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## CASE REPORT

# Exercise-induced cardiac constraint by the lungs

Roberto Cassandro <sup>a</sup>, Sergio Harari <sup>a,\*</sup>, Achille Bianchi <sup>a</sup>,  
Piergiuseppe Agostoni <sup>b</sup>

<sup>a</sup> *Unità Operativa di Pneumologia, Ospedale San Giuseppe, Milano Cuore, via San Vittore 12, 20123 Milano, Italy*

<sup>b</sup> *Centro Cardiologico Monzino, IRCCS, Istituto di Cardiologia Università di Milano, Italy*

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## Summary

A patient of forty-five years old male, with severe emphysema, underwent bullectomy. He was studied, before and one year after surgery, by standard pulmonary function test and cardiopulmonary exercise test. We found that before bullectomy, tidal volume increases up to 45 W and flattens thereafter. After bullectomy tidal volume increases through the entire exercise.

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## Introduction

Cardiopulmonary interactions during exercise have been extensively studied.<sup>1–3</sup> The presence of a pulmonary constrain on the heart has been suggested in several disease such as COPD and heart failure.<sup>2,4–6</sup> However, the possibility to show a therapeutic intervention able to increase the cardiac function by acting on the pulmonary constrain forces has been suggested but never proved.<sup>3</sup> We report a case of a subject with severe emphysema who, after surgical bullectomy, improved his exercise capacity through a reduction of the cardiac constraint by the lungs.

## Clinical report

Forty-five years old male, with severe emphysema, who underwent bullectomy, was studied, before and one year after surgery, by standard pulmonary function test and cardiopulmonary exercise test. The latter was performed on a cyclo-ergometer, with a ramp protocol (15 W/min). After bullectomy pulmonary function and exercise capacity improved (Table 1). The  $\text{VO}_2$ /work relationship is of specific interest. Before bullectomy (Fig. 1, panel A), above 45 W, the  $\text{VO}_2$  increase flattens. After bullectomy (Fig. 1, panel B) the  $\text{VO}_2$ /work relationship is a constant straight line even when a higher workload is achieved. Also ventilation kinetics are of some interest. Ventilation and tidal volume are higher after bullectomy (Table 1). Before bullectomy, tidal volume increase up to 45 Watts and flattens thereafter (Fig. 2). After bullectomy tidal volume increases through the entire exercise.

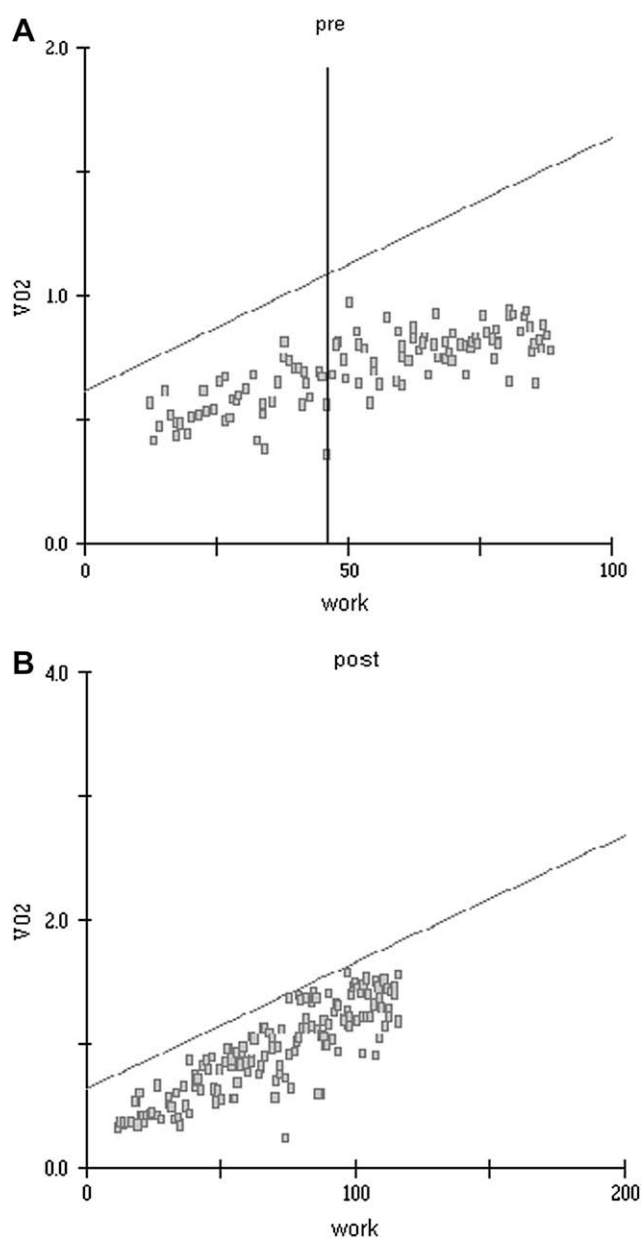
\* Corresponding author. Tel.: +39 02 85994580; fax: +39 02 85994400.

E-mail address: [sharari@ilpolmone.it](mailto:sharari@ilpolmone.it) (S. Harari).

