ATELECTATIC OR COMPENSATORY BRONCHIECTASIS

BY

LEONARD FINDLAY, M.D., D.Sc., M.R.C.P.
(Physician, Princess Elizabeth of York Hospital for Children, London.)

Historical.

Atelectasis or collapse of the lung as a cause of bronchiectasis is not by any means a recent idea. Reynaud, as long ago as 1885\(^1\), in a comprehensive study of bronchial obstruction, noted that dilatation of the lumen might be present both proximal and distal to the obstruction. The dilatation proximal to the obstruction he ascribed to the increased force of the inspired air held up at the obstruction and that beyond the obstruction he considered was due to the bronchi attempting to fill the space vacated by the collapsed lung.

Some ten years later Hasse\(^2\), in the discussion of bronchiectasis in his treatise on ‘Diseases of the Organs of Circulation and Respiration,’ expresses the opinion that the condition is invariably due to collapse or shrinkage in size of the lung. A common cause of the collapse he considered obstruction of the bronchus by secretion from an inflamed mucosa. As he says, ‘the bronchi become plugged with fibrinous exudation occasioning a collapse of adjacent air cells. The space thus set free is sought to be filled by expansion of the neighbouring parts, giving rise in the majority of cases to emphysema; where, however, the collapse does not occur close beneath the surface of the lung, but at a greater depth and near a large bronchial tube, and where it comprehends a larger tract of pulmonary substance, the result is bronchiectasis.’ Hasse attributes the occurrence of bronchiectasis following pneumonia and pleurisy to the same underlying cause, viz.:—the inability of the damaged lung to expand sufficiently to fill the thorax and not, as had been done some years previously (1888) by Corrigan\(^3\), to the contraction of new-formed fibrous tissue. It is interesting to note in passing that Hasse remarks that the variety of bronchiectasis following pneumonia he has met with ‘most frequently, in the inferior lobe, especially of the left lung.’ Even for bronchiectasis developing in a tuberculous focus at the apex of the lung he suggests the same explanation.

The teaching of Reynaud and Hasse did not receive, however, general acceptance. The majority of authors apparently could not visualize dilatation of the bronchi resulting from collapse unless in consequence of some abnormal positive force, particularly of the nature of increased intra-bronchial pressure. Such had been Laennec’s\(^4\) conception of genesis, for in his original description (1819), he ascribed the condition to distension by secretion accumulating within the bronchus. This idea of increased intra-bronchial pressure also underlies the teaching of Stokes (1837)\(^5\) who considered increased expiratory effort, as in coughing, the true aetiological factor. On the other hand, Corrigan as just mentioned, saw the abnormal
force acting from without and caused by the contraction of inter-bronchial, new-formed fibrous tissue.

Curiously, that astute physician, W. T. Gairdner (1850-51)\(^6\), in a critical survey of the 'Consequences of Bronchitis,' while concluding that emphysema results from an attempt on the part of one portion of the lung to fill a space in the thorax previously occupied by some other portion of the lung, and is thus truly complementary, absolutely denied that the same factor could be responsible for the inception of bronchiectasis. It was presumably the fact that bronchiectasis is often localized, and at the same time is saccular in nature, which prevented him from accepting the view of Hasse, although this author had quite unequivocally stated that tubular and saccular dilatations were met with in the same portion of the lung and therefore could not be taken as evidence pointing to any particular aetiology. The primary cause of bronchiectasis Gairdner considered some local weakness of the bronchial wall and he concluded that the dilatations were 'the result of excavations of the lung communicating with the bronchi.' He admitted that once they have originated as ulcerative cavities they might become 'afterwards expanded beyond their original dimensions by inspiratory force.'

The view that pulmonary collapse did play a part in the aetiology of bronchiectasis nevertheless did continue to receive some support. Thus Bastian in 1870\(^7\), while opposing the teaching of Corrigan that cirrhosis of the lung is invariably accompanied by, or invariably accompanies bronchiectasis, mentions that dilatation of the bronchi may be compensative in nature, although his phraseology reveals a certain confusion of thought regarding its modus operandi. He says, 'It is obvious that something must go towards filling up the space left by the shrinking lung, and if displacement of viscer a does not take place, then the bronchi must yield and dilate in some of their weakest parts under the continually increasing pressure of the inspired air.'

