PULMONARY FUNCTION IN KYPHOSCOLIOSIS

BY

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This investigation was part of a study of kyphoscoliosis. Its objective was to determine the functional alterations in the chest cage and lung. These problems have, as we indicate in a brief review, already aroused considerable interest. However, our approach has differed from that of most previous workers in several respects. Our patients were both children (Caro and Gucker, 1958) and adults. We were thus able to compare these two groups and to make some assessment of the natural progress of the condition. Our investigations were centered on the mechanics of breathing, an aspect which seemed to us to be of the greatest significance. Finally, we have supplemented our measurements on patients with experimental studies on normal man (Caro, Butler, and DuBois, 1960; Butler, Caro, Alcala, and DuBois, 1960). From our findings we have attempted to describe both the nature and sequence of development of the underlying physiological disturbances.

REVIEW

The following is a brief review of the literature on alteration of the function of the lungs in kyphoscoliosis. In 1854, Schneevoigt, using a spirometer, reported that the vital capacity was diminished. Flagstad and Kollman (1928) demonstrated that the vital capacity was most reduced when the curvature was in the dorsal spine. Chapman, Dill, and Graybiel reviewed the field in 1939 and observed that the total lung capacity and its subdivisions were decreased. They found that the basal metabolic rate and the cardiac output, measured by the acetylene uptake method of Grollman, were approximately normal. Hypercapnoea was detected in several of their patients. Itoicovici and Lyons (1956) confirmed the finding of reduced total lung capacity, but noted that the residual volume and intrapulmonary gas mixing were normal. They argued that these findings, together with only a moderate reduction of the maximal breathing capacity, were against the presence of emphysema. Similar conclusions were arrived at by Hanley, Platts, Clifton, and Morris (1958), who performed cardiac catheterization and found a raised pulmonary arterial pressure. Ferris, Whittenberger, and Gallagher (1952b) drew attention to the greater pressure required to distend the lungs and thoracic cage of patients with poliomyelitic respiratory paralysis. Airway resistance and pulmonary tissue resistance have been determined in a few patients by Marshall and DuBois (1956), and the functional residual capacity was measured by Bedell, Marshall, DuBois, and Comroe (1956), using both the body plethysmograph method and the nitrogen dilution methods, to search for the presence of "trapped gas" which was absent. Bergofsky, Turino, and Fishman (1959) reviewed the recent literature. In addition, they reported that the work of breathing was greatly increased in patients who were studied when voluntarily relaxed and passively ventilated, the principal cause being diminished compliance of the chest cage. The work done in overcoming the non-elastic resistance of the lung was normal, and they considered that pulmonary emphysema was usually absent. Spontaneous breathing was usually rapid and shallow, and, although this minimized the work of breathing, it frequently led to alveolar hypoventilation. They found that the pulmonary arterial pressure was raised in all the patients during exercise, but detected pulmonary hypertension at rest only in those who had been in right heart failure. In the latter patients, the pulmonary diffusing capacity was decreased.

METHOD

We studied 38 patients (20 women and 18 men) who had kyphoscoliosis. Thirty-three were under 21 years of age and the remaining five were 31 to 56 years old. The spinal curvature was the result of
PULMONARY FUNCTION IN KYPHOSCOLIOSIS

patients. Seven series ofatelectasis before curvature any underwent poliomyelitis (12 patients) or congenital. Only three patients had undergone any orthopaedic treatment for spinal curvature before study.

The anatomical situation and severity of the spinal deformity was measured on supine and erect radiographs, using the method of Cobb (1948). Thoracic or thoraco-lumbar curves were the most common, and the severity varied from 6° to 128° (mean 54°). No patient had been in heart failure, but pulmonary atelectasis had been detected in three poliomyelitic patients. Seven poliomyelitic, two idiopathic, and one congenital sciotic patient had suffered from pneumonia. The incidence of pneumonia in the entire series was therefore 26%.