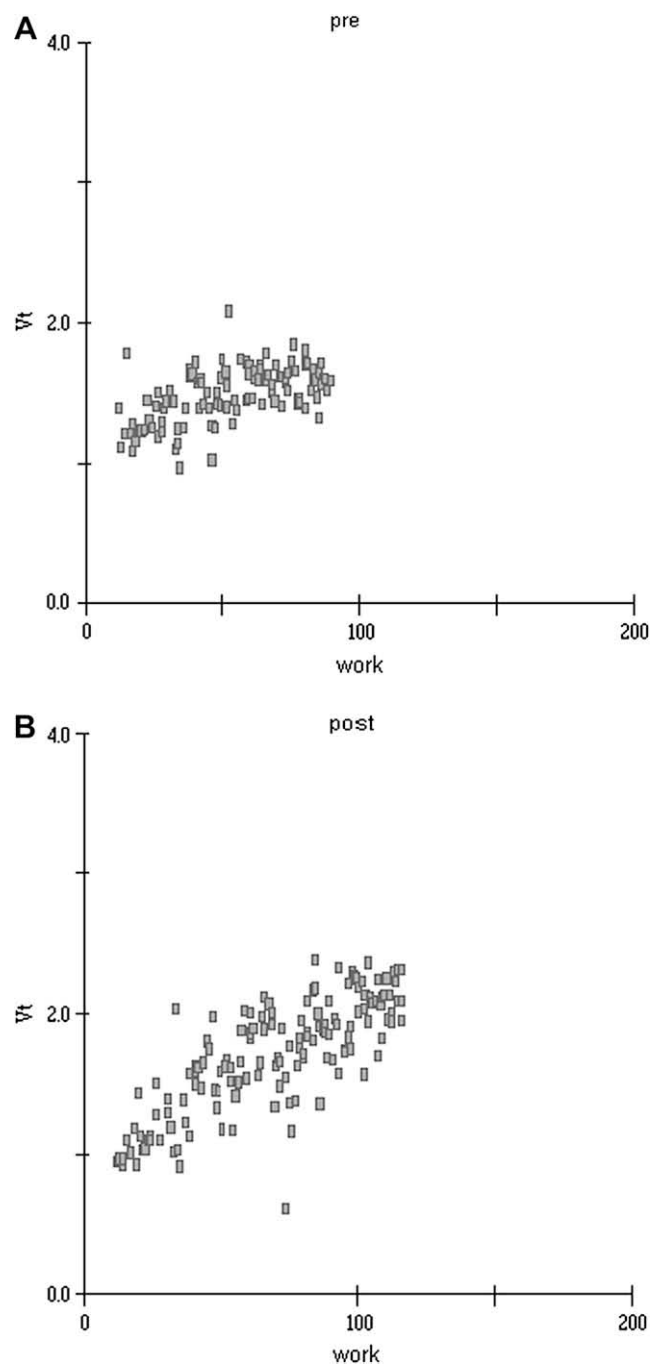
**Table 1** Pulmonary function and cardiopulmonary exercise results before (pre) and 1 year after bullectomy (post).

	Pre	Post
FVC [L (%)]	3.99 (84)	4.81 (103)
FEV <sub>1</sub> [L (%)]	1.89 (49)	2.74 (72)
TLC [L (%)]	7.57 (106)	7.39 (105)
RV [L (%)]	3.58 (171)	2.59 (123)
pVO <sub>2</sub> [L/min]	0.844	1.374
pVE [L/min]	40.5	59.1
pVT [L]	1.593	2.131

FVC = forced vital capacity, FEV<sub>1</sub> = forced expiratory volume in 1 second, TLC = total lung capacity, RV = residual volume, pVO<sub>2</sub> = oxygen consumption at peak exercise, pVE = ventilation at peak exercise, pVT = tidal volume at peak exercise.



**Figure 1** VO<sub>2</sub>/work relationship before bullectomy (panel A) and after bullectomy (panel B). The dotted vertical line indicates when lung constraint start to be evident.



**Figure 2** VT/work relationship before bullectomy (panel A) and after bullectomy (panel B). The dotted vertical line indicates when lung constraint start to be evident.

## Discussion

The VO<sub>2</sub>/work relationship is an index of efficiency of the O<sub>2</sub> delivery toward the working muscles. The flattening of the relationship means a reduction of cardiac output usually due to exercise induced cardiac ischemia.<sup>7,8</sup> In the present case the flattening of the VO<sub>2</sub>/work relationship observed before bullectomy is due to exercise induced increase in lung stiffness which interfere with either right and left ventricle preload as well as the external work of the heart. The latter is the work performed by the heart to

“push and pull” against the lungs during systole and diastole.<sup>1</sup> In the presurgical exercise tidal volume increase flattens when  $VO_2$ /work relationship flattens suggesting that the lungs are unable to further expand and act as a cardiac constrictor. Furthermore, the disappearance of the flattening of both  $VO_2$ /work and tidal volume/work relationships after surgery, even at higher work rate, is another evidence that in this case the lung constrains the heart. The present is the first documentation of lung constraint forces on the heart which reduces cardiac performance in the absence of a primitive cardiac disease.<sup>3</sup>

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