Pectus excavatum (Fig 1) is a relatively common congenital deformity of the chest wall with an incidence of approximately 1 in every 300 births.¹ This condition is more common than Down syndrome, which occurs in 1 in every 600 to 1,000 births.² The condition, often referred to as “funnel chest,” occurs more often in male children than female children (9:1) and accounts for 90% of congenital chest wall deformities. Furthermore, the condition often worsens in late adolescence and early adulthood, and has been associated with diminished exercise capacity.³ The physiologic impact of pectus excavatum (Fig 1) is a relatively common congenital deformity of the chest wall with an incidence of approximately 1 in every 300 births.¹ This condition is more common than Down syndrome, which occurs in 1 in every 600 to 1,000 births.² The condition, often referred to as “funnel chest,” occurs more often in male children than female children (9:1) and accounts for 90% of congenital chest wall deformities. Furthermore, the condition often worsens in late adolescence and early adulthood, and has been associated with diminished exercise capacity.³ The physiologic impact of pectus excavatum.

**Purpose:** Uncertainty exists as to whether pectus excavatum causes true physiologic impairments to exercise performance as opposed to lack of fitness due to reluctance to exercise. The purpose of this study was to examine the effect of pectus excavatum on ventilatory and cardiovascular responses to incremental exercise in physically active patients.

**Methods:** Twenty-one patients with pectus excavatum (age range, 13 to 50 years; mean [± SD] age, 23.6 ± 8.9 years; severity index range, 3.7 to 8.0; mean severity index, 5.1 ± 1.2) were referred for preoperative evaluation. Eighteen of the patients (85%) had a history of performing aerobic activity ranging from 30 min to 2 h per day (mean duration, 1.0 ± 0.61 h per day) for 3 ± 1.5 days per week. Patients performed pulmonary function tests, and submaximal and maximal incremental exercise testing.

**Results:** On maximal exercise testing, the maximum oxygen uptake (Vo2max), and oxygen-pulse were significantly lower than the reference values (t20 = 6.17 [p < 0.0001] and t20 = 4.52 [p < 0.0001], respectively). Furthermore, patients exhibited cardiovascular limitation, but not ventilatory limitation. Despite their high level of habitual exercise activity, the overall metabolic threshold for lactate accumulation was abnormally low (ie, 41% of the reference value for Vo2max) especially in those with a pectus severity index (PSI) of > 4.0 (39% of the reference value of Vo2max), which is consistent with cardiovascular impairment rather than physical deconditioning. Patients with a PSI of > 4.0 were also eight times more likely to have reduced aerobic capacity than patients who had a low severity index, despite their level of exercise participation. On submaximal testing, we found that the time constant for O2 uptake kinetics was 37.4 s for the on-transit and 41.6 s for the off-transit. The observed values for FVC, FEV1, maximum voluntary ventilation, and diffusing capacity of the lung for carbon monoxide were significantly lower than reference values, but those for total lung capacity and residual volume were not significantly lower than reference values.

**Conclusions:** The information derived from this study supports the opinion that pectus excavatum is associated with true physiologic impairment and reduced exercise capacity, predominantly due to impaired cardiovascular performance rather than ventilatory limitation. Furthermore, the impairment is not explained by physical deconditioning.

**Key words:** aerobic fitness; cardiovascular disease; chest wall deformity; clinical exercise physiology; exercise testing; maximum oxygen uptake; oxygen uptake kinetics; pectus excavatum

**Abbreviations:** ATS = American Thoracic Society; DLco = diffusing capacity of the lung for carbon monoxide; fc = cardiac frequency; FRC = functional residual capacity; fRmax = maximum respiratory frequency; HMO = health maintenance organization; MVV = maximum voluntary ventilation; off-τVo2 = off-transit time constant; on-τVo2 = on-transit time constant; PetCO2 = end-tidal gas tension for carbon dioxide; PetO2 = end-tidal gas tension for oxygen; PSI = pectus severity index; RPE = rating of perceived exertion; RV = residual volume; SV = stroke volume; TLC = total lung capacity; τVo2 = time constant for oxygen uptake; UCLA = University of California Los Angeles; Vo2 = carbon dioxide output; Ve = minute ventilation; V˙max = maximum minute ventilation; Vo2 = oxygen uptake; Vo2max = maximum oxygen uptake; Vo2θ = metabolic threshold; Vo2/fc = oxygen pulse; δVo2/δW = work efficiency represented as slope; V˙max = maximum tidal volume; Wss = steady-state work rate
excavatum has been the topic of much debate. Yet, despite numerous published reports, there is no consensus on what degree of physiologic impairment, if any, exists as a result of this anomaly. Symptoms associated with pectus excavatum include fatigue, dyspnea, chest discomfort, and palpitations, often occurring with mild exertion and limited exercise performance. The response to exercise is a function of numerous physiologic mechanisms. The ability to sustain high-intensity exercise is contingent on the following four aerobic parameters: (1) maximum oxygen uptake ($V_{\text{O}_2\text{max}}$); (2) the metabolic threshold ($V_{\text{O}_2}/W$), above which there is a sustained increase in blood lactic acid concentration; (3) work efficiency represented as the slope of the change in $V_{\text{O}_2}$ to the change in work ($\delta V_{\text{O}_2}/\delta W$); and (4) the time constant for oxygen uptake ($\tau V_{\text{O}_2}$). Each of these parameters indirectly relates to the efficiency of the cardiovascular system. The question arises as to whether a deformity of the thorax would affect these parameters of aerobic fitness. Past studies have tried to answer this question; however, there is debate as to whether aerobic fitness is compromised as a function of pectus excavatum or of a sedentary lifestyle leading to physical deconditioning. Thus, the current study sought to examine the strength of the relations between pectus excavatum and physiologic impairment in physically active patients with pectus excavatum.

The degree of physiologic impairment in patients with pectus excavatum is likely to be related to the severity of the deformity. A severity index, based on measurements obtained from a CT scan of the chest, has been advocated as an objective method of determining the degree of deformity in patients. This index is derived, as shown in Figure 2, by dividing the internal transverse distance of the thorax (a) by the vertebral-sternal distance at the most depressed portion of the deformity (b). The normal chest has an index of 2.5. The severity index in pectus excavatum has been reported to correlate with vital capacity as a percentage of the predicted value and with total lung capacity (TLC).

Studies examining the cardiovascular and ventilatory responses during exercise in pectus excavatum patients have not reached a consensus as to the degree of impairment brought about by this deformity. Many of these studies differ in their methodology, thus leading to different findings in similar patients. Also, the majority of studies examining the effect of pectus excavatum have no consistent methodology for determining the degree of impairment.