The dilatation is undoubtedly due to the inspiratory efforts but not because of a rise in the intra-bronchial pressure. Inspiration acts by increasing the capacity of the thorax which, as Nature abhors a vacuum, must be filled and this can only occur by dilatation of any spaces in communication with the external air. Normally the air cells provide for this but when these are not available, as in the state of collapse, then the bronchi must take their place. This point of view is clearly expressed in the recent article on 'Dry bronchiectasis' by Wall and Hoyle\(^8\). The conception of respiration acting through an increase in the intra-bronchial pressure is due to a want of appreciation of the physical conditions during inspiration and expiration and is being continually encountered in the discussions of both emphysema and bronchiectasis not only in the works of the earlier writers but even in some of most recent date (McCallum\(^9\)). The inspired air enters by suction and hence during this phase of respiration there is in reality always a diminished pressure within the bronchi. On the other hand, during expiration the intra-bronchial pressure may be raised but it is most improbable that this can have any dilating effect, since the bronchi are amply supported by the surrounding lung and other thoracic tissues. As Hedblom\(^9\) puts it, the force which would cause increased intra-bronchial pressure is applied to the outside of the bronchi.

An important date in the history of bronchiectasis is 1885. In that year Heller\(^10\) published a communication entitled, 'Die Schicksale
atelektatischen Lungenschnitten,' in which dilatation of the bronchi was referred to as a sequel of congenital atelectasis and in which we find the term 'atelectatic bronchiectasis' employed for the first time. Until this date all writers had recorded bronchiectasis consequent on collapse of a lung previously air-filled, and thus it is not surprising that the condition had never been referred to as atelectatic bronchiectasis because such a designation would not be correct etymologically. For this very reason not a few at the present moment object to the term as employed to-day because no one, not even those who most frequently use it, believe that congenital atelectasis is a common cause. A term which would be more strictly correct, and which would apply equally to dilatations consequent on atelectasis or collapse of the lung is 'compensatory bronchiectasis' and hence it is used as an alternative in the title of the present article.

Heller in his paper describes severe bronchiectasis in several patients dying between the ages of nine months and seven-and-a-half years which he considered as the sequel of congenital atelectasis because of the situation of the lesion, the absence of pigment and the histological appearances. Like Laennec, and in contrast to most present-day observers, Heller found the condition most frequent in the upper lobes. He says it usually involves a whole lobe (most seldom the middle and lower lobes) which is pale, smaller than normal and composed of many pus-containing cavities separated by narrow septa devoid of anything suggesting lung tissue. It is the absence of pigment and alveolar spaces which caused Heller to consider the condition due to changes ensuing in pulmonary tissue which had never expanded. Heller does not discuss the way in which he believes the bronchial dilatation comes about but simply contents himself with recording its occurrence and describing the appearances, naked-eye and microscopic. He does say, however, that he is doubtful if the same results would ensue in lung which had become secondarily atelectatic because the cause of the post-natal collapse would most probably bring about other changes in the lung.

Other writers who have described cases of bronchiectasis which they considered due to congenital atelectasis are Lutmar (1908) and Buchmann (1911). Lutmar records two examples of atelectasis, involving the left and right lower lobes respectively. In both the pulmonary tissue was shrunken, the alveoli were collapsed but not obliterated, and there was much new-formed fibrous tissue around the bronchi but it was only in the example implicating the left lower lobe that bronchiectasis was present. Although pigment was present in the shrunken right lower lobe Lutmar did not consider this fact inconsistent with a diagnosis of congenital atelectasis.

Buchmann described five examples of pulmonary collapse with bronchiectasis discovered post mortem in patients dying between the ages of forty-nine and eight-four years. In two of the patients both upper lobes were involved, in one an upper lobe only and in two the left lower lobe was the seat of the mischief. It is interesting to note that while Lutmar ascribed no significance to the presence of pigment as indicating whether the pulmonary collapse was congenital or acquired, Buchmann, like Heller, considered this a most important differentiating feature. For example, in one of his patients with bilateral disease involving each upper lobe pigment was present on one side and not on the other and in consequence he concluded that the condition was due to acquired collapse and congenital collapse respectively.

