Exercise-induced cardiac constraint by the lungs

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Introduction

Cardiopulmonary interactions during exercise have been extensively studied. The presence of a pulmonary constrain on the heart has been suggested in several disease such as COPD and heart failure. 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. 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 VO₂/work relationship is of specific interest. Before bullectomy (Fig. 1, panel A), above 45 W, the VO₂ increase flattens. After bullectomy (Fig. 1, panel B) the VO₂/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 trough the entire exercise.
The VO2/work relationship is an index of efficiency of the O2 delivery toward the working muscles. The flattening of the relationship means a reduction of cardiac output usually due to exercise induced cardiac ischemia. In the present case the flattening of the VO2/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

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

<table>
<thead>
<tr>
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<th>Pre</th>
<th>Post</th>
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<tbody>
<tr>
<td>FVC [L (%)]</td>
<td>3.99 (84)</td>
<td>4.81 (103)</td>
</tr>
<tr>
<td>FEV1 [L (%)]</td>
<td>1.89 (49)</td>
<td>2.74 (72)</td>
</tr>
<tr>
<td>TLC [L (%)]</td>
<td>7.57 (106)</td>
<td>7.39 (105)</td>
</tr>
<tr>
<td>RV [L (%)]</td>
<td>3.58 (171)</td>
<td>2.59 (123)</td>
</tr>
<tr>
<td>pVO2 [L/min]</td>
<td>0.844</td>
<td>1.374</td>
</tr>
<tr>
<td>pVE [L/min]</td>
<td>40.5</td>
<td>59.1</td>
</tr>
<tr>
<td>pVT [L]</td>
<td>1.593</td>
<td>2.131</td>
</tr>
</tbody>
</table>

FVC = forced vital capacity, FEV1 = forced expiratory volume in 1 second, TLC = total lung capacity, RV = residual volume, pVO2 = oxygen consumption at peak exercise, pVE = ventilation at peak exercise, pVT = tidal volume at peak exercise.

Figure 1  VO2/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 VO2/work relationship is an index of efficiency of the O2 delivery toward the working muscles. The flattening of the relationship means a reduction of cardiac output usually due to exercise induced cardiac ischemia. In the present case the flattening of the VO2/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. In the presurgical exercise tidal volume increase flattens when VO₂/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 VO₂/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.

References