**Figure 1.** Chest of a 16-year-old boy with symptomatic pectus excavatum.

**Figure 2.** The PSI is derived by dividing the internal transverse distance of the thorax (a) by the vertebral-sternal distance at the most depressed portion of the deformity (b) using a CT scan.

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From the Departments of Medicine and Physiology (Dr. Cooper), the Exercise Physiology Research Laboratory (Mr. Malek), and the Division of Pediatric Surgery (Dr. Fonkalsrud), Department of Surgery, David Geffen School of Medicine at UCLA, Los Angeles, CA.

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pectus severity. The purpose of the present study was to investigate the physiologic responses to exercise performance in pectus excavatum patients using a standardized protocol that optimizes the evaluation of the cardiovascular and ventilatory responses to maximal incremental exercise. Also, we quantified the mode, duration, and frequency of habitual exercise activity in each patient. Our aim was to assess whether parameters of aerobic fitness such as VO₂max would be lower than the reference values specific for each patient, despite regular participation in exercise. We also postulated that if aerobic capacity was impaired, then the τVO₂ would be longer than the values reported for healthy subjects. Last, we hypothesized that as a result of the cardiac compression brought about by pectus excavatum, the observed values for oxygen pulse (VO₂/Fc) would be lower than the reference values specific for each patient.

Materials and Methods

Participants

This study includes 21 pectus excavatum patients (15 men and 6 women) who were referred or self-referred to University of California, Los Angeles (UCLA) Medical Center to be evaluated for potential surgical correction of their chest wall deformities. The subjects underwent preoperative evaluation in the sequence in which they presented. They were not selected for this study on the basis of specific symptoms or habitual exercise activity. Each subject underwent pulmonary function testing and exercise testing. Habitual exercise activity was assessed in terms of mode, frequency, and duration of exercise participation. Approval was given by the UCLA Human Subject Protection Committee for the analysis and reporting of these clinical data.

Pulmonary Function Testing

Spirometry was performed in the seated position with a nose clip that was applied after the subject had rested for at least 10 min. Testing was performed in the Pulmonary Function Laboratory at UCLA Medical Center by certified pulmonary function technologists using equipment and procedures that meet the American Thoracic Society (ATS) criteria for the standardization of spirometry. Forced expiratory maneuvers (eg, FEV₁ and FVC) were performed in triplicate with the minimal requirement of at least three “satisfactory” maneuvers and the best two maneuvers meeting reproducibility criteria within 200 mL (or 5%). Spirometric measures were repeated, if necessary, up to a maximum of eight times in an attempt to achieve both satisfactory and reproducible results. Calibration of the spirometer was performed daily using a precision-calibrated, 3-L syringe. All values were corrected to body temperature and pressure (saturated), using an internal thermometer for temperature measurement. The best FVC and FEV₁ measurements were used for analysis.

Ventilatory capacity was measured as the maximum voluntary ventilation (MVV). The MVV maneuver was performed in the seated position with a nose clip applied. The subject was asked to breathe through a flow transducer as deeply and as rapidly as possible for 12 s. The level of ventilation was expressed in liters per minute and was corrected to body temperature and pressure (saturated). MVV maneuvers were performed in triplicate, and the highest value was used as a measure of ventilatory capacity.

Subdivisions of lung volume were measured by helium dilution, using procedures in accordance with the recommendations of the ATS/European Respiratory Society and the British Thoracic Society. Reported values included the functional residual capacity (FRC), residual volume (RV), and TLC. The three highest acceptable and reproducible values for TLC were recorded, and the mean value calculated. Reproducibility was defined as the two smallest of the three TLC values to be within 10% of the largest one. The calibration of flow, volume, and pressure transducers was performed in accordance with the manufacturer’s specifications.

The measurement of the single-breath diffusing capacity of the lung for carbon monoxide (DLCO) was performed according to the standard technique recommended by the ATS using equipment that meets minimum ATS requirements. Prior to each test session, leak testing was performed, and the accuracy of the volume measurements was checked using a precision-calibrated 3-L syringe. The linearity of the helium and carbon monoxide analyzers, and the accuracy of the timing device in the DLCO apparatus was verified. Measurements were performed with the subject seated and wearing a nose clip. At least two acceptable tests, as defined by the ATS, were performed, and the mean value was calculated (uncorrected for hemoglobin). Reproducibility was defined as two values within 10% of each other or within 3 mL CO per min/mm Hg of the mean value. Peripheral venous hemoglobin and carboxyhemoglobin were measured using a CO oximeter prior to performance of the DLCO test. These values were used to adjust the measured values of DLCO using the method of Coté. The carboxyhemoglobin concentration correction was used only if carboxyhemoglobin concentration was elevated. Both mean DLCO and mean alveolar volume were recorded, and the DLCO/alveolar volume ratio was calculated.

The pulmonary function test results were expressed both as measured values and as percentages of gender-specific reference values using the regression equations of Crapo et al for spirometry, Becklake for subdivisions of lung volume, and Coté et al for single-breath DLCO.

Exercise Testing

Maximal exercise performance was assessed using an incremental exercise protocol on a cycle ergometer (Ergoline 900S; SensorMedics Corp, Yorba Linda, CA). Testing was performed in the UCLA Exercise Physiology Laboratory under the supervision of two authors (MMH and CBC). The work rate increment was judged for each individual subject by considering age, gender, height, weight, and patient-reported degree of functional impairment with the intention of obtaining an exercise phase of 8 to 12 min before exhaustion. The subjects wore a nose clip and breathed through a mouthpiece (model 2700: Hans Rudolph, Kansas City, MO). Minute ventilation (Ve) was measured using a mass flowmeter and expired fractional concentrations of oxygen and carbon dioxide were continuously monitored by paramagnetic oxygen analyzer and nondispersive infrared CO₂ analyzer, respectively (2900: SensorMedics Corp). Oxygen uptake (VO₂) and carbon dioxide output (VCO₂) were calculated breath-by-breath using standard algorithms. Breath-by-breath data were presented as a five-breath rolling average. After a period of stabilization at rest, subjects performed unloaded pedaling (ie, 0 W) for 3 min followed by the ramp increase in work rate (ie, 20 W/min). Subjects were asked to maintain a cycling cadence of 60 revolutions per minute. The ramp work rate increased until subjects reached volitional fatigue. Subject data were used if they
met two of the following three criteria: (1) 90% of age-predicted cardiac frequency (fC); (2) respiratory exchange ratio > 1.1; and (3) a plateau of \( \dot{V}O_2 \) (ie; an increase of < 150 mL/min over the last 30 s of the test). After such time, a 5-min cool-down period with no resistance was performed until the fC was near the rate at unloaded pedaling. A 12-lead ECG was obtained every 2 min throughout the exercise test, and the fC rate was continuously recorded (model QS5000; Quinton; Seattle, WA). Peripheral oxygenation was monitored by a pulse oximeter (Biox 3740; Ohmeda; Helsinki, Finland) attached to a finger. Immediately following exercise termination, the subject was asked to give a rating of perceived exertion (RPE) using the Borg RPE scale25 and to score breathlessness using a 100-mm visual analog scale with standardized instructions.