* Heller used the expression "Atelektatische Bronchiektasien."
In spite of communications such as the above, pulmonary atelectasis (either congenital or acquired) was not accepted by the majority of writers as an important factor in the causation of bronchiectasis. Most writers during the latter part of the last, and the first quarter of the present century give it scant notice, although it should be mentioned that D. J. Hamilton was a notable exception when he wrote in 1894 that "pulmonary collapse is a fertile source of bronchiectasis in children." Within the last decade, however, pulmonary collapse as a cause of bronchiectasis has become increasingly stressed until at the present moment it would appear to be the most popular theory of aetiology. This change in opinion is not due to any fresh pathological evidence, nor is it because of a better appreciation of the clinical manifestations of the disease, but depends almost entirely on the view that a triangular shadow in the skiagram and situated at one or other diaphragmatico-vertebral angle invariably spells collapse of the lung.

The triangular shadow.

It is a general law in radiology that any abnormal appearance may be caused by several distinct pathological conditions and it would be remarkable if the so-called triangular shadow formed an exception to this rule. This is, however, not the case for there are at least four different pathological lesions which can give rise to a wedge-shaped shadow occupying one or other diaphragmatico-vertebral angle.

1. Mediastinal pleurisy.—There is no doubt that a radiological shadow, of a somewhat triangular shape with a sharply defined outer margin and situated in one or other diaphragmatico-vertebral angle, may be produced by an accumulation of fluid (usually purulent) in the mediastinum or its neighbourhood. This was first pointed out by Savy who remarked that it resembled the appearance caused by a pericardial effusion. This would suggest that the outer margin would have a convex contour and, as a matter of fact, inspection of the various diagrams illustrating Savy's paper shows that the hypotenuse of the triangle is invariably convex. This is what would be expected, especially if the fluid were under pressure.

Although there have been recorded not a few supposed examples of this condition since the first description of Savy in 1910, it is questionable if they were all of this nature. In only a small proportion of the cases was the presence of fluid confirmed either by exploration or at autopsy, and it is only in those so verified, as for example, in the one recorded by Wallgren (fig. 1), that the skiagram revealed a convex hypotenuse. In the cases in which the diagnosis was not verified, and this obtains as we have already remarked in the majority of published reports, the hypotenuse of the triangle was straight or even concave so that in all probability the pathological lesion was of a different nature. It must be pointed out, however, that a convex hypotenuse, although essential for a diagnosis of fluid, does not necessarily indicate that this is the nature of the lesion. In one personally observed example of unequivocal pulmonary collapse, in which reinflation of the lung
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caused the immediate disappearance of the shadow, the hypotenuse was definitely convex (fig. 4).

Assmann\textsuperscript{16}, while discussing mediastinal pleurisy, says that in his experience the lesion causing this radiological appearance is more frequently cicatricial in nature due to tuberculosis of the mediastinal glands or more rarely following pneumonia. Assmann speaks of the hypotenuse being concave so that he certainly was not dealing with an accumulation of fluid. That pleural thickening and mediastinal fibrosis may explain some of the triangular-shaped shadows is not improbable, since we have recently observed the persistence of a typical triangular shadow in a case of bronchiectasis after removal by lobectomy of a supposed atelectatic lobe.

\begin{figure}
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\includegraphics[width=0.5\textwidth]{image.png}
\caption{Skiagram in case of mediastinal pleurisy on right side recorded by Wallgren. Note the convex contour of triangular shadow in right vertebro-diaphragmatic angle. The chest was explored and 20 c.c. straw coloured, serous fluid removed. [From Beiträge, z. Klinik d. Tuberkulose, 1928, LXIX, 641.]}\label{fig:skiagram}
\end{figure}

2. **Fibrosis of lung.**—In 1927 Rist, Jacob and Trocémé\textsuperscript{17} recorded five cases in which a triangular shadow in the diaphragmico-vertebral angle was not due to a collection of fluid but to fibrosis of the lung with bronchiectasis. It is interesting to note that in three of the patients the radiological appearances were responsible for a diagnosis in the first instance of a mediastinal pleural effusion.

One typical example of the series is that of a girl aged twenty years who had a cough and purulent sputum with occasional haemoptysis since an attack of measles at the age of two years. She came under observation during an acute exacerbation with fever, haemoptysis, severe cough and foetid sputum, and at this time X-ray examination revealed a triangular shadow at the right base. Although the published reproduction of the X-ray picture referring to this case in the series shows the hypotenuse to be concave, a diagnosis of purulent mediastinal pleurisy was made. However, on exploration of the
chest by operation, no pus was discovered but a much fibrosed lung, and a lipiodol injection at a later date disclosed bronchiectasis.

