Chest Deformity, Residual Airways Obstruction and Hyperinflation, and Growth in Children with Asthma

II: Significance of Chronic Chest Deformity

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Gillam, G. L., McNicol, K. N., and Williams, H. E. (1970). *Archives of Disease in Childhood*, 45, 789. **Chest deformity, residual airways obstruction and hyperinflation, and growth in children with asthma. Part II: Significance of chronic chest deformity.** Studies were made on 56 10-11-year-old asthmatic children with onset of symptoms before 3 years of age, frequent and persistent attacks, and objective evidence of their disease with barrel chest deformity and/or reduction of the FEV_{0.5}/VC ratio to 50% or less, by clinical, physiological, and radiological techniques in an interval phase between their attacks.

There was a significant correlation between barrel chest deformity and (a) elevation of lung volumes (FRC, RV, TLC, FRC/TLC, and RV/TLC), and (b) reduction of the FEV_{1.0}/VC ratio, indicating chronic hyperinflation and airways obstruction in the interval phase between attacks.

Radiological evidence of hyperinflation also correlated significantly with barrel chest deformity, increased lung volumes, and airways obstruction. These parameters were found to be important criteria for assessing chronicity in asthma.

The group showed evidence of growth retardation particularly for weight and to a lesser extent for height, these changes being most marked in subjects with barrel chest deformity. Corticosteroid therapy was not a significant factor in the growth retardation observed.

There was a preponderance of males over females in the group (7:3).

The preceding paper (McNicol, Williams, and Gillam, 1970) discussed the prevalence of chest deformity, residual airways obstruction and hyperinflation, and growth patterns, in a randomly selected group of asthmatic children.

It was shown that only 10% of children who had had less than 20 episodes of asthma and no episodes within 12 months of examination at 10+ years (Grades I and II) had either chest deformity, rhonchi, airways obstruction, or hyperinflation, or a combination of these findings when examined in an interval phase between attacks.

In those children who had had over 20 episodes of asthma and were continuing to have attacks at the

10 years + examination (Grade III) almost 50% showed one or more of these findings. However, the presence of rhonchi in an interval phase accounted for almost half the findings in this latter group. Chest deformity occurred infrequently in the children studied (3%). Most of the children with chest deformity were in Grade III.

It was shown that there was no significant difference in the growth patterns of any of the groups studied. However in Grade III there was a trend towards reduction in weight but this did not reach statistical significance.

To elucidate the nature and relationships of chest deformity, airways obstruction and pulmonary hyperinflation, and growth impairment it was necessary to sample a much larger population of asthmatic children in order to obtain a sufficient

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number of severely asthmatic children for meaningful analysis.

The aim of the present report is (1) to relate chest deformity to changes in lung function as assessed by measurements of airways obstruction and lung volumes, (2) to relate radiological evidence of hyperinflation to changes in lung function and, (3) to relate chest deformity to changes in growth.

Criteria for Selection

The subjects selected were 10 to 11-year-old schoolchildren who fulfilled all the following criteria. (a) Onset of symptoms before 3 years of age, (b) persistent symptoms with either, (i) at least 10 attacks within the last 2 years or, (ii) periods of persistent wheezing during this time; and (c) barrel chest deformity and/or reduction of the $FEV_{0.5}/VC$ ratio to 50% or less.

Selection procedure. Approximately 85% of 5th grade schoolchildren (generally 10–11 years of age) in Melbourne, Australia, are examined annually by the Victorian School Medical Service. In 1968, in conjunction with this annual examination, questionnaires relating to a history of wheezing were distributed to the parents of the children. The questionnaires specifically asked whether the child had ever wheezed or had asthma and also the frequency of such episodes and their persistence.

All children satisfying the criteria of early onset (a) and persistent symptoms (b) were seen at the school with the mother or guardian (by K.McN.), where a more detailed history was obtained, the child examined, and spirometry using a 'Vitalor' Dry Spirometer carried out. Any child with barrel chest deformity and/or reduction of the $FEV_{0.5}/VC$ ratio to 50% or less was then assessed in more detail at the hospital generally some weeks after the initial school visit. Approximately 1 in 3 of the subjects seen at the schools met the above criteria and were completely evaluated at the hospital.

