Growth of lung and thorax dimensions during the pubertal growth spurt

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ABSTRACT: In the follow-up study of adolescents residual volume (RV), vital capacity (VC), closing volume (CV), thorax height and thorax width were measured at six month intervals over a six year period; 149 girls and 477 boys, whose ages ranged between 11.5 and 18.5 yr during the follow-up, were investigated. In healthy subjects and in those with respiratory symptoms the rates of change of thoracic dimensions, as calculated by a robust linear regression technique, were moderately well correlated; the growth rates of RV and VC were only slightly correlated, and both were moderately well correlated with changes in thorax dimensions. Thorax height increased relative to thorax width in boys and girls during the follow-up. In boys, thorax height continued to grow, when standing height and thorax width had attained adult values in girls. Thus the thorax attains a more elongated shape. During the adolescent growth spurt the residual volume as a percentage of total lung capacity (RV%TLC) within subjects increased on average 0.6% per yr, and closing volume as a percentage of vital capacity (CV%VC) decreased on average 0.5% per yr. Both of these changes can be accounted for by decreased thorax compliance and increased outward chest recoil at low lung volumes.

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During some period between the age of 10 and 15 yr all children undergo a marked acceleration in their growth rate. This growth spurt affects the long bones first, preceding the time of peak growth velocity of thorax width by about six months and that of thorax height by about a year [21, 24]; Consequently thoracic proportions change continuously during growth. Since the lung is contained in the thorax it seems plausible that the asynchronous changes in thoracic dimensions will have bearing on the development of lung volumes. Also changes which occur in the maximum pressures which respiratory muscles are capable of exerting on the chest wall and lung [2, 4, 9] will affect lung volumes during growth.

In this paper we describe the changes which occur in thoracic dimensions and in subdivisions of the total lung capacity in adolescents aged 11.5 to 18.5 yr. In addition we analyse how and to what extent the growth of the lung is related to that of the thorax.

Lung growth was studied using the vital capacity (VC) and residual volume (RV), measured longitudinally. The growth of the thorax was studied on the basis of measurements of thorax height (ThH) and thorax width (ThW).

Materials and methods

The data were collected in a follow-up study of adolescents, which started in 1978 and ended in 1985. Each subject was measured twice a year, resulting in, at the most, twelve measurements over a six yr period.

We applied the single breath nitrogen test as described by Buist and Ross [1] and Sterk et al. [23]. This test provides the inspiratory and expiratory vital capacities, of which we used the largest as vital capacity. After practice the seated subject was connected to a bag-in-box system at the level of residual volume. Oxygen was inhaled until the level of total lung capacity (TLC) was reached. Without respiratory pause, the subject then performed a slow, full expiration. The single breath manoeuvre was extended by about twelve forced rebreathings, at a rate of about 30 breaths per minute, from which we obtained residual volume [23]. The nitrogen concentration was measured at the mouth with a linear nitrogen analyzer (Hewlett Packard, type 47302 A); the volume signal was obtained from a water-sealed spirometer (Lode D53R). These signals were sampled and processed on line for the computation of both vital capacity and residual volume. Closing volume (CV) was calculated with an algorithm similar to the one used by Hankinson [6]. Measurements and methods have been extensively described in a previous paper [3]. Readings were only accepted if the inspiratory vital capacity was within 90–105% of the expiratory vital capacity.

Thoracic height, width and sternal height were measured with a Holtain anthropometer with the subject standing, at the end of normal expiration.

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Thoracic height was defined as the vertical distance between the middle of the clavicle and the lower border of the rib cage; the mean of the measurements of left and right hemithorax was used. Similarly, the mean of two measurements of thoracic width at the level of the lower sternal border and of sternal height were used. In the majority of subjects sternal height increased not only by appositional growth, but also because the xypoid process is added to it: it goes through a stage of a non-palpable fibrous plate to a firmly ossified bone. Hence this index is not useful, since a systematical error is introduced in the longitudinal measurements. In addition, standing height and weight were measured. All measurements were read to the nearest mm or 0.01 kg.

