ATELECTASIS AND BRONCHIECTASIS IN PERTUSSIS

BY

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Laennec in 1819 reported that he had found at post-mortem examination bronchiectasis in the left lower lobe of a child aged 3½ months who had died after an attack of whooping-cough, and stated that he considered the latter disease to be the commonest progenitor of bronchiectasis.

Lander and Davidson (1938) showed experimentally that aspiration of viscid material into peripheral parts of the bronchial tree may be followed by atelectasis and a mechanical compensatory dilatation of the bronchi in the collapsed area. Lander (1946) concluded that when bronchiolar occlusion produces atelectasis with associated bronchial dilatation the following events may take place: (1) the collapsed portion of lung may re-expand, with disappearance of the compensatory bronchial dilatation ("reversible bronchiectasis"); (2) the collapse and consequently the associated bronchial dilatation may be permanent; and (3) the collapsed portion of lung may re-expand, but the bronchi remain dilated because of damage to their walls by infective processes.

Present Investigation

This work affords a ready explanation for the association noted by Laennec between bronchiectasis and pertussis, because in the latter disease, as Erwin (1939) observed, "the tenacious nature of the sputum bespeaks collapse." It was with this in mind that the present investigation was undertaken, though it was not forgotten that Tannenberg and Pinner (1942) and others had shown that bronchiectasis may also be produced by the damming up of secretions distal to obstruction of a main bronchus from intrinsic or extrinsic causes.

With a view to determining the incidence of atelectasis and bronchiectasis in whooping-cough, 150 consecutive cases of this disease admitted to Ruchill Hospital during 1946-7 were examined clinically and radiologically. Cases which arrived gravely ill and died without having full investigation were excluded from the series. The cases were x-rayed at weekly intervals, and, when atelectasis was detected or suspected clinically or radiologically, lateral films were taken to verify the diagnosis and define the location. Lordotic as well as lateral views were used when right middle-lobe or lingular collapse was suspected.

In cases showing collapse of marked or moderate extent, and in cases with slight persistent collapse, bronchography was carried out except in children under 1 year. Mantoux tests were done on all cases on admission, and, if negative, were repeated not sooner than eight weeks afterwards. All cases were followed up until there was no clinical or radiological evidence of respiratory disease; many were kept under observation for over a year, and one is still under observation almost three years after the onset of pertussis.

Criteria of Atelectasis

Complete collapse of an upper or lower lobe is often easy to detect clinically, though even here an infected atelectasis may be confused with pneumonic consolidation. Collapse of the right middle lobe, however, as Punch (1940) has pointed out, is usually completely masked by compensatory emphysema, and postero-anterior and lateral

x-ray films are necessary for its detection. Doig (1946) and others have stressed that lordotic views often strikingly define atelectasis of the right middle lobe or lingular process.

When complete collapse of the right middle lobe can defy clinical recognition it is obvious that radiology is essential for the detection of small areas of atelectasis, and even then distinction from consolidation is not easy. If collapse is lobar or segmental in extent, postero-anterior and lateral x-ray films may reveal compensatory movements of the diaphragm, mediastinum, or interlobar fissures, and an opacity of characteristic shape, but when the collapse is very small these features are not seen. Diagnosis then depends on the appearance of the opacity and absence of pyrexia, leucocytosis, or other features of pneumonia, but, as infection and atelectasis may coexist, the distinction is often difficult. For this reason the diagnosis of small areas of atelectasis was not recorded unless confirmed by the independent opinion of a radiologist.

Radiological evidence of atelectasis was seen in 65 cases out of 150 (43%). The incidence in age groups is summarized in Table I.

TABLE I.—Age Incidence of Atelectasis in 150 Cases of Pertussis

Age in Years	Cases	Cases with Atelectasis
<1	44	9 (20%)
1—	30	14 (47%)
2—	19	11 (58%)
3—	17	9 (53%)
4—	13	8 (62%)
5—	12	8 (67%)
6—	11	6 (55%)
7—	3	—
8—	1	—
Total	150	65 (43%)

Lobar Incidence.—Some cases had more than one lobe involved, the lobes either showing atelectasis at the same time or becoming affected at different times. Thus, although atelectasis occurred in only 65 patients, more than 65 lobes were involved. In two cases the atelectasis was multilobular in distribution and these are excluded from the analysis. In the remaining 63 cases 85 lobes showed collapse of greater or less extent. Table II illustrates the number of times a particular lobe was involved, whether alone or in combination with other lobes. Table III indicates how

