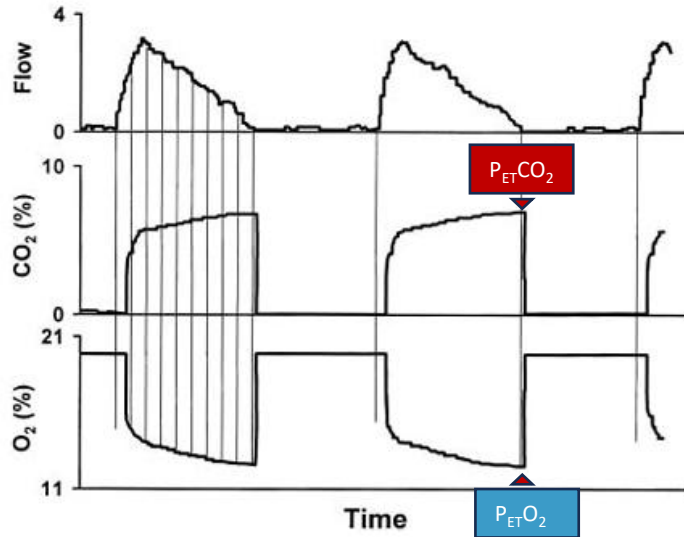
Ventilação	
FVC	3,25 L 92%
FEV1	2,57 L 95%
FEV1/FVC	79%
Reserv. Respiratória	7 %
Osc. Ventilatórias	ausentes
VE/VCO ₂	35
SpO ₂ repouso	100
SpO ₂ esforço	99
ΔP _{ET} CO ₂	1

Cronotrop. e Treino	
Repouso	80 bpm
VT1 (3km/h, 10%)	100 bpm
VT2 (5km/h, 14%)	122 bpm
Limiar de risco	bpm
Pico	142 bpm
% previsto	97 %
1° min. recup.	131 bpm
Reserva VO ₂ 17	99 bpm
Reserva VO ₂ 19	104 bpm

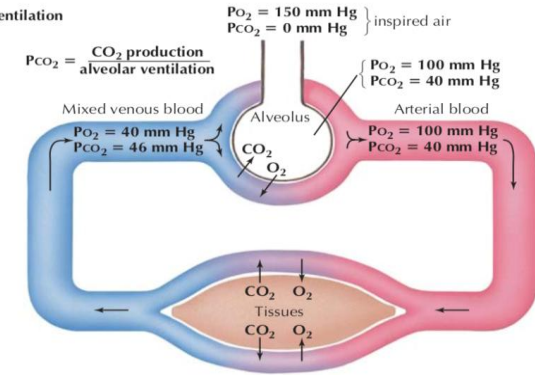
As cores são indicativas do prognóstico conferido pelo respetivo valor obtido nesta prova.

ATS/ACCP Statement on Cardiopulmonary Exercise Testing

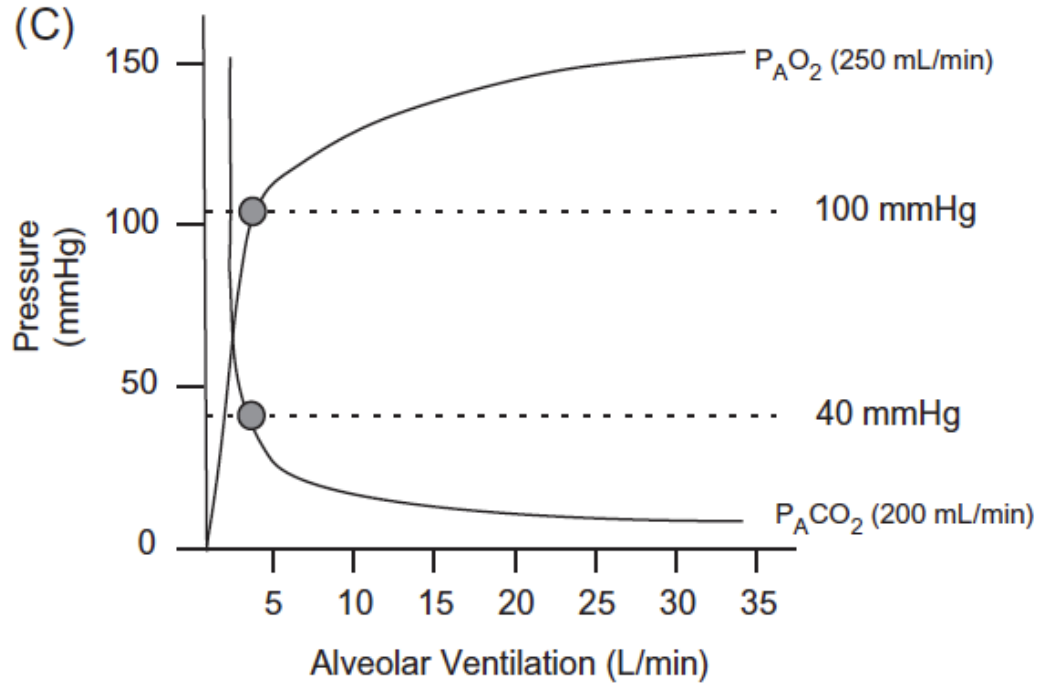
THIS JOINT STATEMENT OF THE AMERICAN THORACIC SOCIETY (ATS) AND THE AMERICAN COLLEGE OF CHEST PHYSICIANS (ACCP) WAS ADOPTED BY THE ATS BOARD OF DIRECTORS, MARCH 1, 2002 AND BY THE ACCP HEALTH SCIENCE POLICY COMMITTEE, NOVEMBER 1, 2001



