Mechanism of pulmonary function changes after surgical correction for funnel chest

L. Derveaux*, I. Ivanoff**, F. Rochette*, M. Demedts*  

Mechanism of pulmonary function changes after surgical correction for funnel chest: L. Derveaux, I. Ivanoff, F. Rochette, M. Demedts.  

ABSTRACT: In 24 subjects with pectus excavatum we evaluated whether the previously detected unfavourable effects of corrective surgery on the ventilatory capacity were attributable to pulmonary or to chest wall factors. We found that 12.2±3.7 yrs postoperatively (i.e. at the age of 23.3±5.4 yrs) the vital capacity was decreased from 89±10% predicted (pred) preoperatively to 64±6% pred (p<0.001) and forced expiratory volume in one second from 88±17 to 66±11% pred (p<0.001). At total lung capacity (TLC; 69±5% pred) we found an obvious reduction in transpulmonary pressure (59±23% pred) and in transdiaphragmatic pressure (30±17 cm H₂O) postoperatively. This indicated an extrapulmonary cause of the restrictive defect, attributable to abnormal chest wall mechanics secondary to the extensive surgery on the sternum and parasternal zones.

Surgical correction of funnel chest has been performed mainly for cosmetic and psychological reasons. However, cardiopulmonary repercussions of mechanical compression by severe chest deformities have also been arguments for the operation [1].

It has been shown that this corrective surgery generally has a favourable subjective functional effect and improves exercise tolerance, mainly due to psychological factors but also to favourable cardiac and haemodynamic effects [2, 3]. However, despite a correction of radiological indices (especially an increase in anteroposterior chest diameter) lung function has often been shown to decrease further [4].

The purpose of the present study was to investigate by more elaborate pulmonary function tests whether this postoperative restriction was attributable to pulmonary or to chest wall abnormalities.

Patients and methods

Twenty-four patients (18 males, 6 females) underwent an extensive lung function examination at the age of 23.3±5.4 yrs (mean±sd) i.e. 12.2±3.7 yrs after surgical correction for a funnel chest (associated in two cases with an S-shaped sternum and in two others with a pectus carinatum). Seventeen of the patients also underwent routine lung function tests prior to surgery. The operation, using a variant of the Barowski technique [5], was performed at the age of 11.9±5.5 yrs. The technique consists essentially of a subperichondrial resection of cartilages 2–8 followed by a sternophrenolysis and wedge osteotomy of the sternum which is subsequently fixed in hypercorrection by nylon fibres. The sternal attachments of the intercostal muscles are detached, but not transected.

The methods of routine lung function examinations and the reference values are outlined elsewhere [6] except for the reference values for children for which those of ZAPLETAL et al. [7] were used. Briefly, spirometry consisted of measurements of vital capacity (VC) and of forced expiratory volume in one second (FEV₁). Residual volume (RV), total lung capacity (TLC) and functional residual capacity (FRC) were obtained with the multibreath helium equilibration technique. Diffusing capacity for carbon monoxide (DLCO) was measured with the single breath method and the Krogh factor (DLCO/VA) was calculated.

Airway resistance (Raw) was obtained from at least three measurements with a constant volume body plethysmograph and specific conductance (sGaw=1/Raw×FRC) was calculated. Three quasi-static lung distensibility curves were obtained using the oesophageal balloon technique; mean static expiratory compliance (Cst) was measured at FRC and specific compliance (sCst) was calculated as Cst/TLC [8]; the largest transpulmonary pressure at TLC (PtpTLC) was retained and related to predicted values [9]. The balloon was passed into the stomach and gastric pressure was measured at TLC (PgstTLC) and the largest value of three trials was retained. Transdiaphragmatic pressure at TLC (PdpTLC) was calculated as the largest difference between PgstTLC and PtpTLC minus their difference at FRC [9, 10]. Maximal...
static inspiratory and expiratory mouth pressures (PimaxFRc, PimaxFRc) were measured at FRC with a pressure gauge [11].

Results

Table 1 shows the results of routine lung function tests before and 12.2±3.7 yrs following corrective surgery for funnel chest in 17 patients. The preoperative values of VC and FEV1 were at the lower limit of normality, and the postoperative values showed a further decrease of about 20–25% (p<0.01) in percentage predicted values, although the absolute values (in litres) were increased due to the increase in body size during adolescence.

Table 2 presents the results of more elaborate lung function tests following surgery in the 24 patients (also including the 17 patients of the previous table). VC and FEV1 were almost equal in both tables which confirms the uniformity of the whole group. The decreased TLC with normal FEV1/VC and Raw indicates a moderate, purely restrictive defect. FRC is normal and FRC/TLC is high, reflecting that the restriction is due mainly to a reduction in inspiratory reserve capacity. DLco is reduced less than TLC, and the DLco/VA is high. The static lung compliance (Cstsr) is decreased more than TLC, but the transpulmonary pressure at TLC (PtpTLC) is also markedly reduced. Pga,LC is within normal ranges but Pdi,LC is also clearly reduced in comparison with our predicted values [9]. PimaxFRc is within the normal range and PmaxFRc is at the lower limit of normality [11].

Discussion

Most patients in our series were operated on mainly for cosmetic and psychological reasons since there were only minor functional limitations preoperatively.

The present study confirms the findings of an earlier unpublished study in this department, which showed that the ventilatory restriction (in percentage predicted terms) worsens after corrective surgery despite the improvement of the radiological indices of the pectus deformity. In the earlier study, a stepwise discriminant analysis demonstrated, furthermore, that the postoperative decline in pulmonary function was worse when the preoperative lung restriction was more pronounced (especially if the VC was less than 75% pred), and that this was not related to other factors such as age, or radiological chest deformity indices. It was also not related to the time elapsed after operation and, therefore, could be attributed to the operation itself and not to a progressive change afterwards.