As a control group, we studied 50 healthy children and adults who ranged in age from 4 to 40 years. The following methods were used for measuring pulmonary function: Vital capacity, inspiratory capacity, expiratory reserve volume, and the maximal mid-expiratory flow (Leuallen and Fowler, 1955) were measured with an Osborne–Blandtett recording spirometer. The maximal breathing capacity was determined using a Hans Rudolph high-velocity breathing valve and a Tissot spirometer. These measurements were made in both the seated and supine positions, and the best of approximately three attempts was recorded. The functional residual capacity was measured seated, by means of a body plethysmograph (DuBois, Botelho, Bedell, Marshall, and Comroe, 1956). Volumes were expressed at body temperature, pressure, and saturation. The distribution of inspired gas was determined by means of the nitrogen meter single-breath test (Comroe and Fowler, 1951) and the diffusing capacity of the lung by the single-breath carbon monoxide and helium method (Ogilvie, Forster, Blakemore, and Morton, 1957).

Lung compliance was measured with the patient seated, both during spontaneous breathing and over the course of slow, deep individual breaths. The tidal volume was obtained by electrical integration of a Lilly flowmeter signal and registered on the vertical axis of a cathode-ray oscilloscope. The oesophageal pressure, minus a voltage proportional to flow, was recorded on the horizontal axis (Marshall and DuBois, 1956).

The pressure-volume relationship of the thoracic cage was measured in patients who were under general anaesthesia and had been rendered apnoeic either by passive hyperventilation or by the administration of a neuromuscular blocking agent (Table I). The lungs and thorax were inflated through an airtight endotracheal tube to a series of pressures not exceeding 35 cm. H₂O, in random sequence. The transthoracic cage pressure (oesophageal pressure minus atmospheric pressure) was recorded with an oesophageal balloon, capacitance manometer, and direct-writing recorder. Pressure was measured after approximately five seconds of sustained inflation and again after the lungs and chest cage had been permitted to deflate into a recording spirometer. Airway conductance, which is defined as the rate of airflow at the mouth divided by the alveolar-mouth pressure gradient, was measured by means of a body plethysmograph during shallow panting at about resting lung volume (DuBois, Botelho, and Comroe, 1956). It was compared with the simultaneously determined thoracic gas volume (Briscoe and DuBois, 1958).

**TABLE I**

<table>
<thead>
<tr>
<th>Name</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Estalactance of Thoracic Cage</th>
<th>Name</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Estalactance of Thoracic Cage</th>
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</thead>
<tbody>
<tr>
<td>RS†</td>
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<td>3</td>
<td>7</td>
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<td>5</td>
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<tr>
<td>MD‡</td>
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<td>4</td>
<td>9</td>
<td>A 8 F 4</td>
<td>3</td>
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<td>5</td>
</tr>
<tr>
<td>JN³</td>
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<td>3</td>
<td>B 11 F 4</td>
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<td>1</td>
<td>4</td>
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<tr>
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<td>0</td>
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<td>8</td>
<td>J 12 F 4</td>
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<td>2</td>
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<tr>
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<td>AP⁷</td>
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<tr>
<td>IC¹⁰</td>
<td>F 12 M 5</td>
<td>14</td>
<td>5</td>
<td>T 15 M 4</td>
<td>11</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

Mean 9-4 S.E. 1-5 Mean 8-6 S.E. 1-2

* Poliomyelitc.
† Idiopathic.
‡ For method of computing elastace see text.
§ Nitrous oxide, trichloroethylene, succinylcholine chloride.
## Sodium thiopental, nitrous oxide, succinylcholine chloride.
△ Cyclopropane, succinylcholine chloride.
□ Cyclopropane and hyperventilation.

Records were also obtained of the maximal pressure that the subjects could exert against a closed breathing tube at about resting lung volume. This pressure was measured with a capacitance manometer. A 2-in. long, 18-gauge needle was inserted into the breathing tube to serve as a slow leak, thereby ensuring that the pressure was developed by the respiratory muscles and not by the muscles of the oropharynx.