A. Normal ventilation



- **P_{ET}CO₂ = PCO₂ (mmHg)** at the end of an exhalation. Commonly, the highest PCO₂ measured during the alveolar phase of the exhalation.
 - Normal values at rest: 36 - 44 mmHg
- **P_{ET}O₂ = PO₂ (mmHg)** at the end of an exhalation. Is usually the lowest PO₂ during the alveolar portion of the exhalation.
 - Normal values at rest: 100 - 120 mmHg



Arroyo JP, Schweickert AJ. Chapter 4 - The Respiratory Cycle. <https://doi.org/10.1016/B978-0-12-801768-5.00004-6>

At rest

Normal values at rest: 100 - 120 mmHg.

- Lower $P_{ET}O_2$ levels are associated with:
 - **Age:** slightly lower values in the elderly due to changes in pulmonary function.
 - **Pulmonary diseases:** restrictive pulmonary diseases (eg, pulmonary fibrosis) and PAH, reduce pulmonary perfusion and increase the alveolar-arterial oxygen gradient.
 - **Cardiovascular diseases:** HF can affect pulmonary perfusion and gas exchange.
 - **Neuromuscular diseases:** the weakness of the respiratory muscles can lead to a decrease in alveolar ventilation.

Exercise

Normal Response:

- Usually, there is short decrease until VT1 and an increase from VT1 until the peak effort.
- The interpretation of $P_{ET}O_2$ should always consider the patient's clinical picture, including a history of pulmonary or cardiac diseases.
- The isolated use of $P_{ET}O_2$ is rare in cardiopulmonary exercise testing.
- Other CPET parameters (VO_2 , VCO_2 , VE, SpO_2) are essential for a comprehensive assessment.

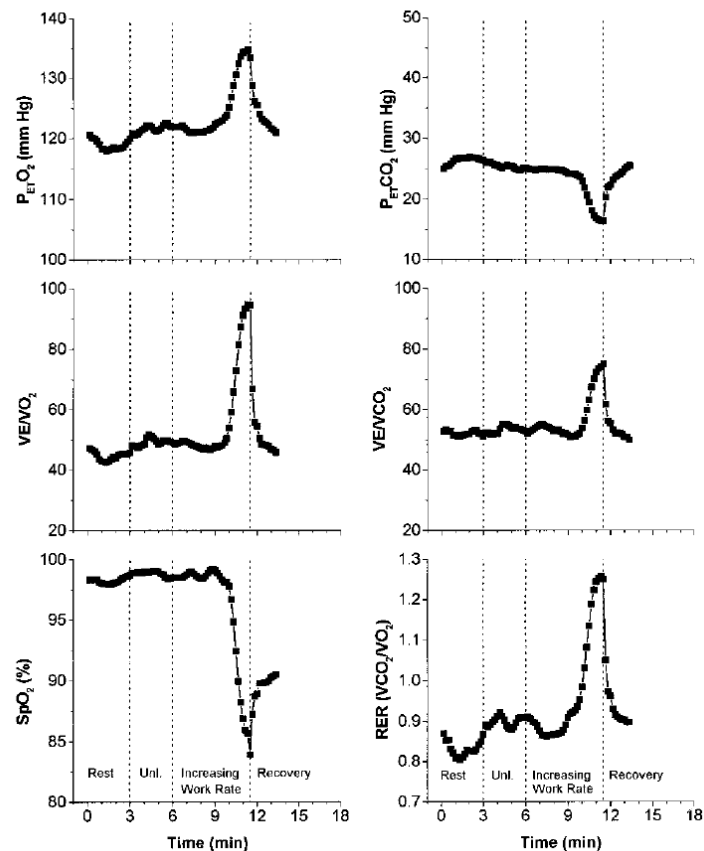
Gas Exchange Detection of Exercise-Induced Right-to-Left Shunt in Patients With Primary Pulmonary Hypertension

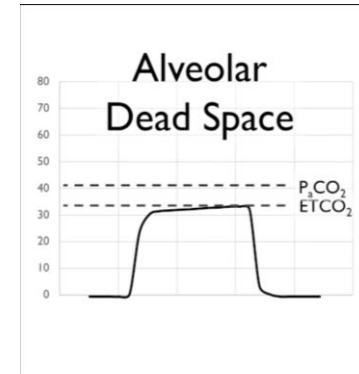
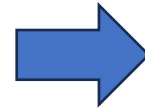
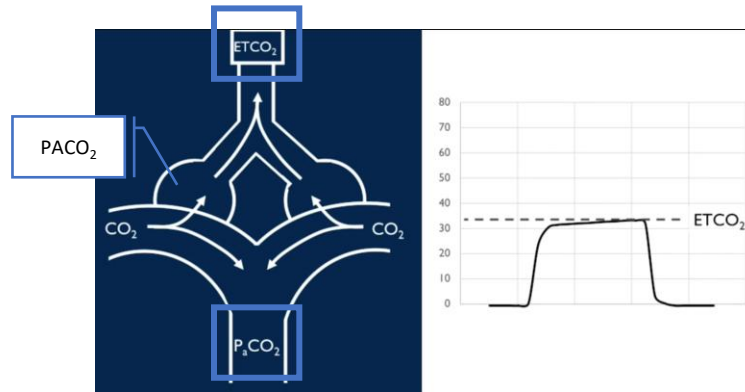
Xing-Guo Sun, MD; James E. Hansen, MD; Ronald J. Oudiz, MD; Karlman Wasserman, MD, PhD