TABLE II.—Lobar Incidence of Atelectasis in 150 Cases of Pertussis

Lobe	No. of Times Involved
Left lower	43 (50.5%)
Right lower	31 (36.5%)
Right middle	8 (9.4%)
Right upper	2 (2.4%)
Left upper	1 (1.2%)
Lingula	—
Total	85 (100%)

TABLE III.—Anatomical Distribution of Atelectasis in 63 Cases of Pertussis

Lobe or Lobes	No. of Times Involved
Left lower alone	25
Right lower alone	15
Left lower and right lower	14
Left lower and right middle	4
Right middle alone	2
Right upper alone	1
Right middle and right lower	1
Right lung and left upper	1
Total	63

often the lobes were affected singly and how often in combination.

Extent.—To indicate roughly the degree of lobar atelectasis encountered the collapse may be described as slight, moderate, or marked, the latter category indicating that the lobe was completely or almost completely collapsed, and

the other categories indicating lesser degrees of involvement. Of the 85 instances of atelectasis, 60 (70.6%) were slight, 15 (17.6%) moderate, and 10 (11.8%) severe.

Time of Onset.—Atelectasis affected 85 lobes, and the time of onset was as follows: first week of illness, 2 cases of lobar collapse; second week, 10; third week, 18; fourth week, 24; fifth week, 12; sixth week, 8; seventh week, 4; eighth week, 4; ninth week, 1; tenth week, 1; eleventh week, 1. It will be seen that atelectasis occurred with increasing frequency until the fourth week of illness, and then its incidence declined.

Duration.—The duration in weeks of the collapse noted in the 85 affected lobes was as follows: 1 week, 16 instances; 2 weeks, 12; 3 weeks, 20; 4 weeks, 16; 5 weeks, 9; 6 weeks, 3; 7 weeks, 3; 8 weeks, 3; 9 weeks, 1; 13 weeks, 1; 18 weeks, 1. It will be seen that atelectasis seldom lasted longer than five weeks. In general, it could be said that atelectasis of slight extent cleared up more quickly than that of marked degree, but there were exceptions to the rule. In one case, for example, in which there was complete collapse of the right lung and extensive collapse of the left upper lobe, complete re-expansion occurred in four weeks.

Bronchopneumonia in Relation to Atelectasis

Bronchopneumonia is an extremely serious complication of whooping-cough and is probably responsible for most of the deaths occurring early in the disease. It has been mentioned, however, that those patients who were admitted to hospital gravely ill and who died before radiological investigation was possible are not included in this series.

Bronchopneumonia was detected radiologically and clinically in 30 of 150 whooping-cough cases. Seven of them developed the condition after admission to hospital. Atelectasis occurred in 12 (40%) of these 30 cases with bronchopneumonia. In 120 cases x-ray evidence of bronchopneumonia was not found, and of these, 53 (44%) developed atelectasis. In this series, therefore, bronchopneumonia occurring in whooping-cough had no influence on the incidence of collapse. Bronchographic studies failed to reveal any evidence of bronchial dilatation in whooping-cough cases complicated by bronchopneumonia except in atelectatic areas, and follow-up clinical and radiological investigations did not demonstrate the development of bronchiectasis in bronchopneumonic cases.

Slight enlargement of hilar glands was demonstrated radiologically in 28 (18.7%) of the 150 whooping-cough cases, but in no instance could collapse be attributed to this condition.

Indications of Onset of Atelectasis.—When this study was begun it was believed that an elevation of temperature might prove a valuable indication that atelectasis had occurred. This expectation was not realized, for analysis of the cases failed to show any definite correlation between rise of temperature and the occurrence of atelectasis; isolated temperature elevations were noted in cases in which collapse did not take place, while in some cases of extensive atelectasis the temperature was not elevated at all. It is interesting that Gray (1946) observed that pyrexia is less prominent when pulmonary collapse follows operations in the thorax than when it occurs after abdominal operations. None of the children complained of pain in the chest after pulmonary atelectasis, though some were too young to do so. Pleural friction was not detected over collapsed areas.

Conversion of the Mantoux test from negative to positive was not noted, nor did pertussis cause a "flare up" of existing foci of tuberculous infection.