**RESULTS**

**Prediction of Normal Values and Results in Normals.**—It is not easy to predict normal values for respiration in either children or adults whose growth patterns have been disturbed. We adopted methods which were based on values predicted from more than one parameter of body size, as these appeared least subject to error. The predicted vital capacity and similarly the functional residual capacity, residual volume, and total lung capacity, in children aged 6–14 years, was the quadratic mean (the square root of
half the sum of the squares) of the values predicted from both height and weight (Engström, Karlberg, and Kraepelien, 1956). In older children, these volumes were predicted from the body surface area and in adults from the average of the values predicted for sex, age, height, weight, and surface area (Needham, Rogan, and McDonald, 1954). The maximal mid-expiratory flow was predicted from the following regression lines, obtained on 18 normal children during the course of this investigation: M.M.F. (l./min.) = (197 × S.A.) − 52 (Fig. 1). The maximal mid-expiratory flow in adults. The maximal breathing capacity was predicted by averaging the four values obtained from age, height, weight, and surface area in either male or female children (Ferris et al., 1952b; Ferris and Smith, 1953). In adults the predicted values for age and surface area were averaged (Needham et al., 1954).

The N₂ meter single-breath test gave an average value of 1.2% N₂ (S.D. 0.3) in 13 normal children we studied (Fig. 3, left-hand column), and published values were taken for adults. The pulmonary diffusing capacity was predicted from published values based on body surface area.

Lung compliance was measured in 24 normal children and adults and followed the regression line: \( C_L \left( \text{l./cm. H}_2\text{O}\right) = (0.061 \times \text{F.R.C.}) + 0.004 \) (Fig. 4). The approximate range of normal data for adults. The maximal breathing capacity was predicted by averaging the four values obtained from age, height, weight, and surface area in either male or female children (Ferris et al., 1952b; Ferris and Smith, 1953). In adults the predicted values for age and surface area were averaged (Needham et al., 1954).

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is shown. Airway conductance was predicted from the formula: \( G_a = 0.24 \times T.G.V \) (Fig. 5) (Briscoe and DuBois, 1958). The range of normal has been illustrated. The data obtained on normal children during this study fall within the predicted limits. The pressure-volume curves of the thoracic cages of anaesthetized normal children are shown in Fig. 6. The elastance of the thoracic cage was calculated from the transthoracic cage pressure change per unit change of lung volume for an inflation above relaxation lung volume equivalent to 10% of the total lung capacity (Fig. 7 and Table I). In four children the pressure-volume relationship of the thoracic cage was also measured prone in order to test for any possible artefact of oesophageal pressure due to the supine posture. Two of the subjects were kept attached to the spirometer while they were being turned prone. The change in resting lung volume was less than 0.1 litre. Thoracic cage elastance in these four subjects was not significantly different whether they were supine or prone. (Mean thoracic cage elastance: supine 9.4 cm. H₂O/l, S.E. 3.0; prone 7.7 cm. H₂O/l, S.E. 2.0.)

**Table II**

**MAXIMAL VOLUNTARY ALVEOLAR Pressures AT Resting Lung Volume IN Normal Subjects AND Kyphoscoliotic Patients**

<table>
<thead>
<tr>
<th>Age (yr.)</th>
<th>Normal</th>
<th>Kyphoscoliotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>Pressure (mm. Hg)</td>
<td>Pressure (mm. Hg)</td>
<td>Pressure (mm. Hg)</td>
</tr>
<tr>
<td>7</td>
<td>40</td>
<td>24</td>
</tr>
<tr>
<td>8</td>
<td>93</td>
<td>90</td>
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<td>10</td>
<td>37</td>
<td>52</td>
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<tr>
<td>12</td>
<td>60</td>
<td>65</td>
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<td>14</td>
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<td>64</td>
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<td>15</td>
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<td>75</td>
<td>76</td>
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<tr>
<td>31</td>
<td>73</td>
<td>60</td>
</tr>
<tr>
<td>33</td>
<td>66</td>
<td>66</td>
</tr>
</tbody>
</table>

* Poliomyelitic. † Idiopathic. § Post-spinal fusion.
Values of maximal voluntary alveolar pressure in normal children and adults are shown in Table II.