\( \dot{V}O_2 \)max was compared with standard prediction equations.26 The \( \dot{V}O_2 \)θ, above which a sustained increase in blood lactate occurs, was determined by noninvasive gas exchange measurements using the method of Beaver et al27 in conjunction with an analysis of the ventilatory equivalents (ie, \( VE/\dot{V}O_2 \) and \( VE/\dot{V}CO_2 \)) and end-tidal gas tension for oxygen (\( PETO_2 \)) and for carbon dioxide (\( PTECO_2 \)).28,29 This method relies on the detection of excess CO2 derived from bicarbonate buffering of lactic acid and has been found to have a correlation coefficient of 0.95 when compared with the results of blood lactate analysis.29 Both \( \dot{V}O_2 \)max and \( \dot{V}O_2 \)θ were reported as absolute values in liters per minute. In order to evaluate its normalcy, \( \dot{V}O_2 \) was measured using a mixing chamber technique. \( \dot{V}O_2 \) and cumulative \( \dot{V}O_2 \) were calculated every 20 s and were plotted as functions against time. \( \dot{V}O_2 \) was calculated for both the on-transit \( \dot{V}O_2 \) (on-\( \dot{V}O_2 \)) [ie, 15 W to Wss] and the off-transit \( \dot{V}O_2 \) (off-\( \dot{V}O_2 \)) [ie, Wss to 15 W] from the horizontal displacement of the two components of this plot, which define the lower work rate phases using a previously described geometric analysis.28 This protocol was purposely designed to be submaximal and below the \( \dot{V}O_2 \)θ, and was well-tolerated by the subjects.

**Statistical Analysis**

To test the study hypotheses, pulmonary and cardiovascular measures of interest in pectus excavatum patients were compared to relevant population means. The reference values for pulmonary function indexes were based on age, gender, height, and ethnicity.26 The reference values for cardiovascular indexes were based on age, gender, height, and weight.26

A two-tailed paired \( t \) test was conducted to compare the relevant group means specified in each hypothesis. When appropriate, we present the values as the mean ± SD for relevant groups. We set the \( \alpha \) level at \( p \leq 0.01 \) to control for experimenter error.31,32 Analysis was conducted using a statistical software package (SPSS, version 11.1; SPSS Inc; Chicago, IL).

### V\( \dot{O}_2 \) Kinetics With Exercise

The kinetic response of \( \dot{V}O_2 \) to a step change in work rate was determined from a preliminary submaximal testing protocol performed prior to incremental testing to familiarize the patients with the cycle ergometer prior to maximum exercise testing. The exercise protocol consisted of 6 min at a work rate of 15 W, followed by 6 min at a steady-state work rate (Wss) that was calculated to elicit 40% of the reference \( \dot{V}O_2 \)max, then a further 6 min at a work rate of 15 W. Throughout this protocol, \( \dot{V}O_2 \) was measured using a mixing chamber technique. \( \dot{V}O_2 \) and cumulative \( \dot{V}O_2 \) were calculated every 20 s and were plotted as functions against time.\( \dot{V}O_2 \) was calculated for both the on-transit \( \dot{V}O_2 \) (on-\( \dot{V}O_2 \)) [ie, 15 W to Wss] and the off-transit \( \dot{V}O_2 \) (off-\( \dot{V}O_2 \)) [ie, Wss to 15 W] from the horizontal displacement of the two components of this plot, which define the lower work rate phases using a previously described geometric analysis.28 This protocol was purposely designed to be submaximal and below the \( \dot{V}O_2 \)θ, and was well-tolerated by the subjects.

### Results

#### Subject Characteristics

As can be seen in Tables 1 and 2, our sample consisted of 21 patients (15 men, 6 women). Their

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**Table 1—Characteristics of Patients with Pectus Excavatum**