Bronchial Dilatation in Atelectatic Areas of Lung

Six patients were encountered in whom pulmonary collapse of marked extent occurred. Two of them were under 1 year of age, and bronchography was not performed. This procedure was carried out in the four other cases, and in every instance well-marked dilatation of the bronchi in the collapsed area of lung was demonstrated. In one of these four cases there was extensive collapse of both lower lobes, with dilatation of the contained bronchi. Re-expansion of the right lower lobe took place after eight weeks, and re-expansion of the left lower lobe after nine weeks. Bronchography at this point showed very slight cylindrical dilatation of the bronchi in both lower lobes. Another bronchogram three months later showed that the bronchi had fully returned to their normal calibre. A "follow-up" examination showed no evidence of respiratory disease.

In the remaining three of these four cases the lung affected by the pulmonary collapse was relaxed by means of an artificial pneumothorax as a therapeutic measure. This must be mentioned, since the pneumothorax may have affected the duration of the atelectasis. In the first of these three cases the collapse affected the right middle lobe and the anterior basic segment of the right lower lobe. Re-expansion took place in eight weeks from the onset, and subsequent bronchography showed a normal bronchial tree. Eighteen months later a bronchogram revealed no abnormality and clinical observation did not show any evidence of pulmonary symptoms.

In the second of these cases the collapse affected the left lower lobe. Re-expansion occurred in thirteen weeks, and subsequent bronchography revealed a normal bronchial tree. A bronchogram eighteen months later again showed a normal bronchial tree, and there was no evidence of residual respiratory disease in a two-year "follow-up."

The third case had collapse of the left lower lobe on admission to hospital on March 26, 1947, and marked dilatation of the contained bronchi was subsequently demonstrated. The atelectatic area was grossly infected and the patient was gravely ill. Re-expansion took place eighteen weeks after onset, and subsequent bronchography showed an apparently normal bronchial tree. On discharge from hospital in September, 1947, the patient was free from symptoms, and physical and radiological examination of his chest revealed no abnormality. Since that time up to April 1950, he has had three episodes of cough and sputum production lasting a fortnight or so during which crepitations could be detected over the left lower lobe. Bronchography on two occasions after his discharge from hospital revealed no definite bronchiectasis of the left lower lobe, though one set of films suggested slight cylindrical dilatation of the bronchi supplying the anterior basic segment. It seems probable that the left lower lobe bronchi have been damaged, though whether recovery will eventually take place remains to be seen.

Fifteen of the cases in the present series showed atelectasis of moderate extent, but the bronchi in the collapsed portion of lung were outlined in only 10. Of the 10 cases bronchographically investigated, bronchial dilatation was detected in four, but was very slight in extent. In all instances the collapse cleared up in approximately one month, and subsequent bronchography revealed a normal bronchial tree. A "follow-up" did not disclose any evidence of residual damage. Bronchography was also carried out in 10 cases in which atelectasis of slight extent was present, but bronchial dilatation was not seen.

For various reasons bronchographic examinations were performed on 25 of the whooping-cough patients in whom pulmonary collapse was not present. In some of these atelectasis had previously been detected and had cleared up, while others had suffered from the complication of bronchopneumonia. Bronchial dilatation was never demonstrated.

In this series of 150 cases of whooping-cough, therefore, three well-marked examples of "reversible bronchiectasis" were observed, and there was one case in which, though the collapsed area re-expanded and the dilated bronchi apparently reverted to normal, damage was sustained that may yet lead to progressive disease. In four other cases insignificant ephemeral bronchial dilatation was noted. Bronchial dilatation was never detected except in atelectatic areas.

Treatment

When pulmonary collapse is caused by aspiration of sputum into the peripheral parts of the bronchial tree postural drainage in the appropriate position (Nelson, 1934), or rolling the patient about and percussion over the affected area (Santé, 1927), may be employed in an attempt to dislodge the peccant mucous plugs. These methods were employed in the majority of cases and the collapse often cleared up, but it was difficult to evaluate their efficacy. Inhalation of carbon dioxide has been advocated for its effect in stimulating respiratory movements, and also for its reputed tendency to liquefy the mucous plugs (Coryllos, 1930; Brown, 1930; Banyai and Cadden, 1943). Bronchoscopic aspiration has also been employed, but in general has proved somewhat disappointing, for, although a mucous plug in a main bronchus may be easily removed, plugs in the peripheral bronchioles are obviously not so accessible. Blades and Dugan (1944), for example, had no success in four cases of collapse which subsequently resolved spontaneously, and Hennell (1946) failed in six cases, though he produced temporary re-expansion in one. Although desirable, this method was not employed in the present series.

