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# Immediate Impact of Inspiratory Muscle Training on Pulmonary Arterial Pressure, Cardiac Output, and Oxygen Cost of Breathing Derived from Pulmonary Arterial Saturation

**Christina Priegnitz***

[1]Petrus Hospital Wuppertal, Academic Teaching Hospital of the University of Duesseldorf, Department of Pneumology and Cardiology, Carnaper Str. 48, 42283 Wuppertal, Germany

**Corresponding author:**
Christina Priegnitz, Petrus Hospital Wuppertal, Academic Teaching Hospital of the University of Duesseldorf, Department of Pneumology and Cardiology, Carnaper Str. 48, 42283 Wuppertal, Germany. E-mail: christina_priegnitz@gmail.com

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## Abstract

This prospective study explores the effects of inspiratory muscle training (IMT) on pulmonary arterial hemodynamics and cardiac output (CO) in patients with suspected pulmonary hypertension. IMT, which elevates negative thoracic pressure, was performed during right heart catheterization. The study introduces a novel method for assessing the oxygen cost of breathing during IMT. Results from 22 patients indicate that IMT did not significantly alter mean pulmonary arterial pressure or CO. However, a reduction in mixed-venous hemoglobin oxygen saturation was observed. The study establishes the feasibility of measuring the oxygen cost of breathing using the described method.

**Keywords:** Cardiorespiratory Interaction; Chronic Obstructive Pulmonary Disease (COPD); Heart Failure; Oxygen Cost of Breathing; Ventilation

## Introduction

Cardiac output (CO) is influenced by respiration, with inspiratory maneuvers typically affecting stroke volumes in the right and left ventricles differently. Inspiratory muscle training (IMT) has gained attention, primarily in chronic obstructive pulmonary disease (COPD). Still, its effects on cardiac output remain underexplored, particularly in conditions such as chronic heart failure (CHF) [1]. This study aims to investigate the acute hemodynamic impact of IMT on CO, pulmonary arterial pressure (PAP), and the oxygen cost of breathing in patients with suspected pulmonary hypertension (PH).

In recent years, IMT has demonstrated benefits in COPD by enhancing inspiratory muscle strength, endurance, functional capacity, and quality of life. Similarly, in CHF, IMT has shown improvements in dyspnea and overall performance. The common IMT approach involves breathing through a threshold device, impeding inspiration. Given the potential effects on intra-thoracic pressure, we hypothesize that IMT could influence CO, an aspect crucial in both COPD and CHF, where cardiac comorbidities are prevalent.

PH, defined by a resting mean pulmonary artery pressure (mPAP) of ≥25 mmHg, is a common concern in COPD and CHF. Understanding the acute hemodynamic response to IMT is vital for delineating its implications in patients with suspected PH. The study also explores the oxygen cost of breathing associated with IMT, providing valuable insights into its overall impact [2-5].

Patients with COPD and CHF often experience PH, and the prevalence of elevated mPAP in these populations is substantial. Distinguishing pre-capillary and post-capillary PH is critical, as it informs the management approach. While PH is well-documented in left heart diseases, its incidence in specific cardiac conditions remains less elucidated. This study addresses the existing gap by investigating acute hemodynamic changes during IMT, regardless of PH presence.

Exploring the acute effects of IMT on CO, PAP, and oxygen cost of breathing in patients with suspected PH contributes valuable knowledge to the management of COPD and CHF, shedding light on the potential hemodynamic implications of this therapeutic approach.

# Materials and Methods

## Study Design

This prospective study, conducted from January 2009 to December 2013, adhered to the principles of the Declaration of Helsinki. Approval from the local Ethics Committee (University of Witten-Herdecke, Witten-Herdecke, Germany) was obtained, and written informed consent was provided by all participating patients [6-8].

Patients requiring right heart catheterization (RHC) for clinical indications, such as dyspnea, pulmonary hypertension, or right heart failure, were enrolled. All patients were clinically stable, and the study employed the Threshold Inspiratory Muscle Training (Threshold-IMT) device (Philips Respironics, Amsterdam, the Netherlands) for IMT. Respiratory therapists provided training on IMT usage, and supervised practice sessions were conducted before RHC.

