Cardiac and respiratory function before and after spinal fusion in adolescent idiopathic scoliosis

J M SHNEERSON AND M A EDGAR

From the Respiratory Physiology Laboratories, Department of Medicine, Cardiothoracic Institute, Brompton Hospital, and the Royal National Orthopaedic Hospital, London, UK

ABSTRACT Ten girls with adolescent idiopathic scoliosis were studied before and 17–23 months after spinal fusion. None had any cardiac or respiratory disease complicating the scoliosis. They underwent a range of resting lung function tests and a progressive exercise test. The mean angle of scoliosis decreased from 65·8 to 27·3 degrees after operation but the only significant physiological benefit detected in this study was a decrease in the submaximal minute ventilation. The physiological benefit of spinal fusion was therefore much less prominent than the anatomical improvement of the spinal curvature.

Although the obvious effect of thoracic scoliosis is an anatomical deformity, it also causes various cardiac and respiratory physiological abnormalities. These include diminished lung volumes (Larmi et al, 1955), pulmonary hypertension (Bergofsky et al, 1959; Shneerson, 1978a), and diminished maximal oxygen uptake and ventilation during exercise (Shneerson, 1978b). Over half the deaths in a series of 762 scoliotics were from cardiac or respiratory complications (Shneerson et al, 1978).

Spinal fusion is an effective method of correcting the anatomical deformity, but there is little information on its cardiorespiratory consequences. If there were any improvement or prevention of deterioration the indications for operation might be widened. In this study 10 patients underwent a range of resting lung function tests and a progressive exercise test before and after spinal fusion. The results have been compared to assess the effect of operation.

Subjects and methods

Ten girls with adolescent idiopathic scoliosis affecting the thoracic spine were studied before and 17–23 months (mean=19-6, SD=1-9) after spinal fusion with insertion of a Harrington rod. Their ages ranged from 13 to 15 years (mean=13-8, SD=0-9) when first tested. None had any cardiac or respiratory disease complicating the scoliosis. The angle of scoliosis was measured before and after operation by the method of Cobb (1948). The weight, height, and arm span were also measured (table 1).

Peak flow rate (PEFR) was measured with a Wright peak flow meter, the forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) with a dry spirometer (Vitalograph), and maximum voluntary ventilation (MVV) with a low resistance nine-litre wet spirometer (P K Morgan).

Exercise was performed on a bicycle ergometer against progressively increasing loads, and minute ventilation, tidal volume, respiratory frequency, oxygen uptake, and heart rate were recorded. The details of the methods are described in an earlier paper (Shneerson, 1978b).

The regression coefficients of the minute ventilation (VE) and heart rate (HR) on oxygen uptake (VO2) were calculated over the linear part of the relationships by the least squares method. The VE and HR responses were expressed as maximal values (VE max; HR max) and at interpolated values of VO2 of 0·75 l, 1·0 l, and 1·5 l (VE 0·75, VE 1·0, VE 1·5; HR 0·75, HR 1·0, HR 1·5) (Cotes, 1969; Spiro et al, 1974).

The observed results have been corrected to allow for the growth of the subjects between the two tests (table 1). The PEFR, FEV1, and FVC have been expressed as percentage predicted values according to arm span (Godfrey et al, 1970), VO2 max has been corrected for body weight.
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Table 1  Personal data of ten patients

<table>
<thead>
<tr>
<th></th>
<th>Angle of scoliosis (degrees)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Arm span (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before spinal fusion</td>
<td>Range 50-84</td>
<td>38-61</td>
<td>147-170</td>
<td>155-177</td>
</tr>
<tr>
<td></td>
<td>Mean 65-8</td>
<td>49-1</td>
<td>158-7</td>
<td>168-4</td>
</tr>
<tr>
<td></td>
<td>SD 11-3</td>
<td>6-4</td>
<td>6-7</td>
<td>6-1</td>
</tr>
<tr>
<td>After spinal fusion</td>
<td>Range 13-36</td>
<td>44-67-5</td>
<td>155-173</td>
<td>163-178</td>
</tr>
<tr>
<td></td>
<td>Mean 27-3</td>
<td>55-2</td>
<td>164-2</td>
<td>172-7</td>
</tr>
<tr>
<td></td>
<td>SD 8-0</td>
<td>7-6</td>
<td>5-5</td>
<td>4-6</td>
</tr>
</tbody>
</table>

(Davies et al, 1972), VT max for vital capacity, and in the mean dyspnoeic index (VE max/MVV × 100%) (before fusion: mean=65·4%, SD=14·4; after fusion: mean=63·2%, SD=19·7). The submaximal minute ventilation was significantly improved at V02 of 0·75 l (p<0·05) and 1·0 l (p<0·01) by surgery (figure).