Background—Because of high pulmonary vascular resistance in patients with primary pulmonary hypertension (PPH), right atrial pressure may exceed left atrial pressure during exercise, resulting in a right-to-left shunt via a patent foramen ovale (PFO). This shunting would disturb arterial PCO_2 and H^+ homeostasis if the pulmonary blood were not simultaneously hyperventilated to compensate for the high CO_2 and H^+ in the shunted blood. This article first hypothesizes and then describes unique changes in gas exchange when right-to-left exercise-induced shunting (EIS) occurs.

Methods and Results—Retrospectively, the cardiopulmonary exercise tests of 71 PPH patients were studied. Criteria postulated to document hyperventilation of the pulmonary blood flow due to a right-to-left EIS were (1) an abrupt and sustained increase in end-tidal O_2 with a simultaneous sustained decrease in end-tidal CO_2 ; (2) an abrupt and sustained increase in the respiratory exchange ratio; and (3) usually, an associated decline in pulse oximetry saturation. Each patient was evaluated for a PFO with resting echocardiography. The investigators interpreting the gas exchange evidence of EIS were blinded to the echocardiographic readings. Forty-five percent of the patients had demonstrable EIS by gas exchange criteria. Almost all were also positive for a PFO by echocardiography. Using the resting echocardiograph as the reference, the sensitivity, specificity, positive and negative predictive values, and accuracy were all between 90% to 96%.

Conclusions—Exercise-induced right-to-left shunting can be detected by noninvasive, cardiopulmonary exercise testing in patients with PPH. (*Circulation*. 2002;105:54-60.)



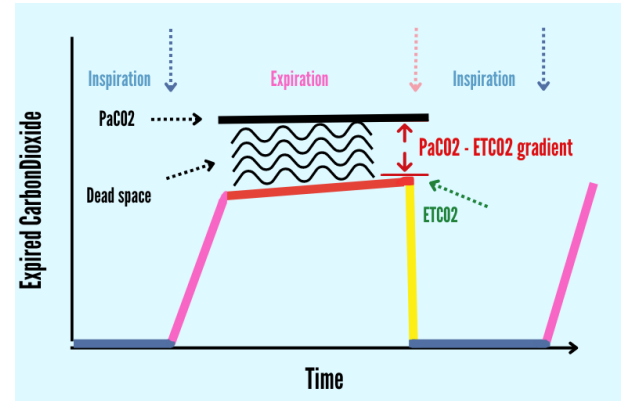


- PaCO₂ = Partial pressure of CO₂ in the pulmonary artery
- PACO₂ = Partial pressure of CO₂ in the alveoli
- P_{ET}CO₂ = Partial pressure of CO₂ at the end of exhalation measured at the mouth piece

$P_{ET}CO_2$

- $P_{ET}CO_2$ is an indirect measurement of pulmonary perfusion and ventilatory efficiency, being the result of $PACO_2$ and ventilation changes.
- $P_{ET}CO_2$ at rest is ~ 2 mmHg below $PACO_2$, but the relation of $P_{ET}CO_2$ is much less exact during exercise and it will be overridden by an exaggerated response to exercise.

- An arbitrary scale for maximum $P_{ET}CO_2$ is:
 - Normal: $\geq 35 - 45$
 - Mildly reduced = 30-35
 - Moderated reduced = 25 - 30
 - Severely reduced < 25



- During exercise, it's normal when it increases by 3 - 8 mmHg till VT2

Rest

- Hypocapnia (< 35 mmHg) may indicate:
 - Hyperventilation
 - Anxiety
 - Pulmonary diseases
- Hypercapnia (> 45 mmHg) may indicate:
 - Hypoventilation
 - Obstructive pulmonary diseases
 - Neuromuscular diseases.

Exercise

- A reduced variation may indicate airflow limitation, decreased cardiac output, or impaired gas diffusion.
- A sharp decrease may indicate heart failure, excessive hyperventilation, ventilation / perfusion mismatch, or an increase in physiological dead space.
- Elevated values (hypercapnia) generally indicate hypoventilation, meaning inadequate ventilation to eliminate the CO₂.

- 200 patients underwent cardiac surgery with cardiopulmonary bypass (CPB), from Jun 1996 to Jun 1997. $P_{ET}CO_2$ measurement at the end of CPB.
- When metabolism and ventilation are controlled, $P_{ET}CO_2$ reflects pulmonary blood flow and, therefore cardiac output (CO):
 - A $P_{ET}CO_2 < 20$ mmHg is associated with low CO ($< 2L/min$), even when other hemodynamic parameters are within normal ranges.
 - A $P_{ET}CO_2 \sim 27$ mmHg indicates that the CO is sufficient for CPB weaning, provided that other hemodynamic and metabolic parameters were adequate.
 - No patient required to return to CPB due to cardiopulmonary failure if $P_{ET}CO_2 > 26$.