Each IMT session consisted of 7 repetitions, with 2 minutes of IMT followed by 1 minute of rest. The Threshold-IMT featured a variable threshold, adjustable from 7 to 41 cmH2O, for inspiration through a mouthpiece. Inclusion criteria included the indication for RHC and age above 18 years, while exclusion criteria involved a lack of written consent or an inability to perform IMT.

## Measurements and Assessments

Lung function parameters were assessed using body plethysmography (Masterlab, Carefusion, San Diego, USA). The inspiratory muscle load was evaluated by calculating the mouth occlusion pressure after 0.1 s of inspiration (P0.1). Maximal inspiratory mouth pressure (Pimax) and respiratory capacity (P0.1/Pimax) were measured as per ATS/ERS recommendations.

RHC was performed in a supine position, with blood sampling from central venous, right atrium, and right ventricle sites. Pulmonary arterial wedge pressure and cardiac output were calculated via thermodilution.

Patients switched to a sitting position after the initial hemodynamic measurements and underwent IMT for at least 2 minutes. The specific pressure for Threshold-IMT was set at 20-30% of Pimax, with adjustments based on individual tolerance.

Hemodynamic parameters, including mean pulmonary artery pressure (mPAP), cardiac output, and stroke volume, were measured before and during IMT. Blood gas samples from the pulmonary artery were obtained during IMT. Analysis was conducted using SPSS software (version 20; IBM, Armonk, New York, USA), employing the Wilcoxon test for calculating hemodynamic changes.

## Statistical Analysis and Endpoints

The primary endpoint was the change in mPAP, and the null hypothesis assumed no significant increase. Sample size calculation considered a clinically relevant increase of 5 mmHg in mPAP, with a standard deviation of 9 mmHg. A sample size of 22 was determined to provide 80% power for the study [9].

Secondary endpoints included changes in cardiac output and stroke volume. Additional analysis involved measuring the oxygen cost of breathing in some patients. Subgroup analysis was performed for patients with only COPD or only chronic heart failure.

Results are presented with an interquartile range or as means ± standard deviations, and statistical significance was set at $\alpha$=0.05.

# Results

A total of 22 patients (10 men, 12 women) were included in the study. The mean ± standard deviation for Pimax was 6.2 ± 2.8 kPa (63.1 ± 29.0 cmH2O), Pimax % pred 59.5 ± 27.6%, P0.1 0.3 ± 0.2 kPa (3.3 ± 2.2 cmH2O), and the P0.1% 142.3 ± 98.3. Detailed anthropometric and lung function data.

The baseline pulmonary artery pressure (PAP) showed a median systolic/diastolic/mean (s/d/m) of 36/13/22 mmHg (25th-75th percentile 25-54/5-19/13-35) and a mean mPAP of 25 ± 14 mmHg. Pulmonary arterial wedge pressure (PAWP) was 10 (4-14) mmHg. Cardiac output was 5.2 l/min (4.4–6.5), cardiac index (CI) 2.7 l/min/m² (2.4-3.2), and stroke volume index (SVI) 39.5 [mL/min/m2] (28.6–59.7). Pulmonary vascular resistance (PVR) was calculated as 145 (107-349) [dynes cm-5] (Table 1).

**Table 1:** Hemodynamic baseline parameters. CVP=central venous pressure. RA=pressure right atrium. SPAO2=Hemoglobinsaturation of the pulmonary artery. Data are expressed as median with interquartile range (25th–75th percentile except SPAO2 (mean ± SD).

| n | 22 |
|---|---|
| CVP (mmHg) | 4 (1-8) |
| RA (mmHg) | 4 (1-8) |
| PA s/d/m (mmHg) | 36/13/22 (24.5-53.5/5.0 -18.5/12.8-34.5) |
| PAWP (mmHg) | 10 (4-14) |
| CO (litres/min) | 5.2 (4.4-6.5) |
| CI (litres/min/m²) | 2.7 (2.4-3.2 |
| Heart rate (/min) | 78 (70-99) |
| SVI (mL/min/m²) | 39.5 (28.6-59.7) |
| PVR (dynes·s·cm-5) | 145 (107-349) |
| SVR (dynes·s·cm-5) | 1352 (904-1671) |
| $S_{PA}O_2$ (%) | 71.8 ± 2.3 |