Results

PEAK FLOW RATE, SPIROMETRY, AND MAXIMUM VOLUNTARY VENTILATION

The results before and after spinal fusion are shown in table 2. The absolute values for PEFR, FEV1, and FVC increased by 9-1–15·9% after surgery. When the values, however, were corrected for the increase in arm span between the two tests (Godfrey et al, 1970), the differences were not statistically significant. The MVV also increased slightly but not significantly after spinal fusion.

MAXIMUM OXYGEN UPTAKE

The small increase in V02 max after surgery (table 3) disappeared when it was corrected for body weight (Davies et al, 1972).

MINUTE VENTILATION

VE max hardly altered during the interval between the two tests (table 3) and there was little change in the mean dyspnoeic index (VE max/MVV × 100%) (before fusion: mean=65·4%, SD=14·4; after fusion: mean=63·2%, SD=19·7). The submaximal minute ventilation was significantly improved at V02 of 0·75 l (p<0·05) and 1·0 l (p<0·01) by surgery (figure).

Table 2  Results of resting lung function tests

<table>
<thead>
<tr>
<th></th>
<th>PEFR (l/min)</th>
<th>FEV1 (l)</th>
<th>FVC (l)</th>
<th>MVV (l/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Observed % predicted</td>
<td>Observed % predicted</td>
<td>Observed % predicted</td>
<td>Observed % predicted</td>
</tr>
<tr>
<td>Before spinal fusion</td>
<td>Mean 416</td>
<td>90-1</td>
<td>2-51</td>
<td>2-76</td>
</tr>
<tr>
<td></td>
<td>SD 38</td>
<td>9-0</td>
<td>0-26</td>
<td>6-0</td>
</tr>
<tr>
<td>After spinal fusion</td>
<td>Mean 454</td>
<td>94-8</td>
<td>2-91</td>
<td>85-7</td>
</tr>
<tr>
<td></td>
<td>SD 30</td>
<td>7-5</td>
<td>0-34</td>
<td>9-3</td>
</tr>
</tbody>
</table>

Table 3  Indices of maximal exercise

<table>
<thead>
<tr>
<th></th>
<th>V02 max (l/min)</th>
<th>VR max (ml/kg/min)</th>
<th>VE max (l/min)</th>
<th>VE max × 100% (l/min/VC)</th>
<th>HR max (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before spinal fusion</td>
<td>Mean 1·70</td>
<td>34-6</td>
<td>58-94</td>
<td>1-73</td>
<td>57-0</td>
</tr>
<tr>
<td></td>
<td>SD 0-39</td>
<td>7-1</td>
<td>10-10</td>
<td>0-40</td>
<td>11-2</td>
</tr>
<tr>
<td>After spinal fusion</td>
<td>Mean 1·83</td>
<td>33-6</td>
<td>62-40</td>
<td>1-86</td>
<td>58-4</td>
</tr>
<tr>
<td></td>
<td>SD 0-30</td>
<td>5-3</td>
<td>12-26</td>
<td>0-77</td>
<td>8-4</td>
</tr>
</tbody>
</table>
PATTERN OF VENTILATION
There was a small but insignificant increase in $V_T\text{max}$ after spinal fusion, and this remained insignificant when it was corrected for the increase in vital capacity between the two tests (table 3).

HEART RATE
The mean maximal heart rate was similar in the two tests (table 3). The submaximal heart rate indices of $V_O2$ of 0.75 l and 1.0 l were all lower after surgery ($p<0.05$) (table 4), but when they were corrected for weight gain between the tests (Jones et al, 1975) the improvement disappeared.