End-tidal CO₂ Pressure Decreases During Exercise in Cardiac Patients

Association With Severity of Heart Failure and Cardiac Output Reserve

Akihiro Matsumoto, MD, Haruki Itoh, MD, FACC,* Yoko Eto, MD, Toshio Kobayashi, MD,*
Makoto Kato, MD,* Masao Omata, MD, Hiroshi Watanabe, MD,* Kazuzo Kato, MD, FACC,*
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OBJECTIVES

We measured end-tidal CO₂ pressure (PETCO₂) during exercise and investigated the relationship between PETCO₂ and exercise capacity, ventilatory parameters and cardiac output to determine the mechanism(s) of changes in this parameter.

BACKGROUND METHODS

It is unclear whether PETCO₂ is abnormal at rest and during exercise in cardiac patients. Cardiac patients (n = 112) and normal individuals (n = 29) performed exercise tests with breath-by-breath gas analysis, and measurement of cardiac output and arterial blood gases.

RESULTS

PETCO₂ was lower in patients than in normal subjects at rest and decreased as the New York Heart Association class increased, whereas the partial pressure of arterial CO₂ did not differ among groups. Although PETCO₂ increased during exercise in patients, it remained lower than in normal subjects. PETCO₂ in relation to cardiac output was similar in patients and normal subjects. PETCO₂ at the respiratory compensation point was positively correlated with the O₂ uptake (r = 0.583, p < 0.0001) and the cardiac index at peak exercise (r = 0.582, p < 0.0001), and was negatively correlated with the ratio of physiological dead space to the tidal volume. The sensitivity and specificity of PETCO₂ to predict an inadequate cardiac output were 76.6% and 75%, respectively, when PETCO₂ at respiratory compensation point and a cardiac index at peak exercise that were less than the respective control mean-2 SD values were considered to be abnormal.

CONCLUSIONS

PETCO₂ was below normal in cardiac patients at rest and during exercise. PETCO₂ was correlated with exercise capacity and cardiac output during exercise, and the sensitivity and specificity of PETCO₂ regarding decreased cardiac output were good. PETCO₂ may be a new ventilatory abnormality marker that reflects impaired cardiac output response to exercise in cardiac patients diagnosed with heart failure. (J Am Coll Cardiol 2000;36:242-9) © 2000 by the American College of Cardiology

Cardiac patients = 112
Normal individuals = 29

End-tidal CO₂ Pressure Decreases During Exercise in Cardiac Patients

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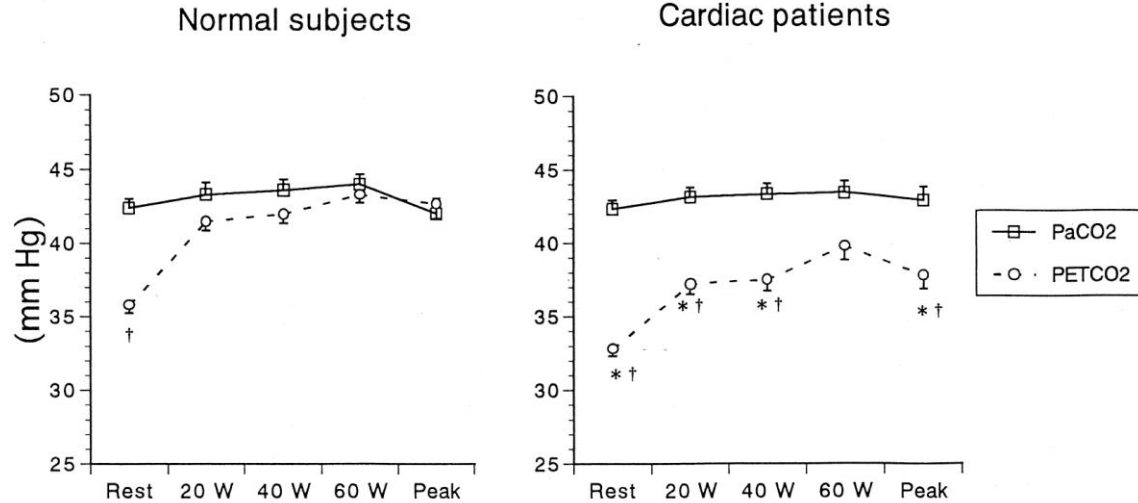


Figure 6. End-tidal CO₂ pressure (PETCO₂) and arterial PCO₂ (PaCO₂) at rest and during exercise in 53 cardiac patients and 15 normal control subjects. There was a significant difference in the change between PaCO₂ and PETCO₂ variables and interaction with time in both the patients (variables $p = 0.0001$, interaction $p = 0.0001$, by ANOVA) and control subjects (variables $p = 0.0001$, interaction $p = 0.0001$, by ANOVA). Peak indicates peak exercise. Values are mean \pm SE. *, $p < 0.05$ versus normal controls. †; $p < 0.05$ versus PaCO₂.

End-tidal CO₂ Pressure Decreases During Exercise in Cardiac Patients

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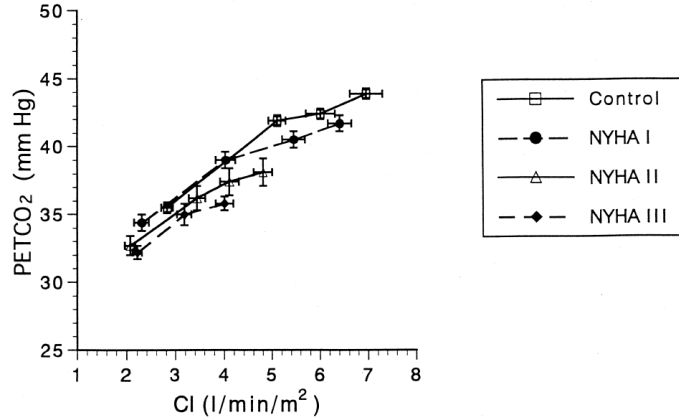


Figure 3. End-tidal CO₂ pressure (PETCO₂) in relation to the cardiac index (CI) at rest, at 20 W, 40 W, and 60 W during exercise according to the NYHA functional class. Values are mean \pm SE.