In spite of the fact that Rist and his colleagues demonstrated at operation that the lung was fibrosed and firmly adherent with a much-thickened pleura these cases have been not infrequently quoted by subsequent writers as examples of atelectasis with bronchiectasis.

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<tr>
<td>1. Left border of heart.</td>
<td>4. Pneumonic consolidation of right upper lobe.</td>
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<tr>
<td>2. Right border of heart.</td>
<td>5. Pneumonic consolidation of right lower lobe.</td>
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<td>3. Left border of infracardial lobe showing pneumonic infiltration.</td>
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**FIG. 2.**—Schema of x-ray picture in case of pneumonia recorded by Ettig. [From Monatsch. f. Kinderheilk., 1924, XXVIII, 209.]

**FIG. 3.**—View of horizontal section through heart and lungs on level with lower part of root of lungs seen post-mortem in case of pneumonia recorded by Ettig. [From Monatsch. f. Kinderheilk., 1924, XXVIII, 211.]

3. **Pneumonia.**—A triangular shadow may also be caused by acute pneumonic consolidation. In this condition the hypotenuse is straight. Such a finding has been described by Gullbring and Ettig in two instances in which the diagnosis was verified by post-mortem examination. In Ettig's patient, it is interesting to note, the pneumonia involved an accessory (infracardiac) lobe (fig. 2 and 3). Considering how frequently a complete...
lower lobe is affected in pneumonia, it is indeed remarkable how seldom such a radiological picture is observed. The only pneumonic consolidation which is commonly accompanied by a sharp margin to the radiological shadow is that involving the right upper lobe when the sharply defined lower and horizontal border is ascribed to the presence of an interlobar pleural exudate. The same accumulation of exudate must take place frequently between the upper and lower lobes, but in these circumstances the layer of exudate is not seen 'end on,' as is the case when it is present in the fissure between the upper and middle lobes, but rather 'on the flat,' and it may be for this reason that it seldom casts a shadow in the radiogram.

4. Collapse of the lung (atelectasis).—I do not know who was the first to suggest that this peculiar and characteristic shadow might be caused by pulmonary collapse but the earliest indication that I have come across is to be found in a paper by Singer and Graham\textsuperscript{20} entitled 'The roentgen-ray study of bronchiectasis.' These authors state that in some cases operated upon the triangular shadow had been found to be due to atelectatic and bronchiectatic lobes.

There is no doubt that a triangular shadow situated in one or other diaphragmatico-vertebral angle may arise from pulmonary collapse\textsuperscript{21}. I have seen several examples in which atelectasis was unequivocally the cause of the x-ray appearances because inflation of the lung brought about a rapid disappearance of the shadow. It should be noted, however, that when the shadow results from collapse of the lung the hypotenuse of the triangle may be convex, straight or concave. This variation in the radiological picture probably depends on the amount of air left in the lung. But whatever the explanation, this fact renders its differentiation from the other types of lesion by radiological means alone impossible. The only criterion by which it can be decided that a particular shadow in the skiagram is due to collapse of the lung, at least during life, is the ability to cause its rapid and complete disappearance by re-inflation. This is the acid test and may be applied by any means which brings about forced and deep breathing. The method which I have generally adopted is the inhalation of carbon dioxide in oxygen.

It is worthy of comment that a sharply defined triangular shadow is never observed in the case of the pulmonary collapse (so-called massive collapse) which occasionally ensues after abdominal or thoracic operations. This only makes it all the more striking that it should be such a constant picture in the case of spontaneous pulmonary collapse in the child. It is just possible that this difference is due to the fact that in the former the condition is not limited to one lobe but invades the whole lung or at least the greater part of one lung. Graberger\textsuperscript{22}, while discussing this very point, says that the only conditions which give a sharply defined shadow radiologically are encapsulated exudates or pneumonia which extends to a fissure, and at the same time he raises the question of some of the shadows being
due to disease of an accessory lobe. He would explain the smaller shadows in this way. Such an assumption, however, seems quite unnecessary since in those cases in which the whole lower lobe was known to be involved in the disease (pneumonia or atelectasis) the size of the shadow in comparison with that of the lobe itself seems relatively small.