Table I shows the total population sampled, and the number seen at the schools meeting the above 3 criteria (approximately 1:200 of the population sampled). Of the 83 subjects presenting for study, 21 subjects were seen in the early phase of the study before pulmonary function methods were established. In fact these subjects did not differ clinically in any significant way

from the group studied. Of the 62 subjects studied in full, 6 were having an acute attack when studied, leaving 56 subjects considered to be in an interval phase between attacks. If the child complained of any tightness in the chest, if the accompanying parent felt the child was still recovering from a recent asthmatic attack, or if an audible wheeze could be heard, the child for the purpose of the study was defined as having an acute attack.

Only the 56 interval phase asthmatics will be considered in the results. For the purposes of analysis they have been divided into four groups on the basis of the degree of barrel chest deformity present, i.e. nil, mild, moderate, and severe barrel chest deformity.

Method

The assessment of chest deformity was made by G.L.G. The radiological assessment was carried out as previously described (McNicol *et al.*, 1970).

Fig. 1 shows a subject with severe barrel chest deformity, some pigeon chest deformity, and Harrison's sulcus. The skin shows eczematous changes, and the child is underweight for her age and height.

Fig. 2 is the chest x-ray of the same child showing severe hyperinflation.

Even with experienced observers there is considerable variation in the interpretation of chest signs (Smyllie, Blendis, and Armitage, 1965; Godfrey, *et al.*, 1969). For this reason one of us (K.McN.) made an independent assessment of the degree of chest deformity present. This observer was studying the same group of children from different aspects.

Vital capacity (VC) and forced expiratory volume at one second ($FEV_{1.0}$) were measured using a 9-litre Godart Expirograph. Three technically satisfactory estimations were performed and the maximum value was recorded. The ratio of $FEV_{1.0}/VC$ was calculated.

Functional Residual capacity (FRC) was determined using the closed circuit helium dilution technique, oxygen being added to the circuit continuously. Carbon dioxide was absorbed using a soda-lime canister in the circuit. The end point was taken when the helium concentration had remained constant for approximately 3 minutes. Helium equilibration times varied from 2 to 3 minutes in subjects with minor changes, and up to 6 or 7 minutes in those with more marked changes. Inspiratory capacity (IC) and expiratory reserve volume (ERV) were read directly from the spirometer tracing. Three estimations of IC were done and the average of the two maximum readings calculated. The average of two ERV estimations was taken.

From the above results, total lung capacity (TLC) and residual volume (RV) and the ratios of FRC and RV to TLC were calculated.

Duplicate determinations of lung volumes were carried out and the mean value used in the results. All results were expressed at BTPS.

The procedure was explained to the subjects before the determination, and they were allowed to accustom themselves to the mouth-piece and nose clip. Excellent co-operation was obtained in almost all subjects.

A control group of 36 normal children matched for

TABLE I

Total Population Sampled and Number of Asthmatics Studied in Interval Phase Between Attacks

Source	No. of Subjects
Population sampled	21,000
No. selected	96
No. presenting for study	83
Lung function studies	62
Acute attack	6
Interval phase	56

FIG. 1.—Subject with severe barrel chest deformity, pigeon chest deformity, and Harrison's sulcus. Postero-anterior view (a) and lateral view (b). The skin shows eczematous changes.

age, sex, and socio-economic status was obtained from the same school population as the asthmatic children and studied in the same way.

Statistical Method. For statistical purposes the asthmatic subjects with moderate and severe barrel chest deformity were considered as one group because of the small number of subjects in these two groups. This resulted in four groups: (1) the control population, (2) asthmatics with no barrel chest deformity, (3) asthmatics with mild chest deformity, (4) a combined moderate and severe group. The mean and standard deviation for data from each group was calculated, and an analysis of variance within groups and between groups was made. A probability of less than 0.05 was considered significant.