During inspiratory muscle training (IMT), the pressure was set to 2.3 ± 0.6 kPa (23.7 ± 5.7 cmH2O), and the duration of IMT was 2.2 ± 0.3 min. At the end of IMT, the s/d/m PAP was 41/9/19 mmHg (26-59/-1-22/9-37). While there was a trend towards lower diastolic pressure, the change was not statistically significant (p=0.06) (Figure 1).

At the end of IMT, cardiac output (6.1; 5.5-6.4 l/min) and cardiac index (3.0; 2.9-3.3 l/min/m²) were measured in 8 patients, showing no significant changes compared to baseline (CO p=0.20 and CI p=0.18) (Figure 2).

The mixed-venous hemoglobin oxygen saturation before IMT was 71.8 ± 2.3%, and it significantly reduced to 65.8 ± 5.9% at the end of IMT (p=0.027) (Figure 3).

Subgroup analysis of patients with only COPD or heart failure did not reveal any other significant findings [10-12].

## Discussion

In our study, we investigated the acute effects of inspiratory muscle training (IMT) on pulmonary artery (PA) pressure, cardiac output (CO), and the oxygen cost of breathing in patients with

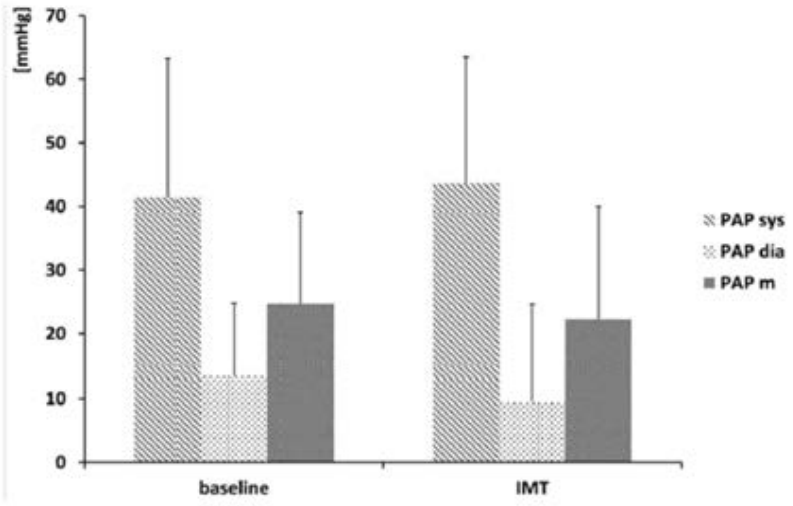


**Figure 1:** HPLC chromatogram of the nine reference compounds in 50% aqueous methanol, measured at 370nm. Retention times for rutin, sutherlandin A, sutherlandin B, kaempferol-3-O-rutinoside, sutherlandin C, sutherlandin D, quercitrin, quercetin and kaempferol were 11.9, 12.7, 13.8, 15.3, 16.2, 17.0, 18.0, 26.2 and 28.1 minutes, respectively.