The presence of an accessory lobe is quite a common condition in the lower animals and in the ape and is not unknown in man. Kerley23, following the teaching of Schaffner24, has stressed the frequency of the accessory lobe in man, especially on the right side, and says that it is present in 40 per cent. of individuals. In my post-mortem experience an accessory lobe is a rare finding and Schonfield25 records that he only observed the condition once during the course of several years' post-mortem examinations. Kerley also makes the statement that this abnormality of the lung is a potent factor in the causation of bronchiectasis but he gives no reason why he has come to this conclusion. One feels that the frequency with which the bronchiectatic condition extends into the neighbouring lobe is rather against such a view. Further, it is difficult to reconcile this aetiological factor, which Kerley and all writers admit occurs most often on the right side, with the predilection of bronchiectasis for the left side. The frequent occurrence of bronchiectasis at the left base, however, is one of the most difficult features to account for by any explanation of the pathogenesis of the disease.

**Atelectatic bronchiectasis.**

Within recent years there have been published not a few cases of so-called atelectatic bronchiectasis but unfortunately in not one is it certain that this diagnosis was correct. For the most part the diagnosis depended simply on the presence in the skiagram of a triangular shadow in association with bronchiectasis. It has been indicated that this shadow may be produced by several different pathological lesions and not only by atelectasis. During life, and it may be noted that practically all the published cases were observed during life, the only way to be certain that the shadow is due to atelectasis is, as already mentioned, its immediate disappearance on re-inflation of the lung. This, according to the authors, was attempted in several of the patients but in not one instance did it meet with success.

An oft-quoted example of atelectatic bronchiectasis is that published by Sparks26 in which the shadow did disappear, but it should be noted that this was spontaneous and not in response to any measure which had been adopted. This case was recorded under the title of 'An unusual case of bronchiectasis.'

The patient was a girl of twenty-two years who had pneumonia at the age of seven years and ever since then had suffered from recurrent attacks of bronchitis and symptoms of asthma. On coming under observation physical examination revealed flattening and relative dulness with a deficient
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respiratory murmur on the left side of the chest and fine, sticky crepitations at the base. X-ray examination at this date demonstrated a triangular shadow at the left base. Some time later, exactly how long is not stated, and immediately after the injection of lipoidol, the triangular shadow had disappeared but a severe degree of bronchiectasis was demonstrated in the situation which had been occupied by the shadow. Unfortunately in the print published all the lipoidol is in the right (healthy) lung. However, at a still later date, injection of lipoidol was repeated and the accompanying print of the skiagram does reveal a severe degree of bronchiectasis in the left lower lobe.

It is quite possible that the triangular shadow in Spark’s case was due to pulmonary collapse, but it is by no means certain that this was so. The radiological appearances could equally well be due to pneumonia. But what is most unlikely is that the pulmonary collapse, if indeed it were such, had any aetiological significance for this would mean that it had been present for sixteen years (the symptoms having been present since the patient was seven years old) and that at the end of this time the lung had been capable of complete re-inflation while the severe bronchiectatic condition persisted. One does know that the lung may be kept collapsed by means of an artificial pneumothorax for several years and yet suffer no damage, but one would have thought that after a period of sixteen years the atelectatic lung would have undergone fibrosis. Hence, if the shadow were caused by pulmonary collapse, it in all probability was an intercurrent event. There was also lacking the acid test—the immediate disappearance following attempts to re-inflate the lung. It might be argued that a failure to cause the shadow to disappear by re-inflation did not necessarily indicate that the condition was not due, at least in the first instance, to collapse. After a time the collapsed lung becomes fibrosed so that its re-expansion is impossible. This possibility has indeed been suggested by Ellis\(^27\), and, as he remarks, makes it exceedingly difficult to draw definite conclusions.

One naturally thinks of pathological evidence as the best means of determining the nature of the lesion. This type of evidence, however, has serious limitations since, as a rule, it is only obtainable many years after the onset of the mischief, when time has been given for secondary changes to set in and ample opportunity for superimposed inflammation, so that it is difficult to unravel the story of events. So far as I am aware there has been only one example of supposed atelectatic bronchiectasis submitted to post-mortem examination within recent years and in this no minute histological examination was carried out\(^29\). One should perhaps include in this category the examples quoted by Singer and Graham\(^29\) which were removed at operation, but here again histological evidence is wanting. In any case, it would seem from the available descriptions\(^59\) that the changes supervening in the atelectatic lung closely simulate those which follow inflammation, so that the end-results in atelectasis and unresolved pneumonia are indistinguishable. The great desideratum at the present moment is minute investigation of material obtained earlier during the course of the disease and it is to be
hoped that the operation of lobectomy, which is becoming more popular, will supply an opportunity of which full advantage will be taken. In the meantime it would seem wise to reserve judgment regarding any particular example of bronchiectasis ascribed to collapse of the lung unless there is proof of the lung being re-inflatable.