Results

Table II shows the four clinical gradings of barrel chest deformity with the number of subjects in each group. Table III shows the three radiological gradings of hyperinflation with the number of subjects in each group. Table IV shows the correlation between the clinical grading of barrel chest deformity and radiological evidence of hyperinfla-

tion. A good correlation is apparent. However 50% of the subjects with no barrel chest deformity had radiological evidence of hyperinflation.

The results are shown in graphical form in Fig. 3 to 14, and the results of statistical analysis in Table V. The code used for the four gradings of barrel chest deformity is shown in Fig. 3.

Chest deformity and lung function studies.

(a) *Lung volumes.* Fig. 3 to 7 show that the asthmatic group over-all had an increase in FRC,

TABLE II
Clinical Grading of Barrel Chest Deformity Showing Number of Subjects per group

Grading of Barrel Chest Deformity						No. of Subjects
Nil ..	..	..	..	..	..	26
Mild ..	..	..	..	..	..	18
Moderate	..	..	..	..	..	8
Severe	..	..	..	..	..	4
Total ..	..	..	..	..	..	56

FIG. 2.—Chest x-ray of subject in Fig. 1 showing severe hyperinflation. Postero-anterior view (a) and lateral view (b).

FIG. 3.—Functional residual capacity plotted against height. Black lines represent 2 SD above and below the mean for the control population. Code for the four clinical gradings of barrel chest deformity in Fig. 3–14. ○ no deformity, ● mild deformity; ▲ moderate deformity; and ■ severe deformity.

TABLE III
Radiological Grading of Hyperinflation Showing Number of Subjects per Group

Radiological Grading of Hyperinflation						No. of Subjects
Nil	..	..	..	..	..	16
Hyperinflation	..	..	..	..	..	42
Severe hyperinflation	..	..	..	..	..	8
Total	..	..	..	..	..	56

TABLE IV
Correlation Between Clinical Grading of Barrel Chest Deformity and Radiological Evidence of Hyperinflation

Clinical Grading		Radiological Grading		
		Nil	Hyperinflation	Severe Hyperinflation
Nil (26)		13	13	—
Mild (18)		2	14	2
Moderate (8)		1	4	3
Severe (4)		—	1	3
Total		16	32	8

FIG. 4.—Residual volume plotted against height. Black lines represent 2 SD above and below the mean for the control population.

FIG. 5.—Total lung capacity plotted against height. Black lines represent 2 SD above and below the mean for the control population.

FIG. 6.—FRC/TLC ratio plotted against height. Black lines represent 2 SD above and below the mean for the control population.

FIG. 7.—RV/TLC ratio plotted against height. Black lines 2 SD above and below the mean for the control population.

TABLE V
Statistical Analysis of Data from Control Group and 3 Groups of Asthmatic Children*

	Control Group (36)		Nil (26)		Mild (18)		Mod. and Severe (12)		F Value	P Value
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
Age (mth.)	130.7	6.7	128.6	7.0	130.9	6.3	130.9	5.0	0.67	NS
Height (cm.)	141.2	6.2	138.2	7.3	137.6	5.1	135.3	6.2	3.22	<0.05
Weight (kg.)	37.8	6.4	33.2	4.7	29.9	3.8	28.9	4.3	13.90	<0.001
FRC (ml.)	1508	299	1644	307	1929	289	2189	396	16.99	<0.001
RV (ml.)	685	163	817	251	1029	309	1336	405	21.60	<0.001
VC (ml.)	2559	337	2462	444	2397	273	2235	419	5.85	<0.001
TLC (ml.)	3304	432	3316	566	3560	412	3693	484	44.50	<0.001
FRC:TLC (%)	45.4	6.3	49.0	4.5	54.1	3.7	59.3	7.4	24.60	<0.001
RV:TLC (%)	20.3	3.4	23.5	4.9	28.5	6.3	36.5	10.7	48.50	<0.001
FEV _{1.0} :VC (%)	87.3	4.8	71.9	11.0	65.5	9.7	57.3	14.2	42.7	<0.001

* Showing means and standard deviations (SD) for age, height, weight, and various parameters of lung function. Asthmatic children are graded into those with nil, mild, and a combined moderate plus severe barrel chest deformity.