A questionnaire about the respiratory health status of the pupils was filled in by the parents on each occasion. At the first survey the questionnaire dealt with the period preceding the study; information covering the elapsed period was obtained at each subsequent investigation. In addition, a confidential, brief form on their smoking habits was filled in by the pupils themselves on each occasion.

Subjects

Caucasian subjects at two different schools in The Hague participated in the study. The age range over the whole follow-up period was 11.5–18.5 yr. At a secondary school, 198 boys and 149 girls, who entered the lowest class either in September 1978 or in September 1979, were participants during the whole of their school career. At a technical school, another 279 boys were measured for the first time when they entered the lowest class in September 1980 or in September 1981. The following groups were formed: non-smokers who had never smoked (NS); smokers (S), regardless of the respiratory history; and subjects having a negative (NH) or a positive history (PH) neglecting smoking habits. Boys and girls were analysed separately. A subject was considered to be a smoker from the time he/she reported smoking at least eight cigarettes a week. Subjects were considered to have a positive respiratory history whenever there were recent or past symptoms of cough or phlegm for more than three months per yr, or in case of shortness of breath, or complaints of wheezing, asthma or bronchitis. The subgroups comprised only pupils of whom at least six sets of measurements were available. A preliminary analysis of covariance did not show an effect of smoking on level and growth of the lung volumes. We therefore neglect smoking habits in the following analyses, i.e. only the results for groups of subjects having a positive history of respiratory symptoms (PH) and of subjects having a negative history (NH) will be described.

Analyses

We studied proportional growth of lung volumes from the residual volume as a percentage of vital capacity (RV%VC), rather than the RV%TLC. In the latter, RV forms part of both numerator and denominator, which obscures a possible change with growth in the different volumes. The proportional change of thorax dimensions was studied from the thorax width as a percentage of thorax height (ThW%ThH).

In order to describe changes in lung volumes and thoracic dimensions during the growth spurt we calculated the mean annual growth rates of RV, VC, RV%VC, ThW, ThH and ThW%ThH in each subject. During the growth spurt these indices increase more or less linearly with age. The inclusion of data points prior to and after the growth spurt leads to S-shaped curves; we wanted to exclude such points as well as obvious outliers. This could be achieved by applying a robust linear regression method [18], because such a technique tends to ignore points which do not behave like the majority of the data. We chose a method similar to least median squares, but instead of minimizing the median of the squared residuals we chose the 75% quantile of the points as well as obvious outliers. This could be achieved by applying a robust linear regression method [18], because such a technique tends to ignore points which do not behave like the majority of the data. We chose a method similar to least median squares, but instead of minimizing the median of the squared residuals we chose the 75% quantile of the distribution of the residuals as the object function. An example illustrating how inclusion or exclusion of ‘outliers’ affects the estimated annual rate of change, is given in figure 1. It appeared (table I) that there

\[
\begin{array}{l|ll|ll}
\text{Age(years)} & 6 & 12 & 18 & 0.8 & 1.0 \\
\hline
\text{Boys} & 72.4 & 72.5 & 71.6 & 73.8 \\
\text{Girls} & 15.4 & 9.9 & 5.9 & 4.8 \\
\end{array}
\]

**Table I.** Distribution of outliers at the start and end of individual growth curves of RV, VC, ThH and ThW as detected by robust regression analysis. NH and PH: negative and positive history of respiratory symptoms respectively.
were more boys than girls with a growth spurt that had not started at the first measurements, and conversely more girls than boys had finished their growth spurt before the end of the survey. This is as expected on the basis of different age of onset and duration of the growth spurt in adolescents [24]. On average, less than one outlying data point was detected among 6–12 (mean 9) available data points for each individual.

We analysed in which way and to what extent growth rates of lung volumes within subjects are related to those of the thorax dimensions for each subgroup. As a first step Pearson correlation coefficients were calculated between individual growth rates of lung volumes and thorax dimensions. Simultaneously, the relationships between the sets of growth rates of the lung indices on the one hand and those of the thorax dimensions on the other were analysed by classical canonical correlation analysis [8, 13].