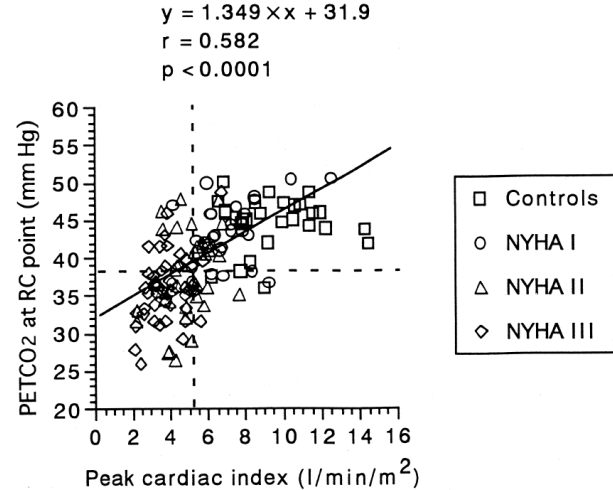


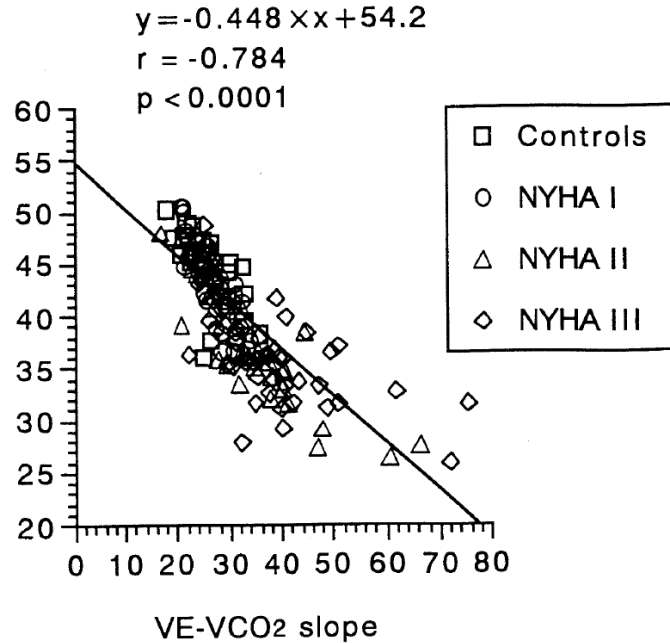
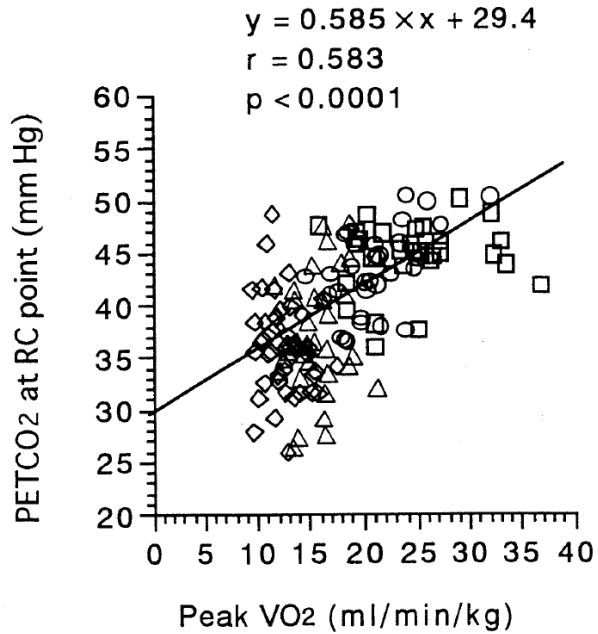
Figure 4. Relationships of end-tidal CO₂ pressure (PETCO₂) at the respiratory compensation (RC) point with the cardiac index at peak exercise in 112 cardiac patients and 29 normal subjects. Dotted lines indicate mean \pm 2SD values of PETCO₂ at the respiratory compensation point and the cardiac index at peak exercise in the normal control subjects.

End-tidal CO₂ Pressure Decreases During Exercise in Cardiac Patients

Association With Severity of Heart Failure and Cardiac Output Reserve

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End-Tidal CO₂ Pressure and Cardiac Performance during Exercise in Heart Failure

JONATHAN MYERS¹, PRADEEP GUJJA², SURESH NEELAGARU³, LEON HSU¹, TIMOTHY VITTORIO⁴, TAMIKA JACKSON-NELSON⁴, and DANIEL BURKHOF⁵

¹Cardiology Division, Veterans Affairs Palo Alto Health Care System, Stanford University, Palo Alto, CA; ²Texas Tech University of Health Sciences, Amarillo, TX; ³Lonestar Arrhythmia and Heart Failure Center, Amarillo, TX; ⁴Albert Einstein College of Medicine/Montefiore Medical Center, Bronx, NY; and ⁵Columbia University, New York, NY

ABSTRACT

MYERS, J., P. GUJJA, S. NEELAGARU, L. HSU, T. VITTORIO, T. JACKSON-NELSON, and D. BURKHOF. End-Tidal CO₂ Pressure and Cardiac Performance during Exercise in Heart Failure. *Med. Sci. Sports Exerc.*, Vol. 41, No. 1, pp. 18–24, 2009.

