Observations

ON

DRY BRONCHIECTASIS

BY

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(With Special Plate)

Accounts of bronchiectasis which have appeared hitherto have connoted almost invariably the accumulation and decomposition of secretions in the bronchi. Until Sicard and Forestier¹ began to investigate the condition of the bronchial tree by radiography after the introduction of lipiodol there was no certain method by which bronchiectasis could be recognized during life if the characteristic sputa were not present. It is now possible to prove that bronchi may be dilated even when there is not any expectoration. The purpose of this paper is to describe a series of cases in which bronchiectasis of this "dry" or "silent" type was demonstrated, and to discuss the symptoms and signs with which it may be associated, together with problems which arise with regard to aetiology, prognosis, and treatment.

The first account of dry bronchiectasis was given by Bezançon, Weil, Azoulay, and Bernard in 1924.² In a paper entitled *Sèche hémoptoïque de la dilatation des bronchies*, they described two patients in whom recurrent haemoptysis was found by lipiodol radiography to be associated with bronchial dilatation. Neither patient had any sputum or other symptoms apart from haemoptysis. A few other examples of the condition have been described by various writers,^{3 4 5 6 7 8 9 10} and Pinchin and Morlock¹¹ have recorded nine cases in all of which, with one exception, there was recurrent haemoptysis. In his recent paper on bronchiectasis Moll¹² also mentions two dry cases, but gives no details.

The important features of all the cases of which we have found records are given in Appendix I, and they will be mentioned later in the discussion. Nevertheless, it appears that dry bronchiectasis has not been generally recognized, because several recent reviews give it no special mention.^{13 14 15 16 17 18 19 20} We believe, however, that it is comparatively common.

ANALYSIS OF CASES

The present series consists of twenty patients who have come under our personal observation in the last two years. A short account of each patient is given in Appendix II. Reproductions of some typical lipiodol radiograms are given on the special plate. Of the twenty patients, seven were males, and thirteen females. Their ages ranged from 5 to 64 years, and nine were under and eleven over 15 years.

Previous Medical History.—There was a history of measles in childhood in eight, of whooping-cough in eleven, and of bronchitis or bronchopneumonia in thirteen of the twenty patients. In only three did we fail to obtain a history of at least one of these conditions, and together they form by far the most striking feature of the previous history. Recurrent attacks of bronchitis or bronchopneumonia had occurred in no fewer than ten of the twenty patients. A few patients in the series, however, had suffered from other illnesses, which may have contributed to the production of the bronchiectasis. Thus, four gave

a history of influenza, two of lobar pneumonia, three of diphtheria (tracheotomy in one), one of chronic otorrhoea, one of chronic tonsillitis and adenoids, one of double mastoiditis, three of scarlet fever, two of chicken-pox, one of rheumatic fever, and one had been gassed by chlorine during the late war. When there was a previous history of otorrhoea, diphtheria, scarlet fever, or rheumatic fever—except for one instance in each case—there was also a history of either measles, whooping-cough, influenza, or pneumonia. The length of time which elapsed between these various illnesses and the onset of bronchiectasis could not be determined; but in two patients the interval before it was recognized seemed to be only a few months, and in one patient as long as fifty years.

Symptoms.—The symptoms recorded in the few cases of dry bronchiectasis hitherto published have been few, and of these haemoptysis has been the most common, having been noted in nineteen out of thirty patients (see Table). In many of the patients the bronchiectasis was discovered only when investigation for the cause of an unexplained haemoptysis was undertaken. In our series haemorrhage was not the most prominent symptom, and had occurred only in five out of twenty patients. Cough was the commonest symptom, and was present in all but three of our patients. In nine it was slight, in eight definite. Sputum was absent in nine, scanty and occasional in six, up to approximately half a drachm daily in one, and to about two or three drachms daily in four cases. In all those patients where sputum could be obtained for examination tubercle bacilli were not found. The characteristic cumulative fetid expectoration of infected bronchiectasis had not been present at any time in any patient. Of other local symptoms, pain in the chest was noted in eight and dyspnoea in nine cases. Constitutional symptoms were generally mild; they were completely absent in nine patients; in the other eleven loss of weight, or failure to gain weight normally, occurred in nine, night sweats in five, and weakness in two.