One does not deny the possibility that some of the cases above referred to, and others appearing in the literature, were examples primarily of pulmonary collapse with compensatory bronchiectasis. The view held is that in none of them has it been shown that collapse of the lung had ever occurred and hence that this lesion had played any part in the production of the bronchial dilatation. In all of them there must have been fibrosis, otherwise the lung could have been inflated. The fibrosis itself, which it must be remembered has several causes, could account for the bronchiectasis. At any rate, it would appear in view of the cases recorded below that it is a subsequent fibrosis or cirrhosis of the lung, whatever its cause, which not only hinders the bronchial tree from returning to its normal state but which also increases the abnormality. At least when I have been able to demonstrate by causing re-inflation of the lung that atelectasis or pulmonary collapse really was present, any bronchiectasis which existed was slight in extent and was completely recovered from. The following case is such an example.
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Case records.

Case 1. A. R., a boy, first came under observation at the age of eight years in March, 1932. He had had a healthy infancy and had remained well till he was aged five years when he suffered from an attack of pneumonia. Since then he is reported 'never to have been the same, to be puny and undersized and frequently ailing.' There was no history of sputum. Three months previously there developed considerable enlargement of the cervical glands on the left side which, however, had largely but not entirely disappeared. The tonsils had been removed at the age of six years.

![Image of lung X-ray](http://adc.bmj.com)

**Fig. 5.** Case 1.—Pulmonary collapse. Skiagram immediately after the inhalation of carbon dioxide in oxygen. Note that the shadow is less dense and has retracted from chest wall and diaphragm. Compare with fig. 4.

Three weeks before coming under observation he had contracted measles. The attack was uncomplicated, but because the Mantoux tuberculin test was strongly positive he was referred to me for observation. At this time (March, 1932) he was definitely underweight (weighing 43 instead of 55 lb.), he complained of a troublesome cough at night but with the exception of some enlargement of the glands on the left side of the neck and a slight degree of rhinitis, nothing abnormal was detected on physical examination. The boy was given tonic treatment and recommended for a period at a convalescent home.
Although he increased in weight the cough persisted. In August, 1932, the Mantoux test was strongly positive. On September 7, the house physician, during his routine examination, detected for the first time some impairment of the percussion note at the right base behind with tubular breath sounds and x-ray examination of the chest revealed a definitely abnormal and unusual picture (fig. 4). There was at the right base a shadow, convex and sharply defined above and merging below with the liver shadow. I saw the child on October 5, when it was noted that expansion of the right chest was defective and that the percussion note was impaired all over the right side with definite dulness at the base behind where
the breath sounds were tubular in character. On screening, the shadow at the right base, was observed to be very dense, to be sharply defined above, and to be continuous below with the diaphragm which was immobile. A provisional diagnosis of pulmonary atelectasis was made and the boy was admitted to hospital for investigation.

On October 11, after one week in hospital, the following note was made: 'Patient is quite comfortable but has a troublesome cough. There is no sputum and no fever. The pulse which on admission was 110 has during the last three days declined to between 60 and 70. Movement of right side of chest is still defective. Note over right side is generally of higher pitch but definitely dull at base behind, where the breath sounds are deficient. Shadow in skiagram at right base if anything less dense but still very distinct.

![Image of a chest X-ray showing pulmonary collapse](http://adc.bmj.com/)

**FIG. 7.** Case 1.—Pulmonary collapse. Skiagram of chest taken six minutes after disappearance of triangular shadow and showing normal lung fields. [2.V.33.]

In order to test the question of atelectasis inhalation of carbon dioxide in oxygen was practised. The child became cyanosed, breathed deeply and was rigid (? tetany) and excitable for a short period. A skiagram taken afterwards revealed the shadow practically unchanged. On October 14, an inhalation of carbon dioxide in oxygen was again resorted to. On this occasion there did result a change in shadow (compare fig. 4 before and fig. 5 after inhalation of carbon dioxide). In fig. 5 the shadow seems less dense and more retracted from the chest wall. As the cough persisted and the shadow had not absolutely disappeared
it was decided to give a lipiodol injection. This was done on October 18, and revealed a definite though slight degree of bronchiectasis (fig. 6). In this picture the shadow seemed to have disappeared.