Introduction: In patients with heart failure (HF), end-tidal CO₂ pressure (PetCO₂) is related to ventricular function at rest and has been shown to predict prognosis. However, little is known about the association between ventricular performance and PetCO₂ responses to exercise. **Methods:** Forty-eight patients with HF and 13 normal subjects underwent cardiopulmonary exercise testing (CPX), while cardiac output and other hemodynamic measurements at rest and during exercise were obtained using a novel, noninvasive, bioreactance device based on assessment of relative phase shifts of electric currents injected across the thorax, heart rate, and ventricular ejection time. CPX responses and indices of cardiac performance were compared between normal subjects and HF patients achieving above and below a PetCO₂ of 36 mm Hg at the ventilatory threshold (PetCO₂@VT). **Results:** HF patients with an abnormal PetCO₂@VT (<36 mm Hg) had a lower exercise capacity, a lower $\dot{V}O_2$ @VT, a higher $\dot{V}_E/\dot{V}CO_2$ slope, and lower oxygen uptake efficiency slope (OUES) values compared with normal subjects and patients achieving a normal PetCO₂@VT. Patients with reduced PetCO₂@VT had lower peak cardiac output responses to exercise (20.0 ± 10 , 17.8 ± 6 , and 13.7 ± 7 L·min⁻¹ for normal subjects and HF patients with normal and abnormal PetCO₂ responses to exercise, respectively, $P = 0.04$). PetCO₂@VT was inversely related to the $\dot{V}_E/\dot{V}CO_2$ slope ($r = -0.78$, $P < 0.001$) and directly related to the OUES ($r = 0.55$, $P < 0.001$). **Conclusion:** Reduced PetCO₂ reflects impairments in the functional, ventilatory, and cardiac performance response to exercise in patients with HF. PetCO₂ can supplement other clinical and CPX indices in the functional and prognostic evaluation of patients with HF. **Key Words:** CARDIAC OUTPUT, OXYGEN UPTAKE, EXERCISE TESTING, VENTRICULAR FUNCTION, EXERCISE CAPACITY

- 48 HF patients | 13 normal subjects
- Compared regarding CO and CI at VT and peak exercise if P_{ET}CO₂ < 36 vs > 36 at VT

End-Tidal CO₂ Pressure and Cardiac Performance during Exercise in Heart Failure

JONATHAN MYERS¹, PRADEEP GUJJA², SURESH NEELAGARU³, LEON HSU¹, TIMOTHY VITTORIO⁴, TAMIKA JACKSON-NELSON⁴, and DANIEL BURKHOF⁵

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TABLE 3. Noninvasive cardiac output data at rest and peak exercise in normal subjects and CHF patients with normal and elevated PetCO₂ at the ventilatory threshold.

	Normals	CHF and PetCO ₂ @VT ≥ 36	CHF and PetCO ₂ @VT < 36	P value ^a
Rest				
Cardiac output (L·min ⁻¹)	5.45 ± 1.7	4.84 ± 1.4	4.77 ± 1.5	0.39
Cardiac index (L·min ⁻¹ ·M ⁻²)	2.89 ± 0.78	2.40 ± 0.56	2.54 ± 0.76	0.12
<i>dx/dt</i> (Ω·s ⁻¹)	171.3 ± 107	100.2 ± 64	148.3 ± 119	0.09
VET (ms)	174.3 ± 36	166.8 ± 25	157.4 ± 16	0.21
Ejection fraction	56.5 ± 9	45.0 ± 15	36.9 ± 17*	0.03
Peak exercise				
Cardiac output (L·min ⁻¹)	20.0 ± 10.0	17.8 ± 5.9	13.7 ± 6.6	0.04
Cardiac index (L·min ⁻¹ ·M ⁻²)	10.6 ± 4.7	9.0 ± 3.3	7.2 ± 3.2*	0.04
<i>dx/dt</i> (Ω·s ⁻¹)	557.8 ± 239	395.2 ± 203	362.7 ± 140*†	0.04
VET (ms)	143.6 ± 17	143.6 ± 12	149.3 ± 22	0.52

VT, ventilatory threshold; VET, ventricular ejection time.

^a P value represents ANOVA main effect between groups.

* P < 0.05 versus normal subjects.

† P < 0.05 versus CHF and PetCO₂ ≥ 36.

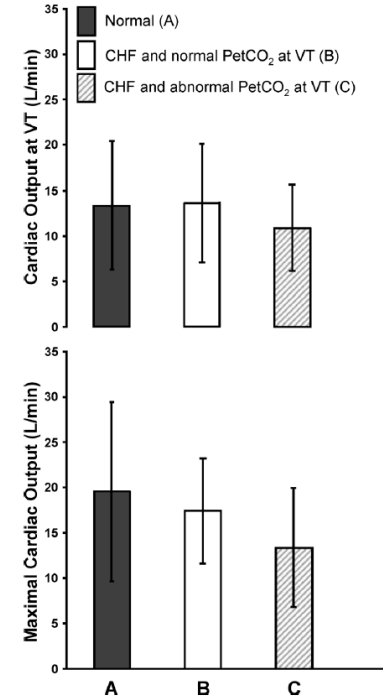


FIGURE 1—Cardiac output among normal subjects and CHF patients with normal and abnormal PetCO₂ responses to exercise at the ventilatory threshold (VT, top) and maximal exercise (bottom; ANOVA main effect $P = 0.03$). Error bars represent the SD.