Physical Signs.—Abnormal physical signs in the chest were present in all but two patients. One of these had some degree of clubbing of the finger-tips, but this was absent in the other. Clubbing of the fingers was present in four only out of the twenty patients. In all four cough with sputum was present; in three this was scanty, in one up to two drachms daily. Evidence of some degree of constitutional disturbance was shown by a history of loss of weight in three out of the four. Dullness on percussion with weak breath sounds and crepitations were the usual abnormal signs on physical examination of the chest. Dullness was present in seventeen, and varied considerably in degree from a slight impairment to complete loss of normal resonance. Weak breath sounds were noted in thirteen patients and crepitations in thirteen. Bronchial breathing was present in four, granular breathing in one, and pleural friction in one. Definite signs of a cavity were found in one patient only. In all patients where abnormal signs were present they were basal in distribution, and, with three exceptions, corresponded with the situation of the bronchial dilatation. These exceptions occurred in one patient who presented bilateral signs, although bronchiectasis was found to be present on one side only, and in two others in whom unilateral signs accompanied bilateral bronchiectasis.

Radiographic Findings.—Radiographs were taken in every patient both before and after introduction of lipiodol into the bronchi. In nineteen the bronchiectasis present was considerable; in the remaining patient, a child of 6 years, there was probably some bronchial dilatation at both bases. The left base was affected in nine patients,

the right in five, and both bases in six. In fourteen the bronchial dilatation was cylindrical or fusiform, in six it was saccular.

DISCUSSION

The fact that we have been able to collect twenty cases of dry bronchiectasis in about two years strongly suggests that the condition is fairly common. The few references in the literature give an erroneous idea of its frequency. Most of the cases described by previous writers have been investigated because of haemoptysis, and this has come to be regarded as the commonest symptom of the condition. In our series haemoptysis had been infrequent, and this suggests that when investigation is made because of symptoms other than haemoptysis, dry bronchiectasis will be found in many patients in whom it would not have been recognized otherwise. It is not improbable that it may prove to be commoner than the fetid type of the disease.

The important features of the cases previously recorded (Appendix I) and of our own series (Appendix II) are given in the accompanying table. This includes in all fifty patients from which to construct a clinical picture of the disease.

Main Features of Cases	30 Cases Recorded in Literature	20 Cases Recorded in Present Paper	Total 50 Cases
Males	14	7	21
Females	16	13	29
Age	8 to 60 years	5 to 61 years	5 to 64 years
Previous medical history:			
Bronchitis or bronchopneumonia	17	13	30
Measles	5	8	13
Whooping-cough	6	11	17
Influenza	7	4	11
Lobar pneumonia	3	2	5
Symptoms:			
Cough	16	17	33
Sputum	10	11	21
Haemoptysis	19	5	24
Pain	1	8	9
Dyspnoea	2	9	11
Constitutional symptoms ...	5	11	16
Physical signs:			
Clubbing	0	4	4
Dullness	18	17	35
Weak breath sounds	6	13	19
Bronchial breath sounds ...	8	4	12
Pleural friction	2	1	3
Crepitations	14	13	27
Signs of cavity	5	1	6
Radiograph:			
Bronchiectasis (Right)	13 (12 basal)	5 (all basal)	18
Left	11 (all ")	9 (")	20
Both	5 (all ")	6 (")	11
	(1 not stated)	(1 not stated)	
Ditto { Cylindrical or fusiform	8	14	22
{ Saccular or ampullary	22	6	28

Both sexes are equally affected and the condition may be met with at any age, though it is most commonly recognized during late childhood. Bronchopneumonia is the most important antecedent, and very often has been recurrent. In the majority of patients it has followed either measles, whooping-cough, or influenza during childhood. Other acute infections in the nose, throat, bronchi, or lungs are recorded less often in the previous history. Whatever the antecedent illness, the patient is generally left with some residual local symptom such as a dry cough, less commonly recurrent haemoptysis, and occasionally pain in the chest. This insidious onset is followed by an equally insidious course that may for long periods be almost free from any symptoms, or be disturbed from

time to time by recurrent acute bronchitis or bronchopneumonia. Apart from these acute infections, though cough is frequent, sputum is scanty or entirely absent. If present it is not fetid. Haemoptysis, should it occur, is not uncommonly copious and repeated. Constitutional disturbance is almost always slight, and clubbing of the finger-tips is found only occasionally. The physical signs in the chest are generally those of some increase in density, with slight contraction of the underlying lung tissue, but abnormal signs may be entirely absent. Evidence of gross fibrosis or cavity formation is exceptional. Radiographic examination is essential for diagnosis, and requires previous introduction of lipiodol into the bronchial tree in order to demonstrate satisfactorily the bronchial dilatation. Three problems arise for consideration—its origin, prognosis, and treatment.