<table>
<thead>
<tr>
<th>Patient No./Gender</th>
<th>Age, yr</th>
<th>Ht, m</th>
<th>Wt, kg</th>
<th>PSI</th>
<th>Exercise, h/d</th>
<th>Exercise, d/wk</th>
<th>Types of Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F</td>
<td>15</td>
<td>1.47</td>
<td>34</td>
<td>4.7</td>
<td>1.0</td>
<td>3</td>
<td>Jogging</td>
</tr>
<tr>
<td>2/F</td>
<td>13</td>
<td>1.65</td>
<td>49</td>
<td>5.8</td>
<td>1.5</td>
<td>5</td>
<td>Volleyball</td>
</tr>
<tr>
<td>3/M</td>
<td>30</td>
<td>1.96</td>
<td>85</td>
<td>3.7</td>
<td>0.5</td>
<td>3</td>
<td>Jogging</td>
</tr>
<tr>
<td>4/M</td>
<td>14</td>
<td>1.78</td>
<td>60</td>
<td>3.9</td>
<td>1.0</td>
<td>4</td>
<td>Soccer</td>
</tr>
<tr>
<td>5/M</td>
<td>16</td>
<td>1.88</td>
<td>64</td>
<td>6.8</td>
<td>1.0</td>
<td>5</td>
<td>Cross country</td>
</tr>
<tr>
<td>6/M</td>
<td>18</td>
<td>1.91</td>
<td>71</td>
<td>4.2</td>
<td>1.5</td>
<td>5</td>
<td>Soccer</td>
</tr>
<tr>
<td>7/M</td>
<td>27</td>
<td>1.83</td>
<td>69</td>
<td>4.1</td>
<td>1.0</td>
<td>3</td>
<td>Jogging</td>
</tr>
<tr>
<td>8/F</td>
<td>17</td>
<td>1.70</td>
<td>70</td>
<td>5.4</td>
<td>1.0</td>
<td>3</td>
<td>Jogging</td>
</tr>
<tr>
<td>9/M</td>
<td>34</td>
<td>1.91</td>
<td>85</td>
<td>4.4</td>
<td>1.0</td>
<td>3</td>
<td>Cycling</td>
</tr>
<tr>
<td>10/M</td>
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<td>64</td>
<td>3.8</td>
<td>0.5</td>
<td>2</td>
<td>Jogging</td>
</tr>
<tr>
<td>11/M</td>
<td>24</td>
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<td>75</td>
<td>8.0</td>
<td>1.0</td>
<td>3</td>
<td>Cycling</td>
</tr>
<tr>
<td>12/M</td>
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<td>1.75</td>
<td>65</td>
<td>7.4</td>
<td>2.0</td>
<td>4</td>
<td>Jogging</td>
</tr>
<tr>
<td>13/M</td>
<td>14</td>
<td>1.75</td>
<td>55</td>
<td>6.0</td>
<td>2.0</td>
<td>5</td>
<td>Basketball</td>
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<tr>
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<td>22</td>
<td>1.93</td>
<td>80</td>
<td>4.4</td>
<td>0.5</td>
<td>3</td>
<td>Jogging</td>
</tr>
<tr>
<td>15/M</td>
<td>24</td>
<td>1.88</td>
<td>80</td>
<td>5.1</td>
<td>0.0</td>
<td>0</td>
<td>Sedentary</td>
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<tr>
<td>16/F</td>
<td>34</td>
<td>1.70</td>
<td>56</td>
<td>4.4</td>
<td>1.5</td>
<td>4</td>
<td>Swimming</td>
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<tr>
<td>17/F</td>
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<td>1.65</td>
<td>47</td>
<td>5.9</td>
<td>0.0</td>
<td>0</td>
<td>Sedentary</td>
</tr>
<tr>
<td>18/F</td>
<td>32</td>
<td>1.55</td>
<td>43</td>
<td>4.0</td>
<td>1.0</td>
<td>3</td>
<td>Jogging</td>
</tr>
<tr>
<td>19/M</td>
<td>23</td>
<td>1.78</td>
<td>59</td>
<td>4.3</td>
<td>0.0</td>
<td>0</td>
<td>Sedentary</td>
</tr>
<tr>
<td>20/M</td>
<td>25</td>
<td>1.83</td>
<td>76</td>
<td>4.8</td>
<td>1.0</td>
<td>3</td>
<td>Jogging</td>
</tr>
<tr>
<td>21/M</td>
<td>27</td>
<td>1.88</td>
<td>75</td>
<td>6.2</td>
<td>2.0</td>
<td>2</td>
<td>Jogging</td>
</tr>
<tr>
<td>Mean</td>
<td>23.6</td>
<td>1.79</td>
<td>65.0</td>
<td>5.1</td>
<td>1.0</td>
<td>3.0</td>
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<tr>
<td>SD</td>
<td>8.9</td>
<td>0.13</td>
<td>14.2</td>
<td>1.2</td>
<td>0.61</td>
<td>1.5</td>
<td></td>
</tr>
</tbody>
</table>

*F = female; M = male; Ht = height; Wt = weight.*
Pulmonary Function Tests

The mean (± SD) age was 23.6 ± 8.9 years, height was 1.79 ± 0.13 m, body weight was 65.0 ± 14.2 kg, and pectus severity index (PSI) was 5.1 ± 1.2. Eighteen of the patients (85%) performed aerobic activity for a duration ranging from 30 min to 2 h per day (mean duration, 1.0 ± 0.61 h per day) for 3 days per week (± 1.5 days per week). Only three of the patients were sedentary, as defined by performing < 30 min of moderate-intensity exercise at least three times per week.

Pulmonary Function Tests

As shown in Table 3, the observed values for FVC, FEV1, MVV, and Dlco were significantly lower than the reference values (t20 = 4.62 [p < 0.0001]; t20 = 3.59 [p < 0.01]; t20 = 4.75 [p < 0.0001]; t12 = 3.54 [p < 0.01]), but the observed values for TLC, FRC, and RV were normal compared with reference values. Table 4 shows pulmonary function stratified by the PSI, and we found that the majority of parameters for pulmonary function were within the low normal range for the percentage of the reference value.

Exercise Tests

The mean observed V02max was 2.14 ± 0.75 L/min, which corresponded to 75 ± 19% of the predicted V02max (Table 5). A paired t test revealed that the observed values for V02max were significantly lower than the reference values (t20 = 6.17 [p < 0.0001]). The V02 at was detected at a mean V02 of 1.16 ± 0.34 L/min, which corresponded to 41 ± 9% of the mean predicted V02max (Tables 5 and 6). A paired t test revealed no significant differences between the observed and reference values for V02 at (Table 5). Table 6 shows the cardiovascular responses to maximal and submaximal exercise tests stratified by the PSI. We identified a PSI of ≥ 4.0 as the point at which aerobic performance became impaired. This point was chosen because patients in this category of pectus severity had reduced values for V02max and V02 at (74% and 41% of the reference V02max, respectively). We conducted an odds ratio analysis by dividing the sample into four groups, according to whether they had a high or low PSI and a normal or reduced V02 at. We used the PSI of 4.0 to divide the sample based on the above rationale. Also, we used 50% of the predicted V02max to create our two groups for V02 at. This value was selected because it has been used traditionally to distinguish between normal and reduced aerobic responses to incremental exercise in the clinical setting. We then conducted an odds ratio analysis and found that pectus excavatum patients in the group with high PSI were eight times more likely to have reduced V02 at than were those in the group with low PSI.

We examined the kinetic response of V02 with the

### Table 2: Indices of Activity Level for Patients Stratified by PSI

<table>
<thead>
<tr>
<th>Indices</th>
<th>Overall (n = 21)</th>
<th>3.0–3.9 (n = 3)</th>
<th>4.0–4.9 (n = 9)</th>
<th>5.0–5.9 (n = 4)</th>
<th>≥ 6.0 (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>23.6 (8.9)</td>
<td>31.3 (18.0)</td>
<td>25.5 (6.8)</td>
<td>18.0 (4.5)</td>
<td>20.0 (5.4)</td>
</tr>
<tr>
<td>Exercise duration, h/d</td>
<td>1.0 (0.6)</td>
<td>0.7 (0.3)</td>
<td>0.9 (0.5)</td>
<td>0.6 (0.5)</td>
<td>1.6 (0.5)</td>
</tr>
<tr>
<td>Exercise frequency, d/wk</td>
<td>3.0 (1.5)</td>
<td>3.0 (1.0)</td>
<td>3.0 (1.3)</td>
<td>2.0 (2.4)</td>
<td>3.8 (1.3)</td>
</tr>
<tr>
<td>Exercise volume, h/wk</td>
<td>3.0</td>
<td>2.0</td>
<td>2.8</td>
<td>1.3</td>
<td>6.1</td>
</tr>
</tbody>
</table>

*Values given as mean (SD).