Low partial pressure of end-tidal carbon dioxide predicts left ventricular assist device implantation in patients with advanced chronic heart failure

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ABSTRACT

Background: This study aimed to clarify the prognostic impact of partial pressure of end-tidal carbon dioxide (PETCO₂) in patients with advanced chronic heart failure (HF).

Methods: Forty-eight patients (mean age 43.1 ± 11.9 years, 32 males) with chronic HF (44 with non-ischemic and 4 with ischemic cardiomyopathy) were prospectively enrolled. Echocardiography, blood tests, pulmonary function testing, and PETCO₂ measurements were performed as noninvasive tests, whereas right heart catheterization and arterial blood gas analysis were conducted as invasive tests. The primary end point of this study was left ventricular assist device (LVAD) implantation or cardiac death.

Results: Eighteen patients underwent LVAD implantation at the Interagency Registry for Mechanically Circulatory Support (INTERMACS) profile 3 during the follow-up period, and no patient died. PETCO₂ was significantly lower in a stepwise manner with New York Heart Association functional class (class I or II, 34.2 ± 9.3 mmHg vs. class III or IV, 27.7 ± 2.5 mmHg; $p < 0.001$). Univariate and multivariate Cox proportional hazard models and time-dependent receiver operating characteristic curve analysis revealed that PETCO₂ ≤ 31 mmHg is an independent noninvasive predictor of LVAD implantation. Univariable and multivariable linear regression analyses showed that pulmonary arterial pressure was independently and highly correlated with PETCO₂ ($r^2 = -0.512$, $p < 0.001$).

Conclusions: Among various noninvasive clinical parameters investigated, PETCO₂ was the independent predictor of LVAD implantation at the INTERMACS profile 3 in patients with chronic HF. Pulmonary congestion may significantly contribute to decreases in PETCO₂ in patients with HF.

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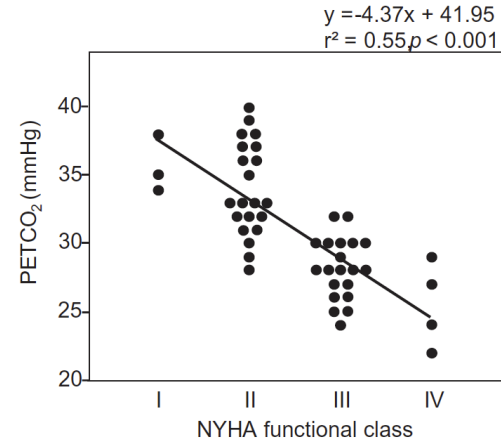
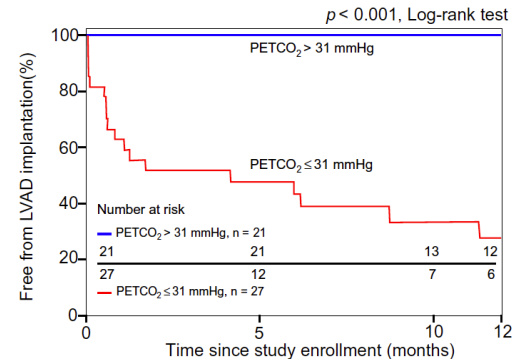


Fig. 1. Correlation analysis between partial pressure of end-tidal carbon dioxide (PETCO₂) and New York Heart Association (NYHA) functional class. Significant correlation was found between PETCO₂ and NYHA functional class ($y = -4.37x + 41.95$, $r^2 = 0.55$, $p < 0.001$).



Predictive Value of Cardiopulmonary Exercise Testing Parameters in Ambulatory Advanced Heart Failure



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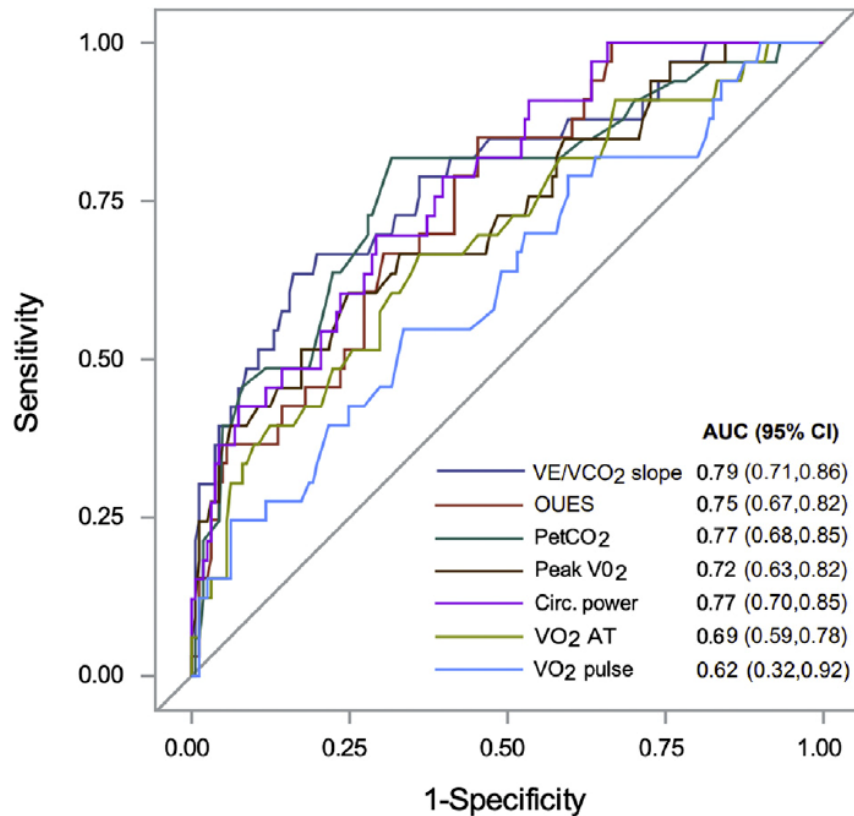
OBJECTIVES This study sought to determine cardiopulmonary exercise (CPX) predictors of the combined outcome of durable mechanical circulatory support (MCS), transplantation, or death at 1 year among patients with ambulatory advanced heart failure (HF).