Aetiology

In none of the patients recorded in the present paper has the bronchiectasis been of the congenital type recently described from the radiological aspect by Möller,²¹ Gibson,²² and Dethmers,²³ or of the so-called atelectatic type described by Sparks²⁴ and others. Apart from these two causes, bronchiectasis has been ascribed in the past to one or other of three factors—raised intrabronchial pressure during forced expiratory efforts, inflammatory changes weakening the bronchial wall, or to fibrous traction. It is not necessary to review here the evidence for and against each of these views, except in so far as it applies to the dry variety of the disease. Here the comparative slowness of the cough and absence of much expiratory effort strongly suggest that intermittent increase in the intrabronchial pressure is not concerned in producing bronchial dilatation during the chronic stage of the disease. Moreover, even though forced expiratory efforts certainly occurred during the antecedent acute pulmonary infections, it is doubtful whether these played any part in producing the dilatation, because, as pointed out by Hedblom,¹⁹ in forced expiration, as in coughing, the force which raises the intrabronchial pressure is applied to the outside of the bronchi.

Again, in these patients there is no evidence of chronic bronchitis, and usually little evidence of pulmonary fibrosis. Moll¹² in his recent paper held that bronchial dilatation is mainly due to inflammatory processes weakening the bronchial wall. This explanation was confined to "wet" cases, and he pointed out that stasis of secretions would not account for "dry" bronchiectasis where this factor is absent. Chronic bronchial infection can therefore be excluded as a constant aetiological factor. The third suggested explanation—fibrous traction upon the bronchi—may sometimes be concerned where evidence of contraction of lung tissue is found; but often this is absent, and radiography does not consistently reveal either an increased density in the affected region or displacement of the mediastinal contents or diaphragm. It seems, therefore, that fibrous traction must be excluded as a common cause of the bronchial dilatation.

The evidence suggests that some other explanation must be found for the production of the dry variety of the disease. We believe that the bronchial dilatation is due to the action of inspiratory efforts. The act of inspiration increases the capacity of the thorax. In health the lung expands to prevent a vacuum, the air entering the alveoli freely. If, however, the air cannot enter the alveoli as the thorax enlarges, one of four things must happen if the visceral pleura remains intact; either the bronchi must dilate, the mediastinum must be drawn over to the affected side, the diaphragm on that side must rise, or a vacuum be produced in the pleural space. The mechanical difficulty is often overcome by a diminished expansion of the chest such as is met with in pneumonia. Forced inspiratory movements commonly occur when

excessive secretion blocks the finer bronchioles in acute bronchiolitis or bronchopneumonia, and in this event the whole force of costal inspiration would be exerted to produce bronchial dilatation unless the mediastinum were drawn over to the affected side or the diaphragm became elevated during each inspiration. Previous fixation of the mediastinum would thus increase the mechanical difficulty and bronchial dilatation be likely to occur. Again, if interference with alveolar expansion was bilateral rather than confined to one side only, compensatory movement of the mediastinum would be much less, because inspiratory traction on the mediastinum would be bilateral. The probability of the bronchi undergoing dilatation in order to prevent a threatened vacuum in the pleura would then be greater and a more or less acute bronchiectasis might develop rapidly. This is very commonly seen at necropsy in such patients. When attacks of bronchiolitis or bronchopneumonia occur frequently the larger bronchi would undergo this tendency to dilatation on each occasion. The likelihood of complete return to the normal size in the quiescent intervals would become less likely with each succeeding attack because of the abnormal stretching of the musculature and elastic tissue.