Results

Figure 2 shows the mean individual growth rates of VC, RV, RV%VC, ThH, ThW and ThW%ThH calculated by robust regression analysis. For each index, the change with age is depicted by a line drawn from the mean age at the first measurements for the various groups of subjects (as allowed by robust regression) up to the mean age of the last measurements. Table II lists the mean growth rates corresponding to figure 2. Except for ThW in girls, all mean growth rates differ from 0 (p < 0.05). The growth rates of RV%VC and of ThW%ThH of the different subgroups do not differ significantly. The change of RV%VC amounts to about 1% per yr, that of ThW%ThH to about -1.25% per year. Hence residual volume grows relatively faster than vital capacity, and thoracic height grows relatively faster than does thoracic width. The group levels of ThW%ThH, estimated at an overall mean age of 15.3 yr, do not differ significantly for boys or girls, whereas the levels of RV%VC do. The growth rates of VC, RV, ThH and ThW in boys are at least twice those in girls. Figure 2 shows that in girls thorax width did not increase during the follow-up, whilst thorax height clearly increased. Therefore, the decrease in ThW%ThH with age (table II) will be due mainly to the growth in thoracic height. In boys, thoracic height grows twice as fast as thoracic width. In boys and girls,

<table>
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<th>Sex</th>
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<th>n</th>
<th>ΔVC</th>
<th>ΔRV</th>
<th>ΔThW</th>
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<th>ΔThW/ThH</th>
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<td></td>
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<td>ml/yr</td>
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<td>cm/yr</td>
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<td></td>
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<td>(5)</td>
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<td>(0.042)</td>
<td>(0.205)</td>
<td>(0.139)</td>
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<td></td>
<td></td>
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<td>(18)</td>
<td>(7)</td>
<td>(0.067)</td>
<td>(0.078)</td>
<td>(0.356)</td>
<td>(0.253)</td>
</tr>
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<td>(9)</td>
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<td>(11)</td>
<td>(7)</td>
<td>(0.035)</td>
<td>(0.047)</td>
<td>(0.177)</td>
<td>(0.142)</td>
</tr>
</tbody>
</table>
GROWTH OF THORAX AND LUNG

Table III.- Fit of the complete set of thorax growth indices (%6RV, %6VC and %6RV%6VC) expressed as the sum of the squared canonical correlations x 100, and percentage variance of the separate indices explained by those of the other set, in boys (M) and girls (F) with negative (NH) and positive (PH) histories of respiratory symptoms.

<table>
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<tr>
<th></th>
<th>overall &quot;fit&quot;</th>
<th>percentage variance explained by thorax growth indices</th>
<th>percentage variance explained by lung growth indices</th>
</tr>
</thead>
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<td>group</td>
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<td>PH</td>
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<tr>
<td>131</td>
<td>M</td>
<td>PH</td>
<td>51.5</td>
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the absolute rate of change of vital capacity exceeds that of residual volume by a factor of two or more. However, in a relative sense, the residual volume grows faster than the vital capacity, as indicated by the increasing RV%VC.

Studying the relationship between lung growth indices and those of thoracic growth, with canonical correlation analysis, was done in two steps. The first one included the growth rates %6RV%6VC and %6ThW%6ThH, whereas the second one did not. Table III summarizes the results of the first approach. The ‘fit’ of the sets, the percentage variance of each lung growth index explained by the total set of thoracic dimensions and the percentage variance of each thoracic index explained by the total set of lung volume parameters is shown. %6VC is most strongly related to the growth of the thorax dimensions, whereas %6ThH is more closely related to the growth of the lung volumes than %6ThW, especially in boys. It is obvious that in the multivariate analysis, in which the sets of lung and thorax growth rates are optimally related, compound indices like %6RV%6VC and %6ThW%6ThH will add little to the description of the relationship between the sets as shown in table III. Consequently the interpretation is simplified and little is lost by leaving out %6RV%6VC and %6ThW%6ThH. This was done in the second canonical correlation analysis. This analysis has the advantage that in one diagram the relationship of %6ThW and %6ThH can be inspected directly with respect to the total variance of the %6RV and %6VC, depicting at the same time the relationship between %6VC and %6RV.