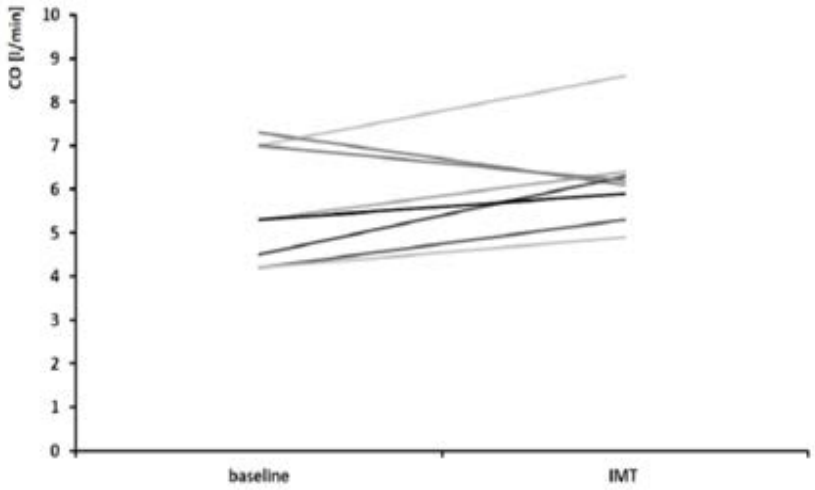


**Figure 2:** HPLC chromatogram of the nine reference compounds in 50% aqueous methanol, measured at 370nm. Retention times for rutin, sutherlandin A, sutherlandin B, kaempferol-3-O-rutinoside, sutherlandin C, sutherlandin D, quercitrin, quercetin and kaempferol were 11.9, 12.7, 13.8, 15.3, 16.2, 17.0, 18.0, 26.2 and 28.1 minutes, respectively.

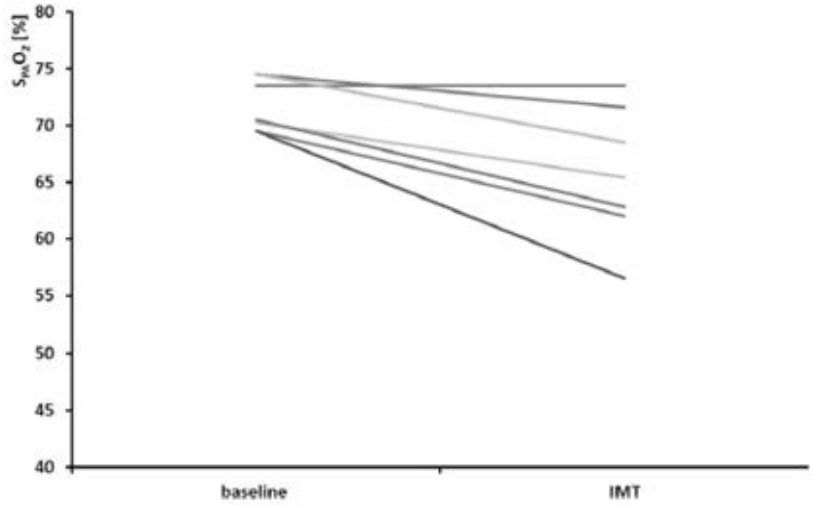


**Figure 3:** HPLC chromatogram of the nine reference compounds in 50% aqueous methanol, measured at 370nm. Retention times for rutin, sutherlandin A, sutherlandin B, kaempferol-3-O-rutinoside, sutherlandin C, sutherlandin D, quercitrin, quercetin and kaempferol were 11.9, 12.7, 13.8, 15.3, 16.2, 17.0, 18.0, 26.2 and 28.1 minutes, respectively.

suspected pulmonary hypertension (PH). Despite the moderate intensity of IMT (23.7 cmH2O), we did not observe significant changes in systolic and mean PA pressure. The study population had a median mean PA pressure below the threshold for pulmonary arterial hypertension (PAH), excluding this condition [13-18].

During spontaneous breathing, the fall in pleural pressure during inspiration typically affects right atrial pressure. IMT, by increasing negative thoracic pressure, could potentially exacerbate this effect. However, our study did not show a significant change in CO, challenging the expected impact on right ventricular preload and pulmonary venous pressure. Notably, there was a trend towards a lower diastolic PA pressure, suggesting a potential reduction in pulmonary arterial hypertension, albeit not reaching statistical significance, possibly due to sample size or IMT intensity.

The comparison with maneuvers like coughing or the Valsalva maneuver, which increase intra-thoracic pressure and pulmonary arterial pressure, is intriguing. IMT, involving inspiration against a threshold, theoretically reduces intra-thoracic pressure, potentially decreasing pulmonary arterial hypertension. While our study hinted at a lower diastolic PA pressure, the lack of statistical significance could be attributed to the sample size or the relatively moderate IMT intensity.