### Table 3: Comparison of Observed Pulmonary Function Test Results and Reference Values

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Observed Values</th>
<th>Reference Values</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>FVC, L</td>
<td>21</td>
<td>4.26</td>
<td>1.25</td>
</tr>
<tr>
<td>FEV1, L</td>
<td>21</td>
<td>3.53</td>
<td>0.96</td>
</tr>
<tr>
<td>MVV, L/min</td>
<td>21</td>
<td>12.2</td>
<td>35.9</td>
</tr>
<tr>
<td>TLC, L</td>
<td>16</td>
<td>5.83</td>
<td>1.86</td>
</tr>
<tr>
<td>FRC, L</td>
<td>16</td>
<td>3.07</td>
<td>1.14</td>
</tr>
<tr>
<td>RV, L</td>
<td>16</td>
<td>1.73</td>
<td>0.47</td>
</tr>
<tr>
<td>Dlco, mL/min/mm Hg</td>
<td>13</td>
<td>28.6</td>
<td>8.15</td>
</tr>
</tbody>
</table>

*NS = not significant; Ref = reference value.

1p ≤ 0.01.
onset of moderate constant-load exercise from a baseline of pedaling at a low work rate (ie, on-\(\dot{\text{V}}\text{O}_2\)) as well as the response on reversion from moderate constant-load exercise to pedaling at a low work rate (ie, off-\(\dot{\text{V}}\text{O}_2\)). In healthy subjects, we would expect these values to be approximately 30 s, with equal values for on-\(\dot{\text{V}}\text{O}_2\) and off-\(\dot{\text{V}}\text{O}_2\). In our subjects, the on-\(\dot{\text{V}}\text{O}_2\) was 37.4 ± 10.1 s and the off-\(\dot{\text{V}}\text{O}_2\) was 41.6 ± 13.1 s. The values were not significantly different from each other but might be considered prolonged compared with the expected normal values.

The \(\delta\dot{\text{V}}\text{O}_2/\delta\text{W}\) was evaluated and found to be normal. The \(\delta\dot{\text{V}}\text{O}_2/\delta\text{W}\) slope has remarkable consistency for apparently healthy subjects (10.3 ± 1.0 mL/min/W). In our sample, the range was 8.7 to 12.5 mL/min/W, which lies within the 95% confidence interval of 8.3 to 12.3 mL/min/W. The observed normalcy of \(\delta\dot{\text{V}}\text{O}_2/\delta\text{W}\) argues against the severe impairment of oxygen utilization by skeletal muscles.

Our third hypothesis maintained that \(\dot{\text{V}}\text{O}_2/\dot{\text{f}}\text{c}\), a surrogate measure of cardiac stroke volume (SV), would be lower than reference values due to the deformity of the chest wall that would result in cardiac compression. As seen in Table 5, consistent with our hypothesis, the observed values for \(\dot{\text{V}}\text{O}_2/\dot{\text{f}}\text{c}\) were significantly lower than the reference values (\(t_{20} = 4.52\ [p < 0.0001]\)). Additionally, the mean observed maximum \(\dot{\text{f}}\text{c}\) was 174 ± 19.3 min, which corresponded to 88 ± 10% of the mean predicted maximum \(\dot{\text{f}}\text{c}\) (Table 6). The mean RPE of 16 ± 2.2, as measured by the Borg scale, was consistent with the observed cardiovascular response. Therefore, patients exhibited cardiovascular limitation with exaggerated cardiovascular response patterns, as demonstrated by a steeper relationship between \(f\text{c}\) and \(\dot{\text{V}}\text{O}_2\) (Fig 3), and the normal perception of effort.

We further examined the ventilatory and gas exchange responses to maximal exercise in our patients (Table 7). The maximum \(\dot{\text{V}}\text{e}\) (\(\dot{\text{V}}\text{emax}\)) was 78.7 ± 29.8 L/min, which corresponded to 66 ± 25% of the mean ventilatory capacity, as measured by MVV. This represents a mean ventilatory reserve of 43.8 ± 33.0 L/min. Therefore, patients did not exhibit ventilatory limitation. The mean maximum tidal volume (\(\dot{\text{V}}\text{tmax}\)) was 1.9 ± 0.5 L, and the maximum respiratory frequency (\(f/\text{Rmax}\)) was 43 ± 10 min, both of which were within the expected ranges. Thus, the ventilatory response pattern for our sample was normal as judged by these parameters. Gas exchange mechanisms were normal as judged by ventilatory equivalents (mean \(\dot{\text{V}}\text{e}/\dot{\text{V}}\text{O}_2\) 28.3 ± 5.0; mean \(\dot{\text{V}}\text{e}/\dot{\text{V}}\text{CO}_2\) 31.9 ± 5.8) and end-tidal gas tensions (mean \(\text{PETO}_2\), 104.5 ± 4.3 mm Hg, and mean \(\text{PETCO}_2\), 47.9 ± 3.8 mm Hg).

### Table 5—Comparison of Maximal and Submaximal Exercise Test Results Between Observed Values and Reference Values*

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Observed Values</th>
<th>Reference Values</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>(\dot{\text{V}}\text{O}_2, \text{L/min})</td>
<td>21</td>
<td>2.14</td>
<td>0.7</td>
</tr>
<tr>
<td>(\dot{\text{V}}\text{O}_2, \text{L/min})</td>
<td>19</td>
<td>1.16</td>
<td>0.3</td>
</tr>
<tr>
<td>(\delta\dot{\text{V}}\text{O}_2/\delta\text{W}, \text{mL/min/W})</td>
<td>21</td>
<td>10.6</td>
<td>0.9</td>
</tr>
<tr>
<td>(\text{on-}\dot{\text{V}}\text{O}_2, \text{s})</td>
<td>13</td>
<td>37.4</td>
<td>10.1</td>
</tr>
<tr>
<td>(\text{off-}\dot{\text{V}}\text{O}_2, \text{s})</td>
<td>21</td>
<td>12.4</td>
<td>3.9</td>
</tr>
</tbody>
</table>

*See Table 3 for abbreviations not used in the text.

1p ≤ 0.01.
mean PETCO₂ (41.8 ± 3.9 mm Hg) at the VO₂θ. The patients’ mean perceived breathlessness score of 66.5 ± 17.2, as measured by the visual analog scale, was thought to be consistent with the mean proportion of ventilatory capacity used by these subjects. Peripheral oxygenation was normal throughout the exercise test, as judged by a mean pulse oximetric saturation of 95.5 ± 2.1%.

**Discussion**

There is considerable uncertainty as to whether true physiologic limitations exist in patients with pectus excavatum. In the medical community, some clinicians advocate for surgical correction, whereas others believe that correction has more esthetic than physiologic benefits. Additionally, questions exist as to whether physiologic impairments would merely reflect deconditioning due to lack of physical activity or psychological discouragement from exercise participation. To address these issues, the current study examined the effect of pectus excavatum on ventilatory and cardiovascular responses to exercise in patients who engaged in moderate aerobic activity, on average, for 1 h per day, 3 days per week.