Prognosis and Treatment

Two main factors have to be considered in the prognosis and treatment—the risk of sepsis ensuing and “wet” bronchiectasis developing, and also the risk of serious haemoptysis. Present knowledge does not allow of a final expression of opinion upon either of these risks. One patient recorded by Bezançon, Weil, Azoulay, and Bernard² developed copious fetid expectoration following bronchopneumonia while under observation (Case 2, Appendix I), and one of Reinberg’s⁴ patients had copious foul sputum when seen three and a half years later (Case 7, Appendix I). None of the other cases described in the literature and none in our own series have shown this feature. Of the cases recorded by others, one of Bezançon, Weil, Azoulay, and Bernard’s patients had a probable duration of eighteen years dating from whooping-cough in infancy (Case 1, Appendix I); one of Reinberg’s patients had a probable duration of fifteen years (Case 4, Appendix I); another eighteen years (Case 5, Appendix I); another twenty years (Case 6, Appendix I); another probably over twenty years (Case 8, Appendix I); and another five years (Case 9, Appendix I); one of the cases recorded by Pinchin and Morlock¹¹ had a probable duration of twelve years (Case 14, Appendix I), and another thirteen years (Case 19, Appendix I); one of Ihre’s¹⁰ patients had a probable duration of approximately forty years (Case 23, Appendix I), and another at least eleven years (Case 25, Appendix I).

Of our patients one (Case 7, Appendix II) had a probable duration of forty-seven years, another (Case 14, Appendix II) approximately thirty years, another (Case 19, Appendix II) twenty to twenty-five years, another (Case 13, Appendix II) fifteen years, another (Case 10, Appendix II) twelve years, two others (Cases 2 and 17, Appendix II) eleven years, and another (Case 3, Appendix II) five years. It is true that in our cases the period of observation has been at most two years, yet in none of these eighteen patients had sepsis supervened, though in every case there is reasonable ground for supposing that bronchiectasis had been present for many years. These records suggest that the risk of sepsis is not very great. Conceivably the typical septic type of the disease may follow a dry bronchiectasis or be septic from the start. If the former sequence be common, then the risk of sepsis in the dry form must be weighed carefully. But if it be uncommon, then the prognosis and management of the two types of the disease must be different. If the risk of sepsis is slight, conservative medical treatment is justifi-

able, but if the risk is considerable then phrenic evulsion or lobectomy should be considered as soon as the condition is recognized, seeing that surgical treatment used prophylactically in the absence of sepsis has far greater opportunity of effecting radical cure than if the decision is postponed until sepsis has supervened.

The risk of haemoptysis would appear to be greater than the risk of sepsis, though it is hardly so frequent as previous writers have held. When it occurs it is usually recurrent, and may be serious enough to endanger life. Both these considerations point to the advisability of surgical procedures if haemoptysis supervenes. Artificial pneumothorax may serve as a temporary measure, but the practical difficulty of maintaining refills makes it unsuitable as a permanent treatment. Moreover, pleural adhesions may entirely prevent the establishment of a satisfactory pulmonary collapse. Hence, although lobectomy does not seem to have been undertaken in the recorded cases, we believe that it should be advised in unilateral affections. The immediate mortality of the operation in skilled hands has become low when sepsis is absent, and the patient is freed permanently from the risk of future danger.

One point in favour of conservative treatment when symptoms are slight is certain evidence which goes to show that a “dry” bronchiectasis may sometimes disappear with spontaneous return of the tubes towards their normal size. Ochsner and Nesbit⁶ noticed a considerable improvement in the degree of bronchial dilatation in one of their cases (Case 2, Appendix I) when a further lipiodol radiograph was taken after two months’ interval. Similar improvement was found in two of our patients. In Case 5 (Appendix II) slight dilatation of the basal tubes, though definite at the first examination, was not seen two years later, and in Case 12 (Appendix II) a fairly well marked degree of cylindrical dilatation at both bases had practically disappeared in eight months. The explanation which we have given to account for the condition would suggest that this sequence of events might not be uncommon, especially in patients seen shortly after an attack of bronchitis or bronchopneumonia. How often improvement of this kind occurs, how complete it may be, and how great a degree of bronchial dilatation may eventually disappear are problems which invite further investigation.

SUMMARY AND CONCLUSIONS

1. Twenty cases of dry bronchiectasis are described, and the opinion is expressed that the condition is comparatively common.

2. Bronchopneumonia, usually following measles, whooping-cough, or influenza, during childhood or adolescence, is the commonest antecedent condition.

