Cardiac and respiratory responses to exercise in adolescent idiopathic scoliosis

J M SHNEERSON

From the Department of Respiratory Physiology, Cardiothoracic Institute, Brompton Hospital, London

ABSTRACT Twenty girls, aged 11-15 years, with adolescent idiopathic scoliosis were studied before spinal fusion was performed. Each underwent a range of lung function tests and a standardised progressive exercise test on a bicycle ergometer. The resting lung function tests showed reduced peak flow rates, lung volumes, and maximum voluntary ventilation. The maximum oxygen uptake was slightly diminished but maximum exercise ventilation was normal. The latter was achieved by using a greater than normal fraction of the vital capacity in tidal breathing while exercising. Mild hyperventilation during submaximal exercise and a trend towards an exercise tachycardia with increasing body weight were observed.

Hippocrates was the first to record that hunchbacks were “affected with difficulty of breathing and hoarseness”, and Hutchinson demonstrated that their vital capacity could be much reduced. However, only recently has their response to exercise been studied, and pulmonary hypertension, hyperventilation, and limited maximal exercise ventilation and maximal oxygen uptake demonstrated in adult scoliotics. There have been few previous reports of cardiac or respiratory responses to exercise in adolescent scoliotics, and the assessment of fitness, for instance for spinal fusion, has depended on clinical judgment and resting lung function tests. In this study patients with adolescent idiopathic scoliosis underwent a standardised exercise test in order to assess their cardiac and respiratory function and to compare it with the findings in adults with scoliosis.

Methods

Twenty successive girls referred with adolescent idiopathic scoliosis requiring spinal fusion were studied. Their ages ranged from 11 - 15 years (mean 13·7, SD=1·2). None had any cardiac or respiratory disease complicating the scoliosis. The angle of scoliosis was determined by the method of Cobb. It ranged from 40 to 84° (mean 62°, SD=11°). Their mean weight was 47·5 kg (SD=6·1), height 159·3 cm (SD=7·1), and arm span 165·6 cm (SD=5·8).

Their 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 their maximum voluntary ventilation (MVV) with a low resistance nine-litre wet spirometer (PK Morgan).

Exercise was performed while sitting on an electrically - braked bicycle ergometer (Lode). The inspiratory minute volume was measured by a Parkinson Cowan CD4 dry gas meter with an electrical output to a direct-writing ink-jet recorder (Mingograf 81). The gas meter was calibrated with a sinusoidal pump operating at various stroke volumes and frequencies. The gas meter was connected by wide-bore tubing to a low resistance two-way respiratory valve (PK Morgan 71522) with a dead space of 60 ml. The resistance of the system to inspiration was 0·43cm H₂O at 10 l/min and 0·95cm H₂O at 70 l/min. The expired gas passed through wide-bore connecting tubing to a mixing chamber of 7·8 l capacity containing an electrically driven fan. A sample of the mixed expired gas was dried with magnesium perchlorate and passed through a paramagnetic Servomex OA 150 oxygen analyser, and an infrared absorption CO₂ analyser (URAS 4). The Servomex response was linear over the range 14-21% O₂ and the URAS 4 over the range 0-5% CO₂. The 95% response times of both to a square wave of gas leaving the mixing chamber were 4 seconds. Both machines were connected electrically to the Mingograf and were recalibrated before and after each test.

The patients were made familiar with the apparatus on the day before the tests. They rested on the ergometer until their inspired ventilation,
mixed expired gas composition, and heart rate were steady. They then began pedalling, initially at a work rate of 15 watts, The work rate was increased by 15 watts each minute and the patients were encouraged to keep exercising for as long as possible. There were no complications of the procedure.

The regression coefficients of 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).⁶

Results

PEAK FLOW RATE, SPIROMETRY, AND MAXIMUM VOLUNTARY VENTILATION

The PEFR, FEV₁, and FVC results are displayed in table 1. The observed values were all significantly (p < 0.01) less than predicted from their height.⁷ The observed MVV values were all within two standard deviations of normal⁸ (fig 1).