We found that pulmonary function values, as measured by various indexes, were lower than predicted values. Contrary to the findings by Wynn et al., we found no significant differences in FEV₁, which best reflects changes in the medium airways. MVV reflects a combination of chest wall compliance, muscular ability, and patient effort. Our finding that MVV was lower than expected is consistent with that of Orzalesi and Cook, who reported similar results. It should be noted that in our sample the percent predicted for the various indexes ranged from 83 to 92%, which is within the normal range. Thus, while the observed values were lower than the predicted values, there was probably, on average, no clinically meaningful pulmonary function abnormality in our patients. This is further corroborated, as is shown in Table 7, by the ventilatory and gas exchange responses to maximal exercise. We found normal breathing patterns, as measured by V˙e max, V˙t max, and f Rmax. Additionally, gas exchange, as measured by ventilatory equivalents and end-tidal gas tensions at VO₂θ, was normal.

**Table 6—Cardiovascular Responses to Maximal and Submaximal Exercise Tests Stratified by PSI**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Overall (n = 21)</th>
<th>3.0–3.9 (n = 3)</th>
<th>4.0–4.9 (n = 9)</th>
<th>5.0–5.9 (n = 4)</th>
<th>≥ 6.0 (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>f c rest, min</td>
<td>15.4</td>
<td>16.0</td>
<td>16.1</td>
<td>13.5</td>
<td>15.4</td>
</tr>
<tr>
<td>f c max, min</td>
<td>185</td>
<td>98</td>
<td>185</td>
<td>98</td>
<td>177</td>
</tr>
<tr>
<td>RPE, Borg scale†</td>
<td>12.4</td>
<td>85</td>
<td>13.9</td>
<td>89</td>
<td>11.9</td>
</tr>
<tr>
<td>f c, mL</td>
<td>1.16</td>
<td>41</td>
<td>1.37</td>
<td>47</td>
<td>1.12</td>
</tr>
<tr>
<td>f c, mL</td>
<td>14.9</td>
<td>92</td>
<td>13.9</td>
<td>89</td>
<td>11.9</td>
</tr>
<tr>
<td>V O₂/f c, L/min</td>
<td>2.14</td>
<td>75</td>
<td>2.57</td>
<td>86</td>
<td>2.04</td>
</tr>
<tr>
<td>V O₂, L/min</td>
<td>1.16</td>
<td>41</td>
<td>1.37</td>
<td>47</td>
<td>1.12</td>
</tr>
<tr>
<td>ΔV O₂/ΔW, mL/min/W</td>
<td>10.6</td>
<td>11.0</td>
<td>10.0</td>
<td>11.2</td>
<td>0.81</td>
</tr>
<tr>
<td>on-r V O₂e, s</td>
<td>37.4</td>
<td>34.7</td>
<td>34.7</td>
<td>33.9</td>
<td>39.2</td>
</tr>
<tr>
<td>off-r V O₂e, s</td>
<td>41.6</td>
<td>46.5</td>
<td>37.2</td>
<td>45.8</td>
<td>40.1</td>
</tr>
<tr>
<td>f c, mL</td>
<td>12.4</td>
<td>85</td>
<td>13.9</td>
<td>89</td>
<td>11.9</td>
</tr>
<tr>
<td>f c max, min</td>
<td>185</td>
<td>98</td>
<td>185</td>
<td>98</td>
<td>177</td>
</tr>
<tr>
<td>RPE, Borg scale†</td>
<td>15.4</td>
<td>16.0</td>
<td>16.1</td>
<td>13.5</td>
<td>15.4</td>
</tr>
</tbody>
</table>

*fc rest = f c at rest; f c max = maximum f c. See Table 3 for abbreviation not used in the text.
†Scale of 6 to 20 points.
above 90% of its reference value due to physiologic adaptations resulting from training at this level.\textsuperscript{38,40} Thus, the mere fact that this group had a low normal mean percent predicted $V_{\dot{O}_2}^{\text{max}}$ does not negate the arguments that pectus excavatum is associated with physiologic impairment.

In apparently healthy individuals, the $V_{\dot{O}_2}^\theta$ is usually 50 to 60% of the reference value for $V_{\dot{O}_2}^{\text{max}}$, with higher percentages representing a greater level of fitness.\textsuperscript{26,38,39} $V_{\dot{O}_2}^\theta$ is defined as the level of exercise above which aerobic energy generation is supplemented by anaerobic mechanisms with an accumulation of lactic acid as a byproduct. In the current study, the overall $V_{\dot{O}_2}^\theta$ was 41% of the predicted $V_{\dot{O}_2}^{\text{max}}$, and thus was more reduced than $V_{\dot{O}_2}^{\text{max}}$ and was likely to represent significant clinical impairment.\textsuperscript{26,39,41} As the severity index for pectus excavatum increased, the $V_{\dot{O}_2}^\theta$ became more reduced (Fig 4 and Table 6). This has significance because with habitual aerobic exercise, the $V_{\dot{O}_2}^\theta$ occurs at a higher exercise work rate as a result of increased lactate clearance due to aerobic training.\textsuperscript{38}

Our sample was composed of younger individuals who had a habitual exercise regimen, as shown in

\begin{table}
\centering
\begin{tabular}{|l|c|c|c|c|c|c|c|c|}
\hline
Parameters & Overall (n = 21) & 3.0–3.9 (n = 3) & 4.0–4.9 (n = 9) & 5.0–5.9 (n = 4) & $\geq$ 6.0 (n = 5) \\
& Mean & % Ref & Mean & % Ref & Mean & % Ref & Mean & % Ref \\
\hline
$V_{\text{R}}^\text{max}/MVV$ & 78.7 & 66 & 90.00 & 76 & 77.9 & 68 & 54.3 & 51 & 92.8 & 69 \\
Ventilatory reserve, L/min & 43.8 (33.0) & 33.7 (42.9) & 40.5 (36.1) & 46.1 (39) & 51.1 (26.5) & 49.8 (34.8) & 41.1 (26.3) & 44 & 39 & 41.5 & 39.4 \\
$V_{\text{R}}^\text{max}, L$ & 43 & 38 & 46 & 39 & 44 & 41.5 & 39 & 41.5 & 39 & 41.5 & 39 \\
$V_{\text{R}}^\text{max}/V_{\dot{O}_2}^\theta$ & 28.3 & 24.0 & 29.8 & 34.0 & 35.5 & 29.4 & 29.4 & 34.0 & 35.5 & 29.4 & 34.0 \\
$V_{\text{R}}^\text{max}/V_{\dot{O}_2}^\theta$ & 31.9 & 27.3 & 34.0 & 35.5 & 35.5 & 29.4 & 29.4 & 34.0 & 35.5 & 29.4 & 34.0 \\
$PET_{\dot{O}_2}$ at $V_{\dot{O}_2}^\theta$, mm Hg & 104.5 & 102.0 & 106.2 & 106.5 & 102.2 & 41.5 & 41.5 & 43.6 \\
$PET_{\dot{CO}_2}$ at $V_{\dot{O}_2}^\theta$, mm Hg & 41.8 & 44.7 & 39.9 & 41.5 & 43.6 & 41.5 & 41.5 & 43.6 \\
Breathlessness† & 66.5 (17.2) & 70.3 (6.1) & 78.6 (8.5) & 44.2 (17.2) & 60.4 (14.5) & 44.2 (17.2) & 60.4 (14.5) & 44.2 (17.2) & 60.4 (14.5) \\
\hline
\end{tabular}
\caption{Ventilatory and Gas Exchange Responses Stratified by PSI*}
\end{table}