Measuring CO during IMT in a subset of patients revealed no significant change, contrary to expectations of increased venous return and pulmonary venous pressure. This contrasts with the use of inspiratory threshold valves in resuscitation, where increased cardiac output and blood pressure can be achieved. The divergent effects could be due to the unique context of IMT and its specific impact on intra-thoracic pressure dynamics.

Another consideration involves the potential increase in left ventricular afterload during IMT, stemming from the heightened negative pressure during inspiration. This could lead to a reduction in cardiac output, contributing to increased oxygen cost of breathing and respiratory muscle deoxygenation. Upper airway obstruction, as seen in tracheal tumors or tonsillar hypertrophy, may induce pulmonary edema due to negative intra-thoracic pressure changes [19-23].

Our study introduces a novel method for measuring the oxygen consumption of inspiratory muscles during IMT, utilizing blood sampling during right heart catheterization. This method, though applied to a minority of patients due to premature IMT cessation, revealed a surprising 6% reduction in pulmonary arterial saturation. Further research is needed to explore the potential of this approach as a monitoring tool for respiratory muscles, especially in intensive care settings and spontaneous breathing trials.

In summary, the oxygen cost of breathing is markedly increased in conditions like chronic heart failure (CHF) and chronic obstructive pulmonary disease (COPD). Our study, while not demonstrating significant hemodynamic changes during IMT, sheds light on potential physiological intricacies. Further investigations should delve into the broader implications of IMT, considering its impact on respiratory muscle function and oxygen cost, with potential applications in optimizing patient care and exercise training programs.

## Limitations

**Single Adjustment in IMT:** The IMT in our study was conducted with a single adjustment, preventing an assessment of the

influence of deep inspiration on the oxygen cost of breathing. A more comprehensive investigation involving variations in IMT parameters could provide a more nuanced understanding of its effects.

**Incomplete Assessment in All Patients:** Due to methodological constraints, not all patients underwent a complete assessment of cardiac output (CO) and oxygen cost of breathing. This limitation reduces the generalizability of our findings and underscores the need for comprehensive data in future studies.

**High Standard Deviation:** The standard deviation of our results was relatively high, introducing variability that necessitates caution in concluding. The wide range of responses among participants may be indicative of individual variations or other influencing factors not accounted for in our study.

**Moderate PH in Study Population:** Our study focused on patients with moderate pulmonary hypertension (PH), and the applicability of our findings to individuals with normal pulmonary artery (PA) pressure remains uncertain. Replicating the study in a broader population could provide insights into the generalizability of results across varying PH severities.

## Conclusion

In conclusion, our investigation into the acute effects of inspiratory muscle training (IMT) in patients with moderate pulmonary hypertension revealed no significant impact on pulmonary artery (PA) pressure or cardiac output. However, a moderate IMT protocol led to a notable 6% increase in the oxygen cost of breathing. These findings suggest that while IMT may not alter hemodynamics in moderate PH, its influence on respiratory muscle oxygen consumption merits further consideration. Future research, addressing the limitations highlighted, is essential to deepen our understanding of IMT implications and its potential benefits in diverse patient populations.