3. Dry cough is the commonest symptom. Haemoptysis is not so frequent as the cases previously recorded would suggest.

4. The condition is usually basal, and abnormal physical signs—dullness, weak breath sounds, and crepitations—often serve to indicate the affected area, though they may be entirely absent. Frank signs of cavitation are exceptional.

5. Lipiodol radiography is the only certain means of diagnosis.

6. The explanation of dry bronchiectasis seems to be that if, for any reason, air does not enter the alveoli during inspiration the force of the inspiratory effort is transmitted to the bronchi to which the air has access.

7. Treatment is governed by two main factors, the risk of sepsis and of haemoptysis. Further information is needed, but the suggestion is made that lobectomy should be considered in each case.

This investigation was made while one of us (J. C. H.) worked as medical first assistant to the London Hospital and as Gillson scholar to the Society of Apothecaries.

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APPENDIX I*

The following cases of dry bronchiectasis are recorded in the literature.

Case 1.²—Male, aged 23. Whooping-cough at 5. Adenoids. Recurrent haemoptysis for four years. *Signs*: Dullness and rales right chest posteriorly. *Radiograph*: Right ampullary bronchiectasis. This patient also had aortic incompetence and (?) congenital syphilis.

Case 2.²—Female, aged 38. Bronchitis at 9-10. Pleurisy at 29. Recurrent haemoptysis for eight years. Weakness. *Signs*: Fibrosis with excavation left lung. *Radiograph*: Left bronchiectasis (fourteen cavities). Also congenital syphilis. *Result*: Developed influenzal bronchopneumonia in hospital. Subsequent course of infected bronchiectasis.

Case 3.³—Male, aged 23. No pulmonary disease. Blow on right chest followed by haemoptysis next day. Recurrent severe haemoptysis. *Signs*: Rales right base. Bronchial breathing and crepitations left apex. *Radiograph*: Ampullary bronchiectasis, right base posteriorly. Also chronic pulmonary tuberculosis, quiescent left Zone I. Right artificial pneumothorax arrested haemoptysis.

Case 4.⁴—Male, aged 60. Bronchopneumonia at 45. Morning cough and sputum from 45 to 47. Nil since, except recent dysphagia. *Signs*: Slight emphysema. *Radiograph*: Right and left multiple saccular bronchiectases.

Case 5.⁴—Male, aged 39. Left pneumonia at 21. Repeated haemoptyses. *Signs*: Fibrosis and excavation left lung. *Radiograph*: Cylindrical bronchiectasis left main bronchus and saccular dilatation of branches.

Case 6.⁴—Male, aged 28. Pneumonia at 8. Recurrent haemoptysis from 8 to 13, and again from 23 on. *Signs*: Poor movements and crepitations right base. *Radiograph*: Multiple saccular bronchiectases right lower lobe.

Case 7.⁴—Female, aged 36. Pneumonia at 28. Periodic cough, fever, rigors, and pain left chest for few months after 28. Recurrent haemoptysis since one year after pneumonia. Febrile symptoms. *Signs*: Dullness and crepitations left base; crepitations right base. *Radiograph*: Cylindrical and varicose bronchiectasis left lower lobe. Cylindrical dilatation right main bronchi. *Result*: Seen three and a half years later—copious foul sputum. Left bronchiectasis worse.

Case 8.⁴—Female, aged 30. No previous illnesses recorded. Dry cough and occasional recurrent slight haemoptysis since childhood. *Signs*: Dullness and rales both bases. Right pleural friction. *Radiograph*: Right and left basal saccular bronchiectasis.

Case 9.⁴—Female, aged 28. Influenzal pneumonia and right empyema at 23. Recurrent haemoptysis for five years. Loss of weight. *Signs*: Dullness, weak breath sounds, and rales right base. *Radiograph*: Bronchiectatic cavity right lower lobe.

Case 10.⁶—Female, aged 25. Recent bronchitis. Cough following bronchitis. *Signs*: None given. *Radiograph*: Saccular bronchiectasis left and right lower lobes.

Case 11.⁶—Female, aged 16. Chronic bronchitis for years. *Signs*: None given. *Radiograph*: Fusiform and saccular bronchiectasis right lower lobe. Lipiodol repeated in two months; dilatation much less.