Table 4  Heart rate during submaximal exercise

<table>
<thead>
<tr>
<th></th>
<th>HR 0.75 (n=10)</th>
<th>HR 1.0 (n=10)</th>
<th>HR 1.5 (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Observed</td>
<td>% predicted</td>
<td>Observed</td>
</tr>
<tr>
<td>Before spinal fusion</td>
<td>Mean 132-2</td>
<td>111-5</td>
<td>147-3</td>
</tr>
<tr>
<td>SD</td>
<td>12-8</td>
<td>13-9</td>
<td>15-5</td>
</tr>
<tr>
<td>After spinal fusion</td>
<td>Mean 116-2</td>
<td>104-0</td>
<td>131-7</td>
</tr>
<tr>
<td>SD</td>
<td>10-4</td>
<td>11-4</td>
<td>9-7</td>
</tr>
</tbody>
</table>

Discussion
Several previous authors have studied the cardiorespiratory effects of spinal fusion performed for scoliosis. The vital capacity and MVV have been found to be unaffected by spinal fusion (Makley et al, 1968; Westgate and Moe, 1969; Lamarre et al, 1971; Shannon et al, 1971; Meister and Heine, 1973; Stoboy and Speierer, 1975; Henche et al, 1977) or to increase slightly after it (Gazioglu et al, 1968; Lindh and Bjure, 1975). Small increases in $Pao2$ (Shannon et al, 1971; Meister and Heine, 1973) and $SaO2$ (Westgate and Moe, 1969) have been found, and Shannon et al (1971) observed an improvement in the physiological dead space and $PA-ao2$ as well. A marginal increase in $V_O2\text{max}$ during exercise (Stoboy and Speierer, 1975) and an improvement in $Pao2$ after exercise (Shannon et al, 1971) are the only changes in response to exercise after spinal fusion that have been reported.

None of the 10 subjects of this study had any cardiorespiratory disease complicating the scoliosis. They underwent identical resting and exercise tests under the same conditions before and after surgery. Postoperatively they had spent four months in a plaster of Paris jacket and then worn a Milwaukee brace until a year after surgery. All had returned to full activity at least five months before being retested, and none had any cardiac or respiratory symptoms when they were retested.

Estimation of the effect of spinal fusion was complicated by the growth that had taken place between the two tests. Several indices of exercise performance, such as the heart rate (Cotes et al, 1973), vary with body dimensions, and these have had to be taken into account in comparing the results. Thus the maximal oxygen uptake has been expressed as ml/kg body weight/min, maximal tidal volume as a percentage of the vital capacity, and the submaximal heart rates as percentages of the predicted value according to body weight. The usual prediction of PEFR, $FEV_1$, and FVC from height is valueless because straightening of the spine increases the height independently of any growth. Values predicted from the arm span (Godfrey et al, 1970) were therefore used.

Spinal fusion considerably decreased the angle of scoliosis in all the subjects, but the changes in PEFR, $FEV_1$, FVC, and MVV were much less striking. The slight improvements after spinal fusion were not statistically significant. The small increase in $V_O2\text{max}$ was due to the growth of the subjects as it disappeared when the correction for body weight was applied. There was, however, a statistically significant improvement in submaximal ventilation at $V_O2$ of 0.75 and 1.0 l/min, although it was of a small amount. There was no increase in $V_E\text{max}$ and the maximal $V_T$ both in absolute volumes and expressed as a percentage of vital capacity remained unchanged. The apparent slowing of the submaximal heart rate indices after spinal fusion was abolished by correcting for the increase in body weight between the tests and did not represent a true physiological improvement.

Thus the anatomical improvement of the scoliosis after spinal fusion is far greater than the physiological changes during exercise shown in this study. The only significant improvement, after allowing for growth between the tests, was a small decrease in the submaximal ventilatory indices. Possibly, however, spinal fusion prevented a deterioration in exercise performance that would otherwise have occurred in the interval between the tests. The long-term effects of spinal fusion on the development of respiratory failure and pulmonary hypertension are unknown, but the im-
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provement in $P_{aO_2}$ that has been shown (for instance Shannon et al, 1971) may delay or prevent these complications.

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References


Requests for reprints to: Dr J M Shneerson, Westminster Hospital, London SW1.
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J M Shneerson and M A Edgar

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