![Graph showing the effect of age on thoracic cage elastance](image)

**Fig. 7.**—The elastance of the thoracic cage in children. Thoracic cage elastance was normal or low in paralytic and non-paralytic kyphoscoliotic children when compared with values obtained in healthy children.

**Effect of Altered Lung Volume on Maximal Expiratory Flow Rate and Maximal Breathing Capacity.**—When the total lung capacity was reduced in normal young adults there was definite limitation of the forced expiratory flow rate. This was less marked when the chest was constricted with a special chest corset than after voluntary reduction of lung volume (Table III). The chest corset also had the effect of reducing the maximal breathing capacity in normal subjects: their tidal volumes became smaller, but respiratory frequency was unchanged or increased.

**Patients**

**Lung Volumes.**—The total lung capacity, vital capacity, and functional residual capacity were considerably reduced, but the residual volume was usually normal (Table IV and Fig. 8). The lung volumes were generally smaller in poliomyelitic than in non-paralytic patients, and the vital capacity was particularly reduced (mean 35\% of normal values, S.E. in parentheses.† Measured between 200 and 1,200 ml. of expired volume.)

**Table III**

<table>
<thead>
<tr>
<th>No.</th>
<th>Control Period</th>
<th>With Chest Corset</th>
<th>Voluntary Reduction of Total Lung Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vital capacity (l.)</strong></td>
<td>4</td>
<td>4.53 (0.67)</td>
<td>1.82 (0.29)</td>
</tr>
<tr>
<td><strong>Maximal expiratory flow rate (l./min.)</strong></td>
<td>4</td>
<td>469 (64)</td>
<td>153 (26)</td>
</tr>
<tr>
<td><strong>Maximal mid-expiratory flow (l./min.)</strong></td>
<td>4</td>
<td>260 (16)</td>
<td>117 (5)</td>
</tr>
<tr>
<td><strong>1 sec. timed vital capacity (%)</strong></td>
<td>4</td>
<td>86 (2)</td>
<td>89 (5)</td>
</tr>
<tr>
<td><strong>Maximal breathing capacity (l./min.)</strong></td>
<td>3</td>
<td>132 (22)</td>
<td>91 (18)</td>
</tr>
<tr>
<td><strong>Tidal volume (l.)</strong></td>
<td>3</td>
<td>0.92 (0.08)</td>
<td>0.50 (0.10)</td>
</tr>
<tr>
<td><strong>Respiratory frequency (r.p.m.)</strong></td>
<td>3</td>
<td>146 (16)</td>
<td>181 (12)</td>
</tr>
</tbody>
</table>

* Mean values, S.E. in parentheses.
† Measured between 200 and 1,200 ml. of expired volume.
VITAL CAPACITY

Airway conductance

Lung

lung of patients

Number

volume Maximal mid-expiratory
predicted, S.D.10) in seven poliomyelitic patients

who had either paradoxical motion of the chest
cage or radiologically detectable diaphragmatic
paralysis. The size of the vital capacity was not
statistically correlated with either the anatomical
site of the spinal curvature or its severity in degree.
Furthermore, the vital capacity was not signifi-
cantly different, whether measured supine or
seated, in the 29 patients studied in these two
positions. However, in three out of four patients
with diaphragmatic paralysis, the mean vital
capacity was increased from 1.15 l., supine, to
1.55 l., when seated. By contrast, the mean vital
capacity fell from 2.67 l., supine, to 1.52 l., seated,
in two other poliomyelitic patients who had very
weak spinal extensor muscles but no diaphrag-
matic paralysis. It seemed likely that a small
vital capacity predisposed to pneumonia, for a
history of one or more episodes was given by seven
of the 10 patients whose vital capacity was less
than 40% of predicted.