Case 12.⁶—Male, aged 49. Influenza six months previously, followed by pneumonia and left empyema. Cough and slight muco-purulent sputum for six months. *Signs*: None given. *Radiograph*: Left saccular bronchiectasis.

Case 13.⁷—Male, aged 23. No previous illnesses. Large haemoptysis. *Signs*: Few moist sounds at right base. *Radiograph*: Right basal bronchiectasis.

Case 14.¹¹—Female, aged 20. Pneumonia at 8. Cough, slight sputum. *Signs*: Dullness and weak breath sounds left base. *Radiograph*: Cylindrical and saccular bronchiectasis left base.

Case 15.¹¹—Female, aged 28. Influenza at 20. Haemoptysis at 26, repeated since. *Signs*: None. *Radiograph*: Glove-finger bronchiectasis (? site).

Case 16.¹¹—Female, aged 14. Pneumonia at 12. Cough, slight sputum. Haemoptysis at 13½. *Signs*: Dullness and weak breath sounds right base. *Radiograph*: Well-marked bronchiectasis of right base.

Case 17.¹¹—Male, aged 51. Empyema at 45. Slight cough and repeated haemoptysis for two months. *Signs*: Emphysema. Bronchial breathing right base. *Radiograph*: Saccular bronchiectasis right base.

Case 18.¹¹—Male, aged 50. Subphrenic abscess and empyema at 30. Haemoptysis at 48 and 49½. *Signs*: Crepitations. *Radiograph*: Cavity seen through heart shadow.

Case 19.¹¹—Female, aged 17. Whooping-cough at 4. Bronchitis since. Recurrent haemoptysis for four years. *Signs*: Dullness and crepitations at left base. *Radiograph*: Left basal bronchiectasis.

Case 20.¹¹—Female, aged 31. Pneumonia at 17, with empyema. Haemoptysis at 30. Slight sputum since. *Signs*: Fibrosis left base. *Radiograph*: Cylindrical bronchiectasis left base.

Case 21.¹¹—Female, aged 40. Haemoptysis, occasional sputum. *Signs*: Bronchitis. *Radiograph*: Bronchiectasis left base.

Case 22.¹¹—Female, aged 28. Influenza at 27. Recurrent haemoptysis and slight sputum for three months. *Signs*: Dullness and weak breath sounds right upper lobe. *Radiograph*: Dilated bronchi right upper lobe.

Case 23.¹⁰—Female, aged 53. Whooping-cough and measles as a child. (?) Pulmonary tuberculosis at 27 and 30, right Zones I, II, and III. Periodic slight cough for a few years. *Signs*: Fibrosis and excavation right lung. *Radiograph*: Multiple "cystic" bronchiectasis right lung. Also high blood pressure. Probably atelectatic bronchiectasis (? congenital.)

Case 24.¹⁰—Female, ? age. Pneumonia at 3 months (? left), and on seven subsequent occasions. Whooping-cough and measles, with pneumonia. Influenza. Slight cough and sputum. *Signs*: Dullness and cavity signs, with rales at left base. *Radiograph*: Saccular bronchiectasis left base.

Case 25.¹⁰—Male, aged 56. (?) pneumonia at 27 and at 43, following influenza; chronic bronchitis, with two acute relapses at 44 and 45. Tuberculous epididymis at 54. Cough with paroxysmal dyspnoea and sputum for four years. *Signs*: Excavations right Zone II. *Radiograph*: Saccular bronchiectasis right lung.

Case 26.⁹—Male, aged 8 years 7 months. Whooping-cough and bronchopneumonia at 5. Bronchopneumonia at 6. Measles and bronchopneumonia at 7. Dry cough since whooping-cough, and small haemoptysis six months ago. *Signs*: Dullness, weak breath sounds, and few crepitations right axilla. Bronchial breathing right interscapular region. *Radiograph*: Right basal bronchiectasis.

Case 27.⁹—Male, aged 9. Bronchitis and influenza at 2. Relapse in three months and recurrent attacks of bronchitis since. Cough following bronchitis. *Signs*: Dullness, bronchial breathing, crepitations, and pleural rub left base. Pleural rub and rough breath sounds right base. *Radiograph*: Saccular and ampullary bronchiectasis left base.