The patient was recalled in May, 1933, six months after the last examination. In the interval he had been quite well with only an occasional cough. Expansion of the right chest seemed still deficient and after inversion of the child, producing cough without any sputum, the breath sounds assumed a questionable tubular quality at the angle of the right scapula where the whispered voice was unduly distinct but not to the extent which had been present before. X-ray examination of the chest at this time did not reveal any shadow (fig. 7) and after injection of
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lipiodol there was no evidence of the previous bronchiectasis, the bronchi having quite regained their normal conformation (fig. 8).

Another probable example of atelectatic bronchiectasis is the following (case 2), although in this instance re-inflation of the lung was not attempted. The patient was one of a series which Dr. Stanley Graham and I reported in an earlier issue of this Journal during a discussion of the 'Prognosis in bronchiectasis'.
seems specially characteristic of pulmonary collapse, and the complete disappearance of the bronchiectasis make it in all its features practically identical with the preceding case.

**Case 2.** (Case 10 of original series\(^a\)). A. S., a boy, came under observation on May 25, 1927, when aged twelve-and-a-half years, with a history of cough with sputum of three months' duration. At the age of two years he had passed through an illness comprising measles, whooping cough and pneumonia, but he apparently made a good recovery and had remained in good health till the onset of the present illness. The cough had steadily got worse and was often severe enough to cause vomiting. There was impairment of the percussion note at both bases behind with somewhat tubular breath sounds and x-ray examination revealed a triangular-shaped

![Fig. 10. Case 2.—Possible pulmonary collapse. Skiagram taken 10 days after fig. 9, showing disappearance of shadow at left base and increase in density of shadow at right base, which has now lost convex contour. [7.VI.27.](http://adc.bmj.com/ArchDisChild: first published as 10.1136/adc.10.56.61 on 1 April 1935. Downloaded from http://adc.bmj.com on 22 October 2023 by guest. Protected by copyright.)](http://adc.bmj.com/ArchDisChild: first published as 10.1136/adc.10.56.61 on 1 April 1935. Downloaded from http://adc.bmj.com on 22 October 2023 by guest. Protected by copyright.)

shadow at either base. The shadow was less dense on the right side but had a convex contour while on the left side the hypotenuse of the triangular area was straight. A provisional diagnosis of pulmonary fibrosis with possible bronchiectasis was made and the boy was admitted to hospital for investigation.
On admission, June 7, he was described as a big well-nourished boy, 152 cm. tall and weighing 29·5 kgm. Examination of the chest at this date revealed impairment of the percussion note only at the right base behind where the breath sounds were amphoric in quality and whispering pectoriloquy was well heard close to the midline. At the same time a skiagram showed the shadow still present at either base, less dense at the left base but more dense on the right side with the hypotenuse now straight (fig. 10), and after the injection of lipiodol a definite but moderate degree of dilatation of the bronchi (fig. 11). This finding at the time was taken to corroborate the diagnosis of fibrosis with bronchiectasis, the now-apparent significance of the disappearance of such a shadow not being appreciated.

On April 3, 1930, two years and nine months later, and when the boy was fifteen-and-a-half years of age, he was again seen.
He appeared in perfect health, and the cough and sputum had entirely disappeared. His height was 167.5 cm. and his weight 48.5 kg. Examination of the chest still revealed a very slightly impaired percussion note with deficient breath sounds at the right base, but neither amphoric breathing nor pectoriloquy was audible. A skiagram after a lipiodol injection showed that the bronchi were normal in their conformation and there was no suggestion of the previously noted triangular shadows (fig. 12).

![Figure 12](http://adc.bmj.com/)

**Fig. 12.** Case 2.—Possible pulmonary collapse. Skiagram after lipiodol injection showing return to normal distribution and conformation of bronchi of right lower lobe, which had been seat of bronchiectasis 3 years previously. [3.IV.30.]

Compare with fig. 11.