RV, TLC, FRC/TLC, and RV/TLC ratio, and that these changes were more obvious in subjects with barrel chest deformity. Table V shows there was good correlation between increased lung volumes and increasing grades of barrel chest deformity. Values falling outside the two lines shown on each of these figures are more than 2 SD from the control mean.

(b) *Spirometry.* Fig. 8 shows that all except two subjects had a normal vital capacity. Table V shows a slight but significant reduction in VC with increasing grades of barrel chest deformity.

Fig. 9 shows that the asthmatic group had a considerable reduction in the FEV_{1.0}/VC ratio. Only one subject was above the mean, and the majority especially those with barrel chest deformity were considerably reduced.

FIG. 8.—Vital capacity plotted against height. Black lines represent 2 SD above and below the mean for the control population.

FIG. 9.—FEV_{1.0}/VC ratio plotted against height. The black line represents the mean and the dotted line 2 SD below the mean for the control population.

Fig. 10 relates the $FEV_{1.0}/VC$ ratio against the RV/TLC ratio, and shows a good correlation between the two ($r = -0.69$, $p < 0.001$).

Radiological findings and lung function studies. Fig. 11 shows that subjects with radiological evidence of hyperinflation had a higher RV/TLC ratio than those without hyperinflation. The good correlation between chest deformity and radiological evidence of hyperinflation is also shown. Those subjects with chest deformity in the second and third columns of the figure with an RV/TLC ratio above 27% show clinical, physiological, and radiological evidence of hyperinflation.

Fig. 12 shows that radiological evidence of hyperinflation was associated with the more pronounced reductions in the $FEV_{1.0}/VC$ ratio.

Growth. Fig. 13 shows that only those subjects with the more severe grades of barrel chest deformity had a reduction in body height though Table V shows a significant reduction in body height and increasing chest deformity.

FIG. 10.— $FEV_{1.0}/VC$ ratio plotted against the RV/TLC ratio. The horizontal dotted line represents 2 SD below the mean for $FEV_{1.0}/VC$ ratio. The vertical dotted line represents 2 SD above the mean for RV/TLC ratio. The regression line is shown $r = -0.69$.

FIG. 11.— RV/TLC ratio plotted against the radiological grading of hyperinflation. Nil = no radiological evidence of hyperinflation. HI, radiological evidence of hyperinflation. SHI, severe hyperinflation radiologically. The horizontal dotted lines represent 2 SD above and below the mean for the control population.

FIG. 12.— $FEV_{1.0}/VC$ ratio plotted against the radiological grading of hyperinflation. Nil, no radiological evidence of hyperinflation. HI, radiological evidence of hyperinflation. SHI, severe hyperinflation radiologically. Continuous line represents the mean and the dotted line 2 SD below the mean for the control population.

FIG. 13.—Body height plotted against age. 10th and 90th centiles for a control population are shown.

Fig. 14 shows the much more striking reduction in weight with increasing grades of barrel chest deformity. This is further analysed in Table V.

Table VI shows the assessment of barrel chest deformity by G.L.G. and K.McN. While there was good agreement in the assessment of the moderate and severe grades of deformity, there was considerable variation in assessment of mild grades of deformity. Of the 12 subjects graded by G.L.G. as having moderate or severe barrel chest deformity, K.McN. agreed that 10 of these subjects had barrel chest deformity but graded 3 as mild. Of the

FIG. 14.—Body weight plotted against age. 10th and 90th centiles for a control population are shown.

8 subjects graded by K.McN as having moderate or severe deformity G.L.G. agreed that all had barrel chest deformity but rated 1 as mild.

Discussion

The results show that barrel chest deformity was associated with physiological evidence of hyperinflation with an increase of FRC, RV, TLC, and FRC/TLC and RV/TLC ratios, and with airways obstruction with reduction in the $FEV_{1.0}/VC$ ratio. These changes were most obvious in those subjects with the more severe grades of barrel chest deformity. All these measurements were made in an interval phase when the child was not having an attack of asthma.