Case 28.⁹—Male, aged 10. Bronchitis at 11 months. Whooping-cough at 3, and bronchopneumonia. Measles at 4. Recurrent bronchitis—serious attack at 9. Bronchitis with haemoptysis at 9. *Signs*: Dullness, weak breath sounds anteriorly, and bronchial breathing posteriorly over right chest. *Radiograph*: Right basal saccular bronchiectasis.

Case 29.⁹—Male, aged 8. Bronchopneumonia at 14 months. Frequent bronchitis since. Bronchopneumonia again at 3. Occasional cough for one year. Under weight. *Signs*: Dullness, bronchial breathing, and rales left chest. *Radiograph*: Left basal saccular bronchiectasis.

Case 30.⁹—Female, aged 11. Measles and bronchopneumonia at 2. Frequent bronchitis. Mastoid at 10 years 9 months. Persistent dry cough. Under weight. *Signs*: Bronchial breathing and rales left chest. *Radiograph*: Right ampullary bronchiectasis, basal. Left cylindrical dilatation of main bronchi to lower lobe.

* Additional cases to those reported here are recorded in two papers^{5, 8} to which we have not had access.

APPENDIX II*

The following are the brief records of our own cases.

Case 1.—Male, aged 6. Admitted for acute bronchitis. (L. H., 31683/1931.) Measles and bronchopneumonia at 18 months. Whooping-cough and pneumonia at 3. Bronchitis each winter since. Dry cough since measles. *Signs:* Dullness, bronchial breath sounds, and rales at both bases. *Radiograph:* Bilateral cylindrical basal bronchiectasis. *Result:* Bronchitis subsided rapidly. Discharged to convalescent home with persistent slight dry cough, the physical signs remaining unchanged.

Case 2.—Female, aged 15. A factory worker. (Br. H., 5710.) Measles in infancy, bronchitis and whooping-cough at 4 years. Recurrent bronchitis since. Cough, scanty sputum, and loss of weight for two weeks two months prior to observation. *Signs:* Dullness and crepitations at left base. *Radiograph:* Saccular bronchiectasis at left base.

Case 3.—Female, aged 24. (Br. H., 4852.) Perfume finisher. Pneumonia at 3. Further attack after influenza at 19. Came under observation for a slight haemoptysis of about half an ounce. *Signs:* Dullness, granular breath sounds, and crepitations at left base. *Radiograph:* Cylindrical bronchiectasis of some branches of left descending bronchus.

Case 4.—Female, aged 32. (L. H., 41897/1932.) Housewife. Bronchopneumonia, following influenza, at 18. Left pneumonia complicated by empyema at 30. Three aspirations, no open drainage. Prolonged convalescence. Dry cough since, and recently slight mucoid or muco-purulent sputum. Recurrent left pleuritis pain for fifteen months, and recurrent slight haemoptysis for four months, with loss of weight, lassitude, and night sweats. *Signs:* Fibrosis of left base. *Radiograph:* Extensive cylindrical bronchiectasis behind the heart shadow (Fig. 1). *Result:* No change during six months' observation. Lobectomy advised, but refused by patient.

Case 5.—Male, aged 6. (Br. H., 2396.) Six attacks of pneumonia, the last attack following influenza shortly before admission. Double mastoidectomy at 3. Whooping-cough and scarlet fever, followed by otorrhoea at 4. At this time a lipiodol radiograph suggested slight dilatation of the bronchi at both bases. Subsequently persistent slight cough and recurrent pain in right chest, loss of weight, night sweats, and occasional vomiting. *Signs:* Dullness at both bases. A further lipiodol radiograph showed no definite bronchial dilatation.

Case 6.—Male, aged 18, traveller. (Br. H., 6500.) Scarlet fever in childhood and bronchopneumonia at 18. Persistent nocturnal cough since, scanty yellow sputum, streaks of haemoptysis, and loss of weight. *Signs:* Moderate clubbing of the finger-tips. No signs in chest. *Radiograph:* Cylindrical bronchiectasis of mesial branches of left descending bronchus.