Dr. Stanley Graham kindly saw the patient for me in February, 1934, when he reported that he was a big, well-developed youth, with neither cough nor sputum and that on x-ray examination of the chest nothing abnormal was detected except a line on either side radiating from about the level of the sixth vertebra to the middle of the diaphragmatic arch and indicating possibly the presence of supernumerary lobes.
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In still another case of the series which Dr. Graham and I reported in 1931, the question of collapse of the lung as a factor in the production of bronchiectasis must be considered. In this case also there was a shadow situated at the right base, but it simulated more what is met with in pneumonia than in pulmonary collapse, there being an absence of the triangular shape with a sharply defined outer margin (fig. 13). I personally incline to the diagnosis of pneumonia in this case which is all the more probable as the boy came under observation during a febrile illness. Nevertheless, it may be that the physical conditions which led to the dilatation of the bronchi in these circumstances, were, as originally suggested by Hasse, fundamentally the same, viz., the inability of the consolidated lung to expand and fill the thorax so that compensatory dilatation of some air-containing space must result. In any case, it is interesting to note that with the disappearance of the physical signs and radiological manifestations of the consolidated pulmonary tissue the bronchi returned to normal dimensions and it would therefore seem appropriate that the case record should be incorporated in this communication.

Case 3. (Case 5 of original series\textsuperscript{30}). J. F., a boy, first came under observation in August, 1925, when he was aged nine years. He

\[\text{Fig. 13. Case 3.—Skiagram showing consolidation of right lower lobe. [17.VIII.25.]}\]
had had influenza at three and measles and whooping cough at four years of age, since when he had been troubled with a cough and subject to febrile attacks. It was during one of these febrile periods that he came under observation. At this time he was noted to be an undersized boy measuring 108 cm. and weighing 17.2 kg. His colour was good and there was no clubbing of the fingers. The Pirquet tuberculin reaction was positive. Examination of the chest gave a percussion note which was dull with deficient breath sounds at the right base behind below the angle

![Image](http://adc.bmj.com/)

**FIG. 14.** Case 3.—Skiagram after lipiodol injection showing slight degree of bronchiectasis in right lower lobe. [20.VIII.25]
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of the scapula. When the child was made to cough while in the inverted posture one drachm of thick purulent sputum, devoid of all odour, was evacuated and the dulness at the right base became less intense and the breath sounds tubular in quality. X-ray examination revealed widening of the mediastinum and a diffuse mottled shadow at the right base (fig. 13). After the

![Image](image_url)

**Fig. 15.** Case 3.—Skiagram showing chest clear. [31.1.29.] Compare with fig. 13.

injection of lipoidol a slight but definite degree of dilatation of the bronchi was evident in the situation occupied by the above-mentioned shadow (fig. 14).

The boy was seen again in January, 1929, when his health was said to be satisfactory and he was eating and sleeping well and playing normally. He still had a cough but there was no sputum. The physical signs were negative.

On March 28, 1930, four-and-a-half years after first coming under observation, he still looked well but had a dry unproductive
cough. Physical examination was negative and skiagrams of the chest before and after the injection of lipoidal revealed no evidence of the shadow previously present at the right base or of the dilatation of the bronchi which had been noted in the same situation (fig. 15 and 16).

Fig. 16. Case 3.—Skiagram after lipiodol injection showing disappearance of bronchiectasis which had been present 3½ years previously. [9.II.29.] Compare with fig. 14.

Summary.

(1) The history of atelectasis (congenital and acquired) in the production of bronchiectasis is reviewed.
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(2) The view that the bronchiectasis accompanying pulmonary collapse is compensatory in nature is supported. The specific cause is the inability of the lung to fill the thoracic cavity and hence the necessity for some space communicating with the external air (alveolus or bronchus) to dilate.

(3) The significance of the so-called triangular shadow in the skiagram occupying one or other diaphragmatico-vertebral angle is discussed. This shadow is shown to be caused by encysted effusions, fibrosis of the lung and thickened pleura, pneumonic consolidation, and collapse of the lung. Stress is laid on the re-inflatability of the lung as essential for a diagnosis of pulmonary collapse.

(4) Some previously recorded cases of atelectatic bronchiectasis are considered and the correctness of the diagnosis submitted to criticism.

(5) Three personally observed examples of bronchiectasis with an accompanying triangular shadow are described. In all the bronchietatic lesion was situated at the right base, was of mild degree, and was completely recovered from. In one the shadow was certainly due to collapse of the lung and it is concluded that this was the cause of the bronchiectasis. In one other case collapse was the probable cause, and in the third case pneumonic consolidation appeared to be the more likely aetiological factor.

In conclusion, I have pleasure in expressing my indebtedness to Dr. Campbell Suttie, Radiologist at the Royal Hospital for Sick Children, Glasgow, and to Drs. B. J. Leggett and G. Calthrop, Radiologists at the Princess Elizabeth of York Hospital, London, for the various skiagrams reproduced in this paper.

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