Radiological evidence of hyperinflation was also associated with hyperinflation as measured by lung volumes, and airways obstruction as measured by the $FEV_{1.0}/VC$ ratio.

There was good correlation between barrel chest deformity, radiological evidence of hyperinflation, and physiological evidence of hyperinflation. However 50% of subjects with no clinical evidence of barrel chest deformity had radiological evidence of hyperinflation, suggesting that radiological examination may be a more sensitive method of detecting lesser degrees of pulmonary hyperinflation.

TABLE VI

Assessment of Barrel Chest Deformity by Two Independent Observers

		Grading by G.L.G.				
		Nil	Mild	Moderate	Severe	
Grading by K.McN.	Nil	17	—	2	—	19
	Mild	9	17	3	—	29
	Moderate	—	1	3	1	5
	Severe	—	—	—	3	3
		26	18	8	4	
		Totals (G.L.G.)				

The results of the present work strongly suggest that barrel chest deformity is a consequence of persisting hyperinflation and airways obstruction, and it therefore follows that an asthmatic child with such deformity in an interval phase is suffering from chronic asthma. Probably barrel chest deformity represents the extreme end of the range of chronic pulmonary hyperinflation.

The good correlation between pulmonary hyperinflation and airways obstruction in the present study differs from the findings of Engstrom and Karlberg (1962) and Engstrom (1964) who found a good correlation between the two in the acute attack but not in the asymptomatic phase. These workers showed that after an acute attack, airways resistance was the first measurement to return to normal, followed by improvement in ventilatory capacity and lastly in lung volumes. Lung volumes in some individuals remained persistently high. Severity of asthma in these studies was based solely on the frequency of attacks, no other clinical details being given. For this reason it is not possible to say whether the clinical status of the subjects in these studies was or was not comparable to that of the present group. It is possible that the difference in the physiological findings may be explained by differences in severity of asthma in the two study groups.

Chest wall changes could of course contribute to the chest deformity present but an evaluation of chest wall factors was not undertaken in the present study.

Lung volumes were probably underestimated in the present study, particularly in the subjects with moderate and severe barrel chest deformity. Meisner and Hughes-Jones (1968) found a significant volume of 'trapped gas' when thoracic gas volume measured by a body plethysmograph was compared with lung volumes measured by a gas dilution method.

With more severe degrees of barrel chest deformity there was a corresponding reduction in weight. Height was only affected in the most severe grades of barrel chest deformity. These changes in growth were probably due to asthma and were not the result of steroid therapy. 11 subjects had been on steroids usually for short periods of time. None of the 4 subjects with severe barrel chest deformity had received steroids.

As bronchial asthma is an episodic disorder, difficulties arise when objective physiological methods are used in its study. In the same subjects such measurements may vary a great deal from time to time especially in subjects who have no residual disability between their attacks.

Thus, when objective physiological measurements are used, it is essential to define the samples of subjects studied using clinical criteria especially the pattern of symptoms (whether episodic or persistent) and the clinical state at the time of examination.

There are numerous reports in the literature on the physiological derangements in lung function that occur in bronchial asthma (Beale, Fowler, and Comroe, 1953; Kraepelien, Engstrom, and Karlberg, 1958; Engstrom *et al.*, 1958; Engstrom *et al.*, 1959; Andrewes and Simmons, 1959; Bernstein *et al.*, 1959; Wells, 1959; Orzalesi, Cook, and Hart, 1964; Tooley, Demuth, and Nadel, 1965). These studies have included measurements of lung volumes, ventilatory tests such as the maximum breathing capacity, and timed vital capacity, and pulmonary mechanics. However, the clinical grading of the asthmatic subjects in these reports has been poorly defined, generally being based on the frequency of attacks (Kraepelien *et al.*, 1958), or combined with other criteria, such as the frequency of administration of bronchodilators, time off school, and the use of steroids (Tooley *et al.*, 1965). These criteria are mainly subjective in nature and as such can be misleading. The present study shows that growth, chest deformity, and the presence of rhonchi in an interval phase between attacks are important factors in the assessment of the asthmatic child. Until recently no previous published work had appeared on the significance of chest deformity.