*Values in parentheses are SD. See Table 3 for abbreviation not used in the text.
†By visual analog scale (100-point scale).

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\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure3}
\caption{Cardiovascular response patterns to maximal incremental exercise in pectus excavatum patients stratified by PSI.}
\end{figure}
Table 2, and were without known chronic cardiovascular or pulmonary disease, other than their pectus excavatum. Nevertheless, we found that pectus excavatum patients who had a PSI > 4.0 were eight times more likely to have reduced aerobic fitness than were patients who had a low PSI. The reduced values observed in our sample may be a result of decreased cardiac output due to cardiac compression and therefore a lack of sufficient oxygen delivery to the working muscles. Thus, there is a greater reliance on anaerobic mechanisms to generate energy (ie, adenosine triphosphate) for muscle contraction. The significantly reduced $\dot{V}O_2$ (ie, 38% of the reference $\dot{V}O_2\text{max}$ value) in the subgroup with PSI $\geq 6.0$ illustrates that physiologic impairment is present despite the relatively high volume of aerobic training.

The kinetic response of $\dot{V}O_2$ adjustment to moderate-intensity exercise is one of the important parameters of aerobic function. Whipp et al\(^5\) reported a normal value for $\tau\dot{V}O_2$ in apparently healthy individuals of 35 s, with an SD of 5 s. It is important to note that this measure included a delay time following the onset of exercise from a resting baseline before fitting the data with an exponential function. Bauer et al\(^{34}\) reported a value of 28 s using a single monoexponential model. Ozyener et al\(^{33}\) reported 33 s for the on-$\tau\dot{V}O_2$ value for moderate-intensity exercise. Based on these reports, we believe that the normal value of on-\(\tau\dot{V}O_2\) and off-\(\tau\dot{V}O_2\) is 30 s from a baseline of pedaling at low work rate, represented by a single monoexponential function, in younger individuals who are free of cardiovascular and ventilatory disease and who engage in aerobic activity. Traditionally, $\tau\dot{V}O_2$ has been measured using multiple exercise transitions with breath-by-breath data acquisition to define the profile of change in $\dot{V}O_2$. While the breath-by-breath approach was deemed necessary to clearly define the exercise transition, the variability of breath-by-breath measurements necessitated multiple exercise transitions followed by data averaging before reliable exponential curve fitting could be undertaken. Thus, the use of $\tau\dot{V}O_2$ for clinical assessment is difficult, if not impractical. Through the use of geometric analysis and mixing chamber technology, we were able to measure both the on-$\tau\dot{V}O_2$ and off-$\tau\dot{V}O_2$ in our patients during a single exercise session as part of their preoperative evaluation. Ozyener et al\(^{33}\) have reported similar values for on-$\tau\dot{V}O_2$ and off-$\tau\dot{V}O_2$. We corroborated this finding using a different methodology, although our values for $\tau\dot{V}O_2$ were numerically larger.

The $\tau\dot{V}O_2$ kinetics is related to physical fitness, being shorter in trained individuals and longer in older sedentary individuals.\(^{42}\) $\tau\dot{V}O_2$ has an inverse correlation with $V_{O_2\text{max}}$ and is prolonged in patients with COPD,\(^{43}\) congenital heart disease, and β-blockade.\(^{44}\) Although there is some debate regarding the
control mechanisms of $\tau_V\dot{O}_2$, its magnitude is likely to be influenced by the central circulatory response to exercise onset as well as by muscle $\dot{O}_2$. Studies examining various cardiovascular conditions such as congenital heart disease, heart failure, and heart transplantation have found $\tau_V\dot{O}_2$ to be prolonged. We suspect that the $\tau_V\dot{O}_2$ for both on-transit and off-transit is longer than normal in our patients, taking into account their age and level of habitual exercise activity. This observation will need to be confirmed in a greater number of patients compared with age-matched control subjects. If so confirmed, the prolongation of $\tau_V\dot{O}_2$ in patients with pectus excavatum may be a reflection of impaired cardiovascular responsiveness to exercise due to the central cardiac compression.

Early pathologic studies of pectus excavatum patients demonstrated a compression of the heart between the vertebral column and the depressed sternum. Several studies have provided evidence that cardiac function is impaired during upright exercise but is relatively normal at rest and when performed in the supine position. During seated exercise, SV in healthy individuals increases and approaches 5 to 15% below the SV achieved during exercise in a supine position. However, in 1962, Bevegård performed cardiac catheterization in 16 patients with pectus excavatum and studied their central hemodynamics during rest and exercise, both supine and upright. Cardiac output and SV were normal at rest and decreased normally from the supine to sitting positions. However, in the seated position the measured increase in SV with exercise (18.5%) was significantly lower than the 51.0% expected in healthy persons. Similarly in another study of pectus excavatum patients, Gattiker and Bühlmann found that the mean SV for exercise in the seated position was 25% below those values for the supine position. This was above the 5 to 15% for healthy individuals. However, Gattiker and Bühlmann did not measure $V_o_2$, but rather measured the work rate.

Garusi and D’Ettore found through the use of cardiac angiography that, while the heart was shifted to the left, the dimensions of the cardiac chambers were essentially normal. Beiser et al performed cardiac catheterization on three patients before and after corrective surgery, and found that cardiac output during intense upright exercise was below the normal range in two patients and was at the lower limits in the third patient. However, because of the small sample size it is difficult to draw generalizations based on these findings. Additionally, Beiser et al used a treadmill protocol of 5 to 6 min to determine physiologic responses to maximum exercise but did not state how the patients were incrementated to the maximum exercise capacity. This is problematic, because a short duration with large increments has been found to reduce $V_o_2\text{max}$ by about 10%. Although studies vary in their methodologies and findings, there appears likely to be some impairment of cardiac function in pectus excavatum patients.