Table 2. - Results of extensive postoperative lung function tests in 24 patients

<table>
<thead>
<tr>
<th>n</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>VC (l)</th>
<th>FEV1 (l)</th>
<th>FEV1/VC (%)</th>
<th>DLco (ml/min/mmHg)</th>
<th>DLco/VA (% pred)</th>
<th>Raw (cmH2O·l·s−1)</th>
<th>Cst (%)</th>
<th>PtpTLC (cmH2O)</th>
<th>Pdi,LC (cmH2O)</th>
<th>Pga,LC (cmH2O)</th>
<th>TLC (% predicted)</th>
<th>PmaxFRc (cmH2O)</th>
<th>PmaxFRc (cmH2O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>23.3±5.4</td>
<td>177±10</td>
<td>63±12</td>
<td>3.37±0.77</td>
<td>2.79±0.56</td>
<td>65±10</td>
<td>28±16.3</td>
<td>112±15</td>
<td>2.08±0.74</td>
<td>162±45</td>
<td>58±19</td>
<td>83±20</td>
<td>18±13</td>
<td>109±51</td>
<td>128±51</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>24.0±9</td>
<td>22.6±8.6</td>
<td>28.1±6.3</td>
<td>95±20</td>
<td>81±18</td>
<td>56±10</td>
<td>24.6±8.6</td>
<td>28.1±6.3</td>
<td>22.6±8.6</td>
<td>24.6±8.6</td>
<td>28.1±6.3</td>
<td>22.6±8.6</td>
<td>22.6±8.6</td>
<td>22.6±8.6</td>
<td>22.6±8.6</td>
<td>22.6±8.6</td>
</tr>
</tbody>
</table>

VC: vital capacity; FEV1: forced expiratory volume in one second; TLC: residual volume and total lung capacity measured by multibreath helium equilibration; RV: residual volume; TLC: total lung capacity measured by constant volume body plethysmograph; DLco: carbon monoxide diffusing capacity; VA: alveolar volume; Raw: airway resistance; sGaw: specific conductance; Cst: mean static expiratory compliance; sCst: specific compliance; PtpTLC: transpulmonary, gastric and transdiaphragmatic pressure, respectively, measured at TLC; PmaxFRc: maximal static inspiratory and expiratory mouth pressures, respectively, measured at functional residual capacity.
The present data provide an explanation for the adverse effects of the operation on lung function. The restrictive defect (i.e., decrease in TLC) is, indeed, characterized by markedly reduced transpulmonary and transdiaphragmatic pressures at TLC, by a high DLCO/VA and by a high FRC/TLC ratio despite a normal FRC (i.e., 47±7% predicted TLC), a normal FEV1/VC and normal Raw. These functional abnormalities point to an extrapulmonary origin of the restriction and are somewhat similar to those described by us in chest wall defects due to neuromuscular diseases [6] and to systemic lupus erythematosus with muscular involvement [9]. The increase in DLCO/VA is typical of lung restriction due to extrapulmonary factors [12]. The secondary reduction in static lung compliance in these instances is quite classical, and has even been shown to be reversible after full inflation of the chest, at least in some subjects [9], but not in others [13]. In the present study the effect of fully inflating the lung (i.e., Ptpmax of at least 40 cmH2O) on compliance was not studied. The low transpulmonary and transdiaphragmatic pressures at TLC (despite low static lung compliance) confirm a chest wall factor as the cause of the restrictive defect. This postoperative restriction in inspiratory capacity is apparently mainly attributable to the extensive surgery on the sternum and para-sternal zones. This comprised subperichondral resection of the cartilages of several ribs, sternophrenolysis and sectioning of para-sternal intercostal muscles, which have been found to be important inspiratory muscles especially near TLC [14, 15].

It is also not surprising that in contrast with the limitation in chest expansion at high volumes, the respiratory muscle force at FRC, measured by Pmaxsc and Pmaxprc, is relatively well preserved. Indeed the latter is mainly determined by the diaphragm and abdominal muscles which were affected neither by the pre-existing configuration abnormality nor by the surgical procedure.

In conclusion, we confirm that surgical correction for funnel chest generally induces a restriction of lung function, and that this is due to extrapulmonary factors which affect chest wall mechanics especially at high lung volumes.

References

RéSUMÉ: Chez 24 sujets atteints de thorax en entonnoir, nous avons évalué si les effets défavorables de la chirurgie correctrice sur la capacité ventilatoire, qui avaient été observés anciennement, doivent être attribués à des facteurs pulmonaires ou parietaux. Nous avons trouvé que 12,2±3,7 années après l'intervention, la capacité vitale a diminué de 89±10% des valeurs prédites en pré-opératoire, à 64±6% des valeurs prédites (p<0,001), et que le VEMS avait baissé de 88±17 à 66±11% des valeurs prédites (p<0,001). A la capacité pulmonaire totale (TLC: 69±5% des valeurs prédites), nous avons trouvé en post-opératoire une diminution de la pression transpulmonaire (59±23% des valeurs prédites) et de la pression transdiaphragmatique (30±17 cmH2O). Ceci indique une cause extra-pulmonaire pour les anomalies restrictives, cause attribuable à une mécanique anormale de la paroi thoracique consécutive à la chirurgie extensive concernant le sternum et les zones para-sternales.