Horowitz (1969) found that approximately 2% of asthmatics seen in a private and clinic practice had 'puffed up' chests between attacks. Most of these subjects had radiological and physiological evidence of hyperinflation, with raised lung volumes, and airways obstruction with reduction in the FEV_{1.0}/VC ratio.

In the present study each subject was only studied at one point in time, the study being so organized that longitudinal studies were not possible. Clinical observation however suggests that barrel chest deformity in a given subject is a constant finding though sometimes varying in degree at different examinations.

The majority of subjects studied appear to have persistent physiological abnormalities, and in some cases mild wheeze occurred each day. Such subjects were included in the study as being in an interval phase as long as they were in their usual state.

The results show that in moderate and severe grades of barrel chest deformity observer error is minimal as regards the presence or absence of barrel chest deformity, the discrepancy being over the degree of deformity present. Confirmation of

pulmonary hyperinflation in an asthmatic subject with moderate or severe barrel chest deformity by means of x-rays or measurements of lung volumes is not necessary. For mild grades of deformity however such aids are of value.

Howatt and Demuth (1965) made measurements of the antero-posterior, transverse, and vertical dimensions of the chest in a group of normal children and a group with fibrocystic disease of the pancreas. In the fibrocystic subjects they found an increase in the above measurements especially the antero-posterior measurements. These measurements were not done in the present study but may be of value in defining barrel chest deformity more precisely.

Observer error is also a factor in assessing chest x-rays. McNicol *et al.* (1970) showed that 6% of a control group were assessed as having radiological evidence of hyperinflation at 7 years of age. This factor appeared unlikely to affect the present results to any significant extent.

Harrison's sulcus deformity occurred in 23% of the present group. As such deformity may occur in a control population (McNicol *et al.*, 1970), it is not as selective as barrel chest deformity. However, this incidence of Harrison's sulcus deformity is higher than in the group of asthmatics studied by McNicol *et al.*

Pigeon chest deformity also occurred in 23% of the present group, only occurring in those subjects with some degree of barrel chest deformity. The cause of this type of deformity is obscure, though its association with barrel chest deformity suggests that chronic pulmonary hyperinflation is a factor in its development.

Twenty subjects had clinical, radiological, and physiological evidence of hyperinflation. Relating this number to the population sampled (21,000) gives an incidence of approximately 0.1% of a general population. This figure only represents 2 to 3% of asthmatics at 10 years of age, who are still having attacks of asthma (3 to 4% of a general population) (Williams and McNicol, 1969).

The criteria used for selection were designed to obtain a relatively severe group of asthmatic children. The first two criteria used, early onset, and frequent attacks, were selected on the basis of the findings of Williams and McNicol (1969) and McNicol *et al.* (1970). The third criterion, i.e. barrel chest deformity and/or reduction of the FEV_{0.5}/VC ratio to 50% or less was used as it was considered that one of the criteria should be objective in type, and that the combination of the above criteria would yield a manageable number of subjects for study. The presence of rhonchi on

auscultation of the chest was a possible criterion to use, but, from the findings of McNicol *et al.*, would not have been as selective. Chest deformity also appears to be a more permanent finding than rhonchi and thus a better criterion for selection.

However, the use of barrel chest deformity and/or reduction of the FEV_{0.5}/VC ratio to 50% or less meant that some bias was built into the study when chest deformity was correlated with physiological measurements and radiological findings. If, for example, the true incidence of chest deformity was only 10% in severe asthmatics, the present method of selection makes it appear 50% or more. However, some form of subsampling was necessary in the study. The large number of subjects with no barrel chest deformity (over 40%) showed obvious differences from those with deformity. The results of McNicol *et al.* (1970) also indicate that such findings are found infrequently in a randomly selected asthmatic population of varying grades of severity.

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