MAXIMUM OXYGEN UPTAKE

Maximum oxygen uptake (VO₂ max) ranged from 1.07 to 2.45 l min⁻¹ (mean 1.60, SD=0.37). Corrected for body weight the mean value was 33.8 ml kg⁻¹ min⁻¹ (range 26.7-50.1, SD=5.6) which is slightly below the normal figure for girls of 40 ml kg⁻¹ min⁻¹.⁹

MAXIMUM EXERCISE VENTILATION

Maximum exercise ventilation (VE max) ranged from 41.09 to 76.34 l min⁻¹ (mean 56.15, SD=9.95). The mean ratio of VE max to MVV (the dyspnoeic index) was normal at 0.65. The individual values for VE max were all within normal limits for girls⁸ (fig 2).

MINUTE VENTILATION

The submaximal minute ventilation (Ve) at VO₂ of 0.75, 1.0, and 1.5 l min⁻¹ is shown in fig 3. The values were not significantly different from the normal

![Graph showing relationship between MVV and height.](http://example.com/graph1.png)

**Fig 1** Relationship between MVV and height showing that the MVV of all 20 subjects fell within two standard deviations of normal (broken lines).

**Fig 2** Relationship between VE max and height showing that VE max of all 20 subjects fell within two standard deviations of normal (broken lines).

<table>
<thead>
<tr>
<th>Table 1 Peak flow rates and spirometry</th>
</tr>
</thead>
<tbody>
<tr>
<td>n=20</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>SD</td>
</tr>
</tbody>
</table>
Cardiac and respiratory responses to exercise in adolescent idiopathic scoliosis

Fig 3  Submaximal ventilatory response during exercise. The broken lines represent the normal limits.10

group.10 The intercept on the y-axis of the regression line of \( \dot{V}E \) on \( \dot{V}O_2 \) was normal (mean = -1.6 l min\(^{-1}\), SD = 3.3; normal: mean = +0.7 l min\(^{-1}\), SD = 1.7). However the regression coefficient of \( \dot{V}E \) on \( \dot{V}O_2 \) was above normal (mean = 33.6, SD = 6.6; normal: mean = 26.9, SD = 2.7), indicating that slight hyperventilation was occurring.

MAXIMAL TIDAL VOLUME AND RESPIRATORY FREQUENCY

Maximal tidal volume (VT max) ranged from 0.96 to 2.67 l (mean 1.70, SD = 0.61). VT max/VC ranged from 0.41 to 0.90. The mean value was 0.67 (SD = 0.13), which is considerably above the normal of 0.50.11 The mean maximal respiratory frequency was normal (45-7 min\(^{-1}\), SD = 13.0).

HEART RATE

The heart rate increased linearly with \( \dot{V}O_2 \) throughout exercise. The mean HR max was 183.5 min\(^{-1}\) (SD = 13.2). The submaximal values of HR at \( \dot{V}O_2 \) of 0.75, 1.0, and 1.5 l min\(^{-1}\) were corrected for body weight and compared to the normal values of Jones et al10 (Table 2). The ratio of the observed/predicted values at each level of \( \dot{V}O_2 \) increased with increasing weight. It exceeded 1.2 for each \( \dot{V}O_2 \) level in the heaviest group of subjects (56–65 kg).

Discussion

The resting lung function tests confirmed the diminished PEFR, FEV\(_1\), FVC, and MVV that are characteristic of severe scoliosis.18 The results obtained in this study have been compared with normal adolescent girls using the observed height to predict the normal values. Although scoliosis shortens the stature, the use of arm span13 is also imperfect as adolescent idiopathic scoliotics have an abnormally large span.14 However, any error incurred would tend to underestimate the severity of the restrictive defect.