It has been mentioned that in four cases in the series well-marked bronchial dilatation was demonstrated in collapsed lobes, and since the collapse was known to be recent it could be assumed that the bronchial dilatation was merely compensatory and likely to disappear if re-expansion of the lobe took place quickly enough. Pending re-expansion it was considered that, if the affected lung were relaxed by an artificial pneumothorax to an extent sufficient to counter the mechanical stresses dilating the bronchi, permanent bronchiectasis would be less likely to result. This measure was instituted in three cases, but, though the effect appeared to be favourable, no conclusions can be drawn.

Discussion

Engel (1947) has suggested that hilar adenitis is an important factor in the production of atelectasis in pertussis, and Paulson and Shaw (1949) have stressed that the right middle lobe bronchus is particularly likely to be compressed by enlarged hilar glands. Although slight enlargement of the hilar glands was noted in 18.7% of cases in the present series, collapse was never apparently due to this factor. In all instances of atelectasis the causative factor seemed to be obstruction of bronchioles or finer bronchi, and the likeliest mechanism aspiration of viscid sputum into the bronchial tree. At the same time it was often noted that a small area of atelectasis appeared which increased in extent over a period of two to three weeks, and if the cause were aspiration of sputum it would be anticipated that the collapse would be maximal from the

beginning. Possibly in these cases the initial collapse was due to swelling of the bronchiolar mucosa or to aspiration of sputum with collapse of adjacent lung tissue due to subsequent "spill-over."

As Gray (1946) and others have pointed out that atelectasis occurs more often in children than in adults, possibly as a result of the narrower calibre of the bronchi, the low incidence of atelectasis in children under 1 year in the present series is interesting. Kohn *et al.* (1944) and Nicholson (1949) reported similar findings in pertussis, and it has been suggested that at this age little mucus is secreted by the bronchial mucosa.

Kohn *et al.* (1944) indicated that in pertussis a high incidence of right middle lobe collapse is to be expected, but in this series the incidence was that commonly found in respiratory affections, except for tuberculosis—namely, highest in the lower lobes, especially the left. In pulmonary tuberculosis Erwin (1939) found that collapse due to bronchiolar occlusion was most common in the upper lobes, and attributed this incidence to the fact that in tuberculosis secretion is found most commonly in this part of the lung. It is not easy, however, to apply this theory to pertussis. It is significant that the lobar incidence of bronchiectasis corresponds to the lobar incidence of atelectasis, and it is interesting that the lobar incidence of bronchiectasis in Tudor Edward's (1939) series of 166 lobectomies corresponds closely to the lobar incidence of collapse in this pertussis series, except that collapse of the lingula was not noted in the present study, though carefully looked for.

The comparatively frequent occurrence of "reversible" bronchiectasis was noteworthy, for, although the condition has been recorded by Findlay and Graham (1931), Findlay (1935), Jennings (1937), Franklin (1938), Raia (1938), Wilson (1939), Fleishner (1941), Ogilvie (1941), Diamond and Van Loon (1942), Blades and Dugan (1944), Lander (1946), and Wearing (1948), it is still regarded as something of a rarity, though it is probably observed much more commonly than reported. Lisa and Rosenblatt (1943) and Hinshaw and Schmidt (1944) denied that it was bronchiectasis at all, and thought the appearance of dilatation was an illusion due to foreshortening and crowding of the bronchi, but, as Ogilvie (1941) and Lander (1946) reported cases in which the collapse cleared up and the affected bronchi remained dilated, little doubt can now be entertained that the bronchi may be mechanically dilated as a result of pulmonary collapse, and that supervening infection may render the dilatation permanent.

How long atelectatic bronchiectasis may remain potentially reversible is unknown. In many of the reported cases the duration of the collapse could not be ascertained with certainty, but in one of Lander's (1946) cases the collapse lasted for four months, in Wearing's (1948) case it lasted for at least four months, and in a case reported by Raia (1938) the duration seems to have been six months. The degree of infection is doubtless the main factor militating against recovery, but it is conceivable that mere prolonged mechanical distension might leave the bronchi dilated even if the collapse cleared up, though this would possibly not be of much importance.