## References

1. Decramer M (1985) Response of the respiratory muscles to rehabilitation in COPD. J ApplPhysiol 107:971-976.
2. Held HE, Pendergast DR (2014) The effects of respiratory muscle training on respiratory mechanics and energy cost. RespirPhysiolNeurobiol200:7-17.
3. Meyer FJ, Borst MM, Zugck C, Kirschke A, Schellberg D, et al. (2001) Respiratory muscle dysfunction in congestive heart failure: clinical correlation and prognostic significance. Circulation 103:2153-2158.
4. Gosselink R, De Vos J, van den Heuvel SP, Segers J, Decramer M, et al. (2011) Impact of inspiratory muscle training in patients with COPD: what is the evidence? EurRespir J 37:416-425.
5. Adamopoulos S, Schmid JP, Dendale P, Poerschke D, Hansen D, et al. (2014) Combined aerobic/inspiratory muscle training vs. aerobic training in patients with chronic heart failure: The Vent-HeFT trial: a European prospective multicentre randomized trial. Eur J Heart Fail.
6. Sutbeyaz ST, Koseoglu F, Inan L, Coskun O (2010) Respiratory muscle training improves cardiopulmonary function and exercise tolerance in subjects with subacute stroke: a randomized controlled trial. ClinRehabil 24:240-250.
7. Darnley GM, Gray AC, McClure SJ, Neary P, Petrie M, et al. (1999) Effects of resistive breathing on exercise capacity and diaphragm function in patients with ischaemic heart disease. Eur J Heart Fail 1:297-300.
8. Lurie K, Voelckel W, Plaisance P, Zielinski T, McKnite S, et al. (2000) Use of an inspiratory impedance threshold valve during cardiopulmonary resuscitation: a progress report. Resuscitation 44:219-230.
9. Freixa X, Portillo K, Pare C, Garcia-Aymerich J, Gomez FP, et al. (2013) Echocardiographic abnormalities in patients with COPD at their first hospital admission. EurRespir J 41:784-791.
10. Hoeper MM, Bogaard HJ, Condliffe R, Frantz R, Khanna D, et al. (2013) Definitions and diagnosis of pulmonary hypertension. J Am CollCardiol 62:D42-50.
11. Galie N, Humbert M, Vachiery JL, Gibbs S, Lang I, et al. (2016) ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension: The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS): Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT). Eur Heart J 37:67-119.
12. Cuttica MJ, Kalhan R, Shlobin OA, Ahmad S, Gladwin M, et al. (2010) Categorization and impact of pulmonary hypertension in patients with advanced COPD. Respir Med. 104:1877-1882.
13. Thabut G, Dauriat G, Stern JB, Logeart D, Levy A, et al. (2005) Pulmonary hemodynamics in advanced COPD candidates for lung volume reduction surgery or lung transplantation. Chest 127:1531-1536.
14. Scharf SM, Iqbal M, Keller C, Criner G, Lee S, et al. (2002) Hemodynamic characterization of patients with severe emphysema. Am J RespirCrit Care Med166:314-322.
15. Vachiery JL, Adir Y, Barbera JA, Champion H, Coghlan JG, et al. (2013) Pulmonary hypertension due to left heart diseases. J Am CollCardiol62:D100-8.
16. Aragam JR, Folland ED, Lapsley D, Sharma S, Khuri SF, et al. (1992) Cause and impact of pulmonary hypertension in isolated aortic stenosis on operative mortality for aortic valve replacement in men. Am J Cardiol 69:1365-1367.
17. Lam CS, Roger VL, Rodeheffer RJ, Borlaug BA, Enders FT, et al. (2009) Pulmonary hypertension in heart failure with preserved ejection fraction: a community-based study. J Am CollCardiol 53:1119-1126.
18. Moraes DL, Colucci WS, Givertz MM (2000) Secondary pulmonary hypertension in chronic heart failure: the role of the endothelium in pathophysiology and management. Circulation 102:1718-1723.
19. Delgado JF, Conde E, Sanchez V, Lopez-Rios F, Gomez-Sanchez MA, et al. (2005) Pulmonary vascular remodeling in pulmonary hypertension due to chronic heart failure. Eur J Heart Fail 7:1011-1016.

20. Meyer FJ, Lossnitzer D, Kristen AV, Schoene AM, Kubler W, et al. (2005) Respiratory muscle dysfunction in idiopathic pulmonary arterial hypertension. EurRespir J 25:125-130.

21. Kabitz HJ, Schwoerer A, Bremer HC, Sonntag F, Walterspacher S, et al. (2008) Impairment of respiratory muscle function in pulmonary hypertension. ClinSci (Lond)114:165-171.

22. Wanger J, Clausen JL, Coates A, Pedersen OF, Brusasco V, et al. (2005) Standardisation of the measurement of lung volumes. EurRespir J 26:511-522.

23. American Thoracic Society/European Respiratory S (2002) ATS/ERS Statement on respiratory muscle testing. Am J RespirCrit Care Med 166:518-624.