The type of progressive exercise test that these subjects carried out has been widely used and, as long as conditions are carefully standardised,18 it gives repeatable results.16 The response to exercise of these adolescent girls shows several differences both from age- and sex-matched normal subjects and from adult scoliotics. Their \( \dot{V}O_2 \) max was 15.5% below the predicted value. The measurement of \( \dot{V}O_2 \) max is technically difficult since it depends on the motivation of the subject to exercise until his physical limit, the type of exercise performed, and the details of the exercise procedure. For these reasons the slightly low value obtained in this study may not be physiologically significant. In any case it is of a smaller degree than that seen in adult scoliotics with a similar severity of deformity.3 The values obtained for \( \dot{V}O_2 \) max were not correlated with \( \dot{V}E \) max. This contrasts with adult scoliotics in whom the low \( \dot{V}E \) max probably limits \( \dot{V}O_2 \) max; in the present study \( \dot{V}E \) max was normal and exercise was able to proceed until, as is normal, it was limited by circulatory factors.

Hyperventilation during submaximal exercise was not detected by analysing the results at \( \dot{V}O_2 \) values of 0.75, 1.0, and 1.5 l min\(^{-1}\). However the mean regression coefficient of \( \dot{V}E \) on \( \dot{V}O_2 \) was 33.6 compared with the normal value of 26.9. Thus some hyperventilation at submaximal exercise is probably occurring as it does in adult scoliotics.3

Interestingly, the pattern of breathing during maximal exercise was abnormal in the same way as seen in adult scoliotics.3 The maximal respiratory frequency was normal but the maximal tidal volume, expressed as a percentage of the vital capacity, was increased. By using a high percentage of the vital capacity the tidal volume is preserved despite the diminution in vital capacity (table 1).

Table 2  Heart rate response during exercise compared with normal10

<table>
<thead>
<tr>
<th>Weight (kg)</th>
<th>n</th>
<th>Observed value (beats min(^{-1}))</th>
<th>% predicted Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR 0.75</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>36-45</td>
<td>8</td>
<td>133-1</td>
<td>9-4</td>
<td>101</td>
</tr>
<tr>
<td>46-55</td>
<td>8</td>
<td>127-6</td>
<td>17-6</td>
<td>108</td>
</tr>
<tr>
<td>56-65</td>
<td>4</td>
<td>130-0</td>
<td>10-4</td>
<td>124</td>
</tr>
<tr>
<td>HR 1.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>36-45</td>
<td>8</td>
<td>154-9</td>
<td>10-2</td>
<td>99</td>
</tr>
<tr>
<td>46-55</td>
<td>8</td>
<td>142-0</td>
<td>19-2</td>
<td>104</td>
</tr>
<tr>
<td>56-65</td>
<td>4</td>
<td>145-5</td>
<td>9-7</td>
<td>121</td>
</tr>
<tr>
<td>HR 1.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>36-45</td>
<td>2</td>
<td>163-0</td>
<td>2-8</td>
<td>85</td>
</tr>
<tr>
<td>46-55</td>
<td>6</td>
<td>162-3</td>
<td>20-7</td>
<td>99</td>
</tr>
<tr>
<td>56-65</td>
<td>4</td>
<td>177-5</td>
<td>9-9</td>
<td>122</td>
</tr>
</tbody>
</table>
The heart rate increased linearly with $\dot{V}O_2$. When it was corrected for body weight an interesting trend of rise in observed/predicted ratio with increasing weight was noted. This is not seen in adult scoliotics. It could indicate that some of these adolescents develop a true exercise tachycardia and that they, therefore, take less exercise and become obese. Alternatively, the heavier, more obese group may simply be less physically fit because of lack of exercise and develop tachycardia during exercise because of this.

Thus these scoliotics, studied within a few years of the onset of their deformity, have acquired the same type of restrictive ventilatory defect and pattern of breathing during exercise as adult scoliotics. Their maximum oxygen uptake is slightly diminished, but much less so than in adults. Their maximum exercise ventilation is still normal, and none had any symptoms of limitation of exercise tolerance. A longitudinal study is required to determine at what later stage in their development the full picture of the abnormalities of the adult scoliotic develop.

I would like to thank Mr M Edgar for allowing me to study patients under his care, Dr F J Prime for his constant help and advice, and Mrs J MacGuigan for typing the manuscript. The work was carried out while I was Clinical Lecturer in the Department of Respiratory Physiology, Cardiothoracic Institute, Brompton Hospital, London and was supported by grants from the Research Committee of the Brompton Hospital and by Boehringer Ingelheim Ltd.

References