Figure 3 shows the relationship of %6RV and %6VC and of %6ThW and %6ThH with respect to the pattern of variation of %6RV and %6VC, for boys and for girls with positive and with negative histories of respiratory symptoms. The magnitude of the vectors for %6RV and %6VC is 1, which means that the total variance of %6RV and %6VC is present in the two-dimensional diagram. The cosine of the angle between the vectors %6RV and %6VC is equal to the correlation between the two. The explained variance of %6ThW or %6ThH in the diagrams equals the square of their vector magnitude and is less than 1. Each squared magnitude is what is explained of the variance of
AThW or AThH by the growth rates of RV and VC together. What the growth rates of ThH and of ThW explain separately of the growth rates of RV and VC can be found by projecting the vectors of the former on the latter. These projections are equal to their respective bivariate (Pearson) correlations and are equal to those in table IV. Note that the correlations of the growth rates of ThW and ThH cannot be reconstructed from the diagram as only a fraction of their respective variances is present in it (namely that part which is relevant with respect to the growth rates of RV and VC).

In all groups the near perpendicularity of the vectors of ARV and AVC shows that RV and VC develop relatively independently; their rates of change are only clearly correlated in males with a negative history of respiratory symptoms (r = 0.30; table IV). Figure 3 also shows differences between boys and girls. In boys, unlike girls, AThW and AThH point more in the direction of AVC than of ARV. Thus, in boys the individual growth rates of residual volume are less related to the growth rates of the thorax than is the case with the growth rates of vital capacity. In girls the thoracic vectors are between those of ARV and AVC: the relationship of AVC with AThH is seen to be weaker than in boys. With the exception of girls with a positive history the AThH vectors are larger than the AThW vectors and more closely related to both ARV and AVC than AThW. As before (table III) in girls with a positive history of respiratory symptoms the canonical correlations are higher than in girls with a negative history: it seems that the development of the trunk is less independent than that of the lung in girls with a positive history. In both boys and girls with a positive history the ARV vector turns away from the AVC vector as compared with subjects with a negative history.

### Discussion

In general our results show that during the pubertal growth spurt the thorax changes not only in size but also in shape, and that this is accompanied by alterations in size and subdivisions of the total lung capacity. Differences between subjects with and without respiratory symptoms are as a rule negligible. This is probably due to the fact that most of the subjects classified as having symptoms had these symptoms in the past; the others had one or two symptoms during the study, which in the majority of these subjects were present only on a limited number of occasions during the follow-up. Hence there was only minimal disease in the group classified as having a positive history. All respiratory symptoms occurred more frequently in boys than in girls with a positive history, yet the relationship between growth of lung and thorax was virtually the same in boys (figs 2 and 3). Therefore in the present study the moderate extent of past and present airway disease seems to have little effect on the growth of lung and thorax.

On average ThW%ThH decreases with age in both boys and girls (fig. 2). This is only partly due to the fact that in general the growth of trunk height leads that of chest width by about six months [24]. In boys thorax height increases about twice as fast as thorax width (fig. 2), which leads to a constant decline of ThW%ThH with age. In most of the girls thorax width, unlike thorax height, did not increase at all. The finding of a relative elongation of the thorax agrees very well with that of Simon et al. [21], who determined lung width and lung length radiologically (at the level of TLC) between ages 5–19 yr; they found that the level of the diaphragm relative to the vertebrae fell progressively. This was not confirmed by Openshaw et al. [17], who found that after the age of ten yr the diaphragm stays at about the same level at full lung inflation; however, they did find increased downward angulation of the ribs, compatible with the relative chest elongation which we observed.