DISTRIBUTION OF INSPIRED GAS.—There was
slight unevenness of distribution of inspired gas
in many of the young poliomyelitic and non-
paralytic patients (Fig. 3). In two of the five adults
the N2 slope measured 6.0%, while in the
remainder it was normal. Grossly uneven
distribution was not found.

MAXIMAL MID-EXPIRATORY FLOW.—The maxi-
mal mid-expiratory flow was slightly to moderately
reduced in the adult patients (mean 62%, S.D. 32)
when normal values were predicted on the basis
of age and sex. It was also decreased in the
younger patients when compared with normal
values predicted from body surface area (Fig. 1
and Table IV). It was, however, notable that the
maximal mid-expiratory flow in these younger
patients was generally normal relative to values
predicted from the functional residual capacity
(Fig. 2).

MAXIMAL BREATHING CAPACITY.—The maximal
breathing capacity was slightly decreased in most
poliomyelitic and non-paralytic patients (Table
IV). The tidal volumes spontaneously selected by

patients during the test were considerably smaller
than those used by normal individuals of compar-
able weight (Fig. 9). However, respiratory
frequency was normal (Fig. 10).

![Graph](https://example.com/graph1.png)

**Fig. 9.**—The tidal volume, during the performance of the maximum breathing capacity, and body weight. When compared with values obtained in normal subjects, the tidal volume was usually small in both paralytic and non-paralytic kyphoscoliotic patients.

![Graph](https://example.com/graph2.png)

**Fig. 10.**—The frequency of respiration during the maximum breathing capacity test and body weight. Respiratory frequency was normal or increased in paralytic and non-paralytic kyphoscoliotic patients when compared with values obtained in healthy subjects.
Lung Compliance.—The lung compliance was low in the majority of patients in the study (Table IV and Fig. 4). This decrease was not the result of small lungs, for the predicted values were based on the observed functional residual capacity. Neither was this change due to uneven distribution of inspired gas and uneven pulmonary time constants (Otis, McIver, Mead, McCroy, Selverstone, and Radford, 1956), for the lung compliance was not significantly different when measured in several patients during slow deep breaths or during spontaneous respiration.

Airway Conductance.—The airway conductance was normal or raised relative to the observed thoracic gas volume in every patient studied (Table IV and Fig. 5).

Pressure-volume Relationship of the Thoracic Cage.—Pressure-volume diagrams of the thoracic cage were obtained on six kyphoscoliotic children at the time of their being anesthetized to undergo orthopaedic surgery (Table I and Fig. 6). The elastance of the thoracic cage was calculated and found to be either equal to or lower than the values obtained in a group of normal children of comparable age.

The pressure-volume relationship of the thoracic cage was also measured in a severely kyphotic elderly woman. She was in respiratory failure, with severe respiratory acidosis, and had been treated with oxygen, antibiotics, intermittent positive pressure breathing, and a tracheotomy. Despite these measures she succumbed. Before death a positive pressure of 15 cm. H₂O, delivered by a Bennett valve, failed to increase her tidal volume to more than approximately 250 ml., giving a calculated elastance for the lung-thorax system of 15/0.250 (or 60 cm. H₂O/l.) (normal is approximately 10 cm. H₂O/l.). Post-mortem examination was performed at a time when, from the flaccidity of the arms, rigor mortis was judged to be absent. Measured volumes of air were introduced stepwise into the lungs via a cuffed endotracheal tube, and the resulting static pressure was read from a manometer. The elastance of the lungs and thorax was 125 cm. H₂O/l. The chest cage was then widely opened, the ribs were retracted, and pressure inflation and deflation of the lungs alone was performed. The compliance of the lungs was 0.031 l./cm. H₂O. The compliance of the chest wall, after death, was therefore 0.011 l./cm. H₂O. While living and also at necropsy the rib cage of this patient seemed hard, inflexible, and immobile. The physiological findings on the chest cage are in marked contrast to those obtained in our young patients. The chest wall of the young patients was inflated with ease, whereas that of the older patient, in respiratory failure, could not be inflated sufficiently to ventilate the lungs adequately.