The $\delta f/c/\delta V_o_2$ is the slope of the relationship between heart rate and $V_o_2$ during incremental exercise. This slope is related both to SV and the oxygen content between arterial and mixed venous blood. $\delta f/c/\delta V_o_2$ is also the reciprocal of the asymptotic $V_o_2/fc$, which is a measure of cardiovascular efficiency in terms of milliliters per beat. Thus, $V_o_2/fc$ is closely related to cardiac SV and can be used to estimate SV at various stages of incremental exercise testing. Wynn et al studied the cardiovascular response to exercise in 13 pectus excavatum patients and found that there was no significant difference in cardiac output at maximal exercise between the group of patients who had undergone corrective surgery and the group of patients who elected not to have corrective surgery. Also, the investigators found that both groups exhibited an appropriate increase in SV with exercise and that the relationship of cardiac output to $V_o_2$ was normal. Quigley et al reported a significant increase in $V_o_2/fc$ following corrective surgery in 15 pectus excavatum patients and concluded that the increase was due to relief from cardiac compression. When seated, the heart of the pectus excavatum patient is positioned ventrally by the hyperventilated lungs during exercise, while the right atrium is compressed because it is directly posterior to the sternum. Also, studies have found that the right ventricle is less concentric, more anterior relative to the sternum, and more distensible than the left ventricle. During exercise, the right atrium may not be completely filled, thus an inadequate amount of blood is delivered to the right ventricle resulting ultimately in a decreased SV. Peterson et al found, in preoperative radiographs, that the sternum impinged on the right side of the heart. Additionally, the investigators reported that the decrease in right ventricular ejection fraction at rest following corrective surgery was due to the increase in the right ventricular end-diastolic volume. Kowalewski et al found increases in right ventricular systolic BP, diastolic BP, and SV after corrective surgery in 42 pectus excavatum patients due to the relief of cardiac compression by the sternum. Mocchegiani et al found that in pectus excavatum patients the right ventricle outflow diameter at the aortic root level was significantly narrower and that the right ventricular end-diastolic and end-systolic areas were larger than those in control subjects. Additionally, the investigators
found that 71% of their pectus excavatum patients showed unusual morphologic features of the right ventricle.

Morshuis et al\(^5\)\(^7\) studied 35 pectus excavatum patients before and 1 year after corrective surgery, and found a significant increase in \(V_{o2}^{\text{max}}\) and \(V_{o2/\text{fc}}\) after corrective surgery, but they did not state whether subjects were physically active during the 12 months following the surgery. Thus, the improvements in \(V_{o2}^{\text{max}}\) and \(V_{o2/\text{fc}}\) may be a result of physical conditioning rather than of the corrective surgery. Quigley et al\(^7\) found no significant differences in \(V_{o2}^{\text{max}}, V_{o2/\theta}\), and \(V_{o2/\text{fc}}\) between pectus excavatum patients and control subjects. However, it should be noted that these investigators found that only \(V_{o2/\text{fc}}\) increased significantly in pectus excavatum patients after corrective surgery. These findings are consistent with those of other studies\(^5\)\(^4\),\(^5\)\(^8\) that have found a significant difference in \(V_{o2/\text{fc}}\) when comparing pectus excavatum patients to control subjects or in preoperative and postoperative research designs.

The current study is uniquely different from past research on pectus excavatum, because of the following: first, we examined exercise impairment in relation to the PSI, and, to our knowledge, this is the first study that has documented this type of assessment; and, second, we quantified and evaluated habitual exercise activity, and were able to conclude that deconditioning was an unlikely explanation of aerobic impairment. This contradicts arguments that reduced aerobic capacity in pectus excavatum patients is a result of deconditioning and not the chest wall deformity. Previous studies have only mentioned that patients were physically active, but have not reported the frequency, duration, or mode of the exercise regimen or the control groups used for comparison. The ability to determine whether reduced aerobic capacity in pectus excavatum patients is a result of the deformity or a sedentary lifestyle is important because the former is anatomic in nature, interfering with circulatory responses and can be corrected only through surgical intervention, whereas the latter can be corrected through an exercise regimen. Third, we evaluated central cardiac performance by examining \(V_{o2/\text{fc}}\) and \(\tau V_{o2}\). To our knowledge, no other studies have reported \(\tau V_{o2}\) values in this population.

Hall et al\(^1\)\(^9\)\(^0\) have commented on methodological problems that make it difficult to judge the reliability and validity of previous findings in studies of patients with pectus excavatum. Future studies examining the physiologic effect of pectus excavatum would benefit by incorporating a number of standards.\(^5\)\(^9\)--\(^6\)\(^1\) First, a single, objective, reliable, and universally accepted approach to measuring the severity of pectus excavatum should be implemented. This will allow investigators to examine the relevance of physiologic impairment based on the degree of deformity across studies. Second, studies examining preoperative cardiovascular parameters in pectus excavatum patients should attempt to quantify the mode, frequency, and duration of habitual exercise activity. Third, studies examining cardiopulmonary function in pectus excavatum patients would benefit from using a standardized exercise-testing protocol such as that described by Buchfuhrer et al\(^1\)\(^4\), which would allow for a more reliable and valid measure of cardiorespiratory function. By following these recommendations, a better understanding of the physiologic effect brought on by pectus excavatum would be established and would help to clarify the inconsistencies found between studies.

Symptoms of pectus excavatum are recognized infrequently during early childhood, and most patients are therefore advised by well-intentioned family physicians or pediatricians that the anomaly will improve with age without affecting the heart and lung function. Furthermore, in the present managed care environment, health maintenance organizations (HMOs) seem increasingly reluctant to authorize corrective surgery for pectus excavatum. The reason for this is that HMOs believe that corrective surgery for pectus excavatum benefits the patient more esthetically than physiologically. Raggi et al\(^6\)\(^2\) stated, “... the heart appears trapped in a chest cavity too small for its size, and its anatomic architecture is seemingly altered in an attempt to accommodate these insufficient dimensions.” In the current study, we found that cardiovascular function was significantly impaired in patients in whom pectus excavatum was diagnosed, even though the majority of patients were aerobically very active. This information supports the opinion that pectus excavatum is associated with true physiologic impairment and, thus, challenges the common belief held by HMOs that the purpose of corrective surgery is cosmetic in nature. Our findings strengthen the clinical justification for corrective surgery, particularly in patients with severe deformity.

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