Case 7.—Female, aged 64, secretary. (L. H., 40522/1932.) Measles and pleurisy at 14. Whooping-cough at 17. Sent to Davos because of "weak lungs" at 19. Since good health, except for occasional recurrent cough, with moderate sputum. Three months before observation, attack of "influenza," with severe cough and expectoration for three weeks. Weakness and anorexia since, but no further cough or sputum. *Signs:* Slight dullness, with crackling rales at left base. *Radiograph:* Left basal bronchiectasis of cylindrical type (Fig. 2). *Result:* Well under observation. Special treatment not advised.

Case 8.—Male, aged 6. (Br. H., 6596.) Double pneumonia at 3 years. Whooping-cough at 4. For four years attack of bronchitis each year. Slight persisting dry cough, shortness of breath, loss of weight, and night sweats. *Signs:* Dullness and crepitations at right base. *Radiograph:* Probable dilatation of bronchi at right base. *Result:* No change during short period of observation; right phrenic evulsion.

Case 9.—Female, aged 6. (Br. H., 2824.) Whooping-cough and double pneumonia at 2. Several further attacks of acute pneumonia since. Persistent slight cough, with frothy sputum (about half drachm daily), shortness of breath, night sweats, and one attack of left pleuritic pain. *Signs:* Dullness and weak breath sounds at right base. *Radiograph:* Bilateral basal bronchiectasis.

Case 10.—Female, aged 24, domestic servant. (Br. H., 6565.) Previous illnesses: nasal catarrh with otorrhoea and influenza at 12. Slight nocturnal cough, scanty mucoid sputum, and shortness of breath from 21. *Signs:* Dullness and weak breath sounds at right base. *Radiograph:* Saccular bronchiectasis of right descending bronchi (Fig. 3).

Case 11.—Female, aged 8. (Br. H., 1693.) Bronchitis and pneumonia at 2 years. Whooping-cough and pneumonia at 5. Also chicken-pox. Since last attack of pneumonia persistent cough, with loss of weight. *Signs:* Dullness, bronchial breath sounds, and crepitations at both bases. *Radiograph:* Extensive bronchiectasis at both bases, with multiple small cavities on right side.

Case 12.—Female, aged 13. (Br. H., 6938.) Marasmus at 2 months. Double pneumonia at 1, 2, and 3 years. Measles at 5. Diphtheria and whooping-cough at 7. Colds since; (?) asthmatic attacks at night. *Signs:* Dullness and weak breath sounds at right base. *Radiograph:* Cylindrical dilatation of basal bronchi at both bases. *Result:* Sputum up to 2 drachms in hospital. T.B. negative three times. To convalescent home. Eight months later occasional slight cough, no sputum. A second lipiodol radiograph showed the bronchi to be almost normal.

Case 13.—Male, aged 16, messenger. (Br. H., 4916.) Slight nocturnal cough with scanty sputum as long as he could remember, shortness of breath, and slight loss of weight. Whooping-cough, diphtheria, and measles in infancy. *Signs:* Clubbing of the fingers, dullness and crepitations at both bases. *Radiograph:* Bilateral basal bronchiectasis, with sacculcation.

Case 14.—Female, aged 32, street trader. (L. H., 41826/1931.) Since whooping-cough in infancy, cough with occasional slight sputum. Slight recurrent haemoptysis and pain in left chest for two months before admission to hospital. *Signs:* Dullness, weak breath sounds, and crepitations over the left base. *Radiograph:* Left basal bronchiectasis. *Result:* Haemoptysis not repeated while under observation. Special treatment was not advised in view of slight symptoms.

Case 15.—Female, aged 10. (Br. H., 5959.) Measles and whooping-cough during infancy. Mumps at 4. Enlarged tonsils and adenoids at 5. Dry cough from 2, with slight mucoid sputum since pneumonia at 8. Recurrent pain in left chest, with loss of weight and night sweats. *Signs:* Clubbing of fingers, dullness, diminished breath sounds, and crepitations over the left base. *Radiograph:* Left basal bronchiectasis.

Case 16.—Female, aged 23. (Br. H., 6689.) Only previous illness an attack of rheumatic fever a few months before admission to hospital. Attack of right pleuritic pain four weeks before admission. No abnormal physical signs. *Radiograph:* Considerable dilatation, with some sacculcation of the branches of the right descending bronchus.

Case 17.—Male, aged 33, fruit porter. (L. H., 30734/1932.) Admitted for attack of acute basal bronchopneumonia. Gassed by