Maximal Voluntary Alveolar Pressure.—The wide variation of pressure observed among normal subjects made it impossible to establish any precise limits. However, the maximal pressures developed by patients were generally within the normal range (Table II).

Pulmonary Diffusing Capacity.—The diffusing capacity was frequently slightly decreased, though not to levels that would constitute a severe diffusion defect (Table V). The mean diffusing capacity was 4.3 ml./min./mm. Hg less than that predicted (S.D. 5, S.E. 1.7, P<0.05).

### Table V

<table>
<thead>
<tr>
<th>Name</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>DLCO (ml./min./mm. Hg)</th>
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<tr>
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<td>M</td>
<td>13-7</td>
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<td>M</td>
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</tbody>
</table>

* Poliomyelitic.
† Histiopathic.
‡ Supine in cast.
§ Wearing a brace and corset, body cast and spinal fusion.

Discussion

In our patients, measurements of lung volume, maximal breathing capacity, distribution of inspired air, and the pulmonary diffusing capacity corresponded closely to those reported by other authors and cited in the introduction. However, our studies of the mechanics of breathing, coupled with the results of our various experiments on normal subjects, have led us to new conceptions of the functional changes in the lung and chest cage in kyphoscoliosis.

The chest cage was not rigid in children with kyphoscoliosis; but its distensibility was reported to be decreased in adult patients (Ferris, Mead,
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Whittenberger, and Saxton, 1952a; Bergofsky et al., 1959), and this finding was also evident in the single elderly kyphotic we studied. It appears that rigidity of the chest cage is a late complication of a disease in which other forms of impairment of breathing can be demonstrated during youth.

Surprisingly the static pressures developed by the muscles of respiration at resting lung volume were roughly normal; yet their effectiveness in expanding the lungs, as measured by the vital capacity and total lung capacity, was reduced. In the absence of a stiff chest cage, other causes must be considered for this reduction in lung volumes. These are as follows:

In patients with diaphragmatic paralysis or paradoxical motion of the thorax, the thoracic cage was insufficiently firm to expand uniformly. In other poliomyelitic and in non-paralytic patients the mechanical advantage of the respiratory muscles may have been decreased by distortion of the normal relationship of their points of skeletal attachment. Finally, the compliance of the lung was reduced in the majority of our patients. This latter change was present regardless of age, the aetiology of the spinal curvature, or of presence or absence, by history, of pulmonary infection.

The functional residual capacity and total lung capacity were decreased in almost every patient. When these changes in lung volume were experimentally simulated in normal man by tightly strapping the chest, a fall of lung compliance resulted (Caro et al., 1960). There was evidence that the altered pressure-volume relationship of the lung was due to occlusion of some parts to ventilation (probably terminal lung units) (Caro, 1959). We believe that we have thus experimentally simulated the functional changes in the lung in kyphoscoliosis.

It follows from our experimental findings that the aim of orthopaedic treatment of kyphoscoliosis should be to correct the spinal curvature without further impeding the expansion of the chest cage. Long-term restriction of chest expansion, as in a tight body plaster jacket, might lead to permanently occluding additional regions of the lung to ventilation, or to changes in the mobility of the chest wall.

It is desirable to correct or prevent the collapse of the spine, or slump, which compresses the rib cage, limits the motion of the diaphragm and abdomen, and probably results in progressive stiffening of the joints and ligaments of the chest wall with age. Effective methods should be sought to maintain and develop expansion of the lungs and chest during childhood and adolescence, and during and after the period of orthopaedic therapy.

**Summary**

In young and adult patients with kyphoscoliosis the lung volumes were small and the lung compliance was reduced, but there was no airway obstruction. However, the thoracic cage was normally distensible in younger patients, but rigid in older ones with spinal curvature, and it appeared that chest cage rigidity was a late complication of the disease. Tightly strapping the chest of normal man, which causes parts of the lung to become occluded to ventilation, reproduced the pulmonary changes found in kyphoscoliosis.

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