It was anticipated that small areas of collapse would not give rise to bronchial dilatation, as compensatory emphysema would be sufficient to relieve strain on the bronchi, and, as has been noted above, this expectation was realized in practice.

The incidence of respiratory disease following pertussis in this series was very low. Pulmonary collapse was noted in 43% of the patients, but in general it was small in extent

and ephemeral. Re-expansion took place in all instances before the patient was discharged from hospital, and in only one case did prolonged clinical and radiological follow-up reveal residual damage to the respiratory tract, and even here eventual recovery seems to be possible. Kohn *et al.* (1944), in a clinical and radiological study of 222 cases of pertussis, did not mention any sequelae, but Nicholson (1949) reported 22 cases of collapse of more than two months' duration in a series of 48 pertussis patients, and stated that in seven followed for a year the collapse was still present.

Whatever the general incidence of pulmonary collapse in pertussis, its importance is obvious, for its relationship to bronchiectasis seems clear. As in many cases it is impossible to detect atelectasis clinically, an x-ray film before supervision ceases would seem to be the minimum demand on the radiological department. If, however, respiratory symptoms follow pertussis it cannot be too strongly emphasized that a negative x-ray film is of little significance, for atelectasis may have cleared up, leaving bronchiectasis behind. It still does not appear to be widely appreciated that recurrent attacks of cough or "pneumonia" and persistent localized crepitations call for bronchography; even to-day there is a tendency to wait for the appearance of copious sputum and clubbing of the fingers before suspecting bronchiectasis.

If atelectasis is detected during or after the attack of pertussis every effort should be made to promote re-expansion of the affected lung, remembering that this has happened six months after the onset of atelectasis (Raia, 1938), and may perhaps occur even up to a year afterwards. In general, surgical removal of a lobe collapsed for less than a year would appear to be inadvisable. When bronchial dilatation remains after re-expansion of a collapsed lobe it is no longer merely compensatory, and so far as is known will be permanent.

Where bronchial dilatation is pronounced, and evidence of infection obvious, surgical intervention is the treatment of choice, for the prognosis in such cases treated medically, as judged by the work of Findlay and Graham (1927), Roles and Todd (1933), Ford (1948), and others, is poor, though antibiotics and chemotherapy can nowadays generally cope with acute exacerbations. On the other hand, it may safely be said that the prognosis of cases with mild bronchial dilatation and little evidence of infection cannot be stated with any confidence, and a prolonged period of observation before advising lobectomy would appear to be indicated.

Summary

Atelectasis was noted radiologically in 43% of 150 pertussis cases, but in general it was slight in extent and of comparatively short duration. Six instances of virtually complete lobar collapse took longer than two months to resolve; in five of these the bronchi contained in the collapsed lobes were shown to be dilated and were seen to revert to their normal calibre when the lobes re-expanded. In the remaining case bronchography was not performed.

In four partially collapsed lobes slight bronchial dilatation was noted, which disappeared when the collapsed lung re-expanded.

In only one case did the attack of pertussis lead to persistent respiratory symptoms. In this case there was evidence of residual bronchial infection in a lobe which had been atelectatic for eighteen weeks. Although the bronchi in this lobe were markedly dilated when it was collapsed, bronchiectasis was not obvious after re-expansion.

The treatment of atelectatic bronchiectasis in pertussis is discussed, and a trial of artificial pneumothorax mentioned.

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SOME ASPECTS OF COLOUR-BLINDNESS*

BY

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In spite of the existence of numerous tests for colour-blindness, each to some extent based on one of the various theories of colour-vision and largely directed to the selection of personnel for the armed Forces and for certain branches of industry, there is no single test which can be regarded as infallible. This fact perhaps reflects more than anything else the inadequacy of colour-vision theories and the insecure basis upon which most of them rest. The occasion is thus opportune for a review of some of the fundamental aspects of the problem and for an evaluation of certain recent observations on colour-blind subjects.

Ever since the days of Thomas Young the necessity for three variables in the mechanism for normal colour-vision has been recognized, and three separately excited pathways from the eye to the brain have been postulated. More than three such pathways are usually considered unnecessary, though at times more have been suggested in order to account for special aspects of colour-vision. Perhaps because of the common experience of mixing pigments, and among physicists and physiologists of mixing lights, in which three primary colours are required in order to match all the spectral colours, it has generally been assumed that each pathway registers the sensation of one of the primary

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