BACKGROUND Optimal CPX predictors of outcomes in contemporary ambulatory advanced HF patients are unclear.

METHODS REVIVAL (Registry Evaluation of Vital Information for ventricular assist devices [VADs] in Ambulatory Life) enrolled 400 systolic HF patients, INTERMACS (Interagency Registry for Mechanically Assisted Circulatory Support) profiles 4-7. CPX was performed by 273 subjects 2 ± 1 months after study enrollment. Discriminative power of maximal (peak oxygen consumption [peak VO₂]; VO₂ pulse, circulatory power [CP]; peak systolic blood pressure • peak VO₂], peak end-tidal pressure CO₂ [PETCO₂], and peak Borg scale score) and submaximal CPX parameters (ventilatory efficiency [VE/VCO₂ slope]; VO₂ at anaerobic threshold [VO₂AT]; and oxygen uptake efficiency slope [OUES]) to predict the composite outcome were assessed by univariate and multivariate Cox regression and Harrell's concordance statistic.

RESULTS At 1 year, there were 39 events (6 transplants, 15 deaths, 18 MCS implantations). Peak VO₂, VO₂AT, OUES, peak PETCO₂, and CP were higher in the no-event group (all p < 0.001), whereas VE/VCO₂ slope was lower (p < 0.0001); respiratory exchange ratio was not different. CP (hazard ratio [HR]: 0.89; p = 0.001), VE/VCO₂ slope (HR: 1.05; p = 0.001), and peak Borg scale score (HR: 1.20; p = 0.005) were significant predictors on multivariate analysis (model C-statistic: 0.80).

CONCLUSIONS Among patients with ambulatory advanced HF, the strongest maximal and submaximal CPX predictor of MCS implantation, transplantation, or death at 1 year were CP and VE/VCO₂ slope, respectively. The patient-reported measure of exercise effort (Borg scale score) contributed substantially to the prediction of outcomes, a surprising and novel finding that warrants further investigation. (Registry Evaluation of Vital Information for VADs in Ambulatory Life [REVIVAL]; NCT01369407) (J Am Coll Cardiol HF 2021;9:226-36) © 2021 by the American College of Cardiology Foundation.



EDITORIAL COMMENT

Cardiopulmonary Exercise Testing-Based Risk Stratification in the Modern Era of Advanced Heart Failure Management*



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Degree to which CPET Measurements Reflect Exercise Hemodynamic Responses



VO₂ pulse
CavO₂ x Svol

AUC: 0.62

VO₂ peak
CavO₂ x Svol x HR

AUC: 0.69

Circulatory Power
CavO₂ x Svol x HR x SBP

Surrogate for cardiac power

AUC: 0.77

VE/VCO₂ and PETCO₂
Components: **Vd/Vt** and **PaCO₂**
Reflect RV perfusion of the pulmonary circulation

AUC: 0.79 and 0.77

**ROC Analysis
In Lala et al:**

- Lower $P_{ET}O_2$ levels are found in restrictive lung disease and PAH, heart failure and neuromuscular diseases.
- A decrease in $P_{ET}O_2$, indicates exercise-induced hypoxemia.
- An abrupt increase at the start of exercise may indicate R-L shunt or nonspecific hyperventilation.
- $P_{ET}O_2$ should be used with other CPET parameters (VO_2 , VCO_2 , VE, SpO_2) for a comprehensive assessment.

- The interpretation of $P_{ET}CO_2$ changes in a non-invasive CPET must be cautious. Nevertheless, its abnormalities suggest:
 - **at rest:**
 - $P_{ET}CO_2 < 35$ mmHg (**hypocapnia**): hyperventilation due to anxiety or pulmonary disease.
 - $P_{ET}CO_2$ at rest > 45 mmHg (**hypercapnia**): hypoventilation, obstructive pulmonary diseases or neuromuscular disease.
 - **during exercise:**
 - A significant drop suggests hyperventilation and/or V/Q mismatch.
 - A significant increase in $P_{ET}CO_2$ means alveolar hypoventilation, *eg*, severe COPD, obesity, hypoventilation syndrome, or neuromuscular disease.
 - $P_{ET}CO_2$ **decrease or non-increase** may mean low cardiac output, with a prognostic value similar to circulatory power and VE/VCO_2 slope.



Merci pour votre attention!