The changes in the thoracic dimensions are rather loosely related to those in RV and VC. Inclusion of measurements of the antero-posterior chest dimensions might have changed the picture a little, but the correlation between the growth of lung and thorax will also be influenced by the fact that the measurements of thorax width and height were performed at resting level, unlike those of residual volume and vital capacity which entailed maximum tidal excursions. Sternal height is not influenced by the level of lung inflation, but in many subjects there was spurious growth due to the xyphoid process becoming palpable only during the course of the study, so that it became

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sex</th>
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<th>ARV</th>
<th>AVC</th>
<th>AThW</th>
</tr>
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<tr>
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<td>M</td>
<td>PH</td>
<td>0.28</td>
<td>0.54</td>
<td>0.39</td>
</tr>
</tbody>
</table>
useless for our analyses. Cross-sectionally FRC% TLC is constant during growth according to most authors [5, 7, 14-16, 22, 25], minimal age- and height-dependence being described in only two reports [26, 27]. Hence, resting thoracic measures are related to both FRC and TLC, as are their growth rates, and attention should focus on the determinants of RV.

From figure 3 we see that in healthy subjects the growth rate of residual volume is largely independent of the development of the vital capacity. The average RV%VC increases during this age period, with wide dispersion between subjects, indicating that these volumes do not increase in proportion. Also, the average level of RV%VC at age 15.3 is higher in girls than in boys (fig. 2). There are various explanations for this, such as an alteration in the balance of static muscle and recoil forces. In adolescents the static force exerted by the inspiratory muscles increases in proportion with lung surface area and curvature of thorax and diaphragm. This is evidenced by either no, or only slight age dependence, on maximum inspiratory pressures at the level of TLC and RV [2, 4, 9, 19]. Measurements of the compliance of the respiratory system are very scarce in adolescents. Sharp et al. [20] made measurements in anaesthetized subjects and found that the shape of the pressure/volume curve of the respiratory system remained the same, with only very minor differences in the position of the volume standardized curves between 10–18 yr of age. Hence there is no evidence suggesting a change of the specific compliance curve of the respiratory system in this age range at the cost of VC in relation to RV. It is therefore reasonable to assume that on average in growing adolescents at TLC, an effort dependent lung volume, the respiratory system is inflated to comparable levels.

Unlike inspiratory pressures, the maximum expiratory pressures generated at the level of TLC and FRC increase [2, 4, 9, 19]. On that account, in the presence of an unaltered compliance curve of the respiratory system, the VC should on average increase relatively more than RV, which is contrary to what we found. The most likely explanation for our findings is that increased outward recoil pressure of the chest wall at the level of residual volume, as suggested by Leith and Mead [10], must outweigh the increased transrespiratory pressure generated by the respiratory muscles. This provides an explanation for the fact that, in spite of unaltered volume-standardized P-V characteristics of the lungs between 10–18 yr [12], closing volume (CV) as a percentage of the VC drops. When we pool all data on CV%VC and regard these as a cross-sectional data set CV%VC decreases on average 0.3% per yr in healthy boys and girls. Within individuals the longitudinal decrease is 0.5% per yr (fig. 4). This is less than in the study of Mansell et al. [11], who reported an annual fall of 1.25%. Their data on CV%VC agree better than ours with the observed increase in RV%VC; this may be because they used a large air bolus to obtain more pronounced phase IV and thus obtained larger closing volumes, particularly in young subjects. Clearly the increase in chest recoil at low lung volumes, which starts soon after birth, suffices to explain both the greater relative change of RV over VC and the drop in CV%VC. In principle, if the heart and mediastinum lag behind in growth or grow proportionally less, this might affect the relationship between RV and VC. If such extrapulmonary tissues were to affect the RV only, they would have to account for 250 ml in the present study, and more if this disproportional tissue growth were also to affect the VC. This possibility can be discarded, as this volume approximates the adult heart size.

In conclusion, our data show that in adolescents neither the subdivisions of total lung capacity, nor chest dimensions, grow strictly in proportion; the growth rates of lung volumes and chest width and height are also rather loosely correlated. The net result is that between 11.5 and 18.5 yr the thorax becomes relatively elongated; also the RV%VC increases on average by about 1% per yr, i.e. about 0.6% per yr for the more commonly used RV%TLC. This apparent age dependence is probably due to decreased compliance and increased outward recoil of the chest at low lung volumes, which is not balanced by an increase in expiratory pressures generated by the respiratory muscles. This limitation of lung deflation by the chest wall can also account for the decrease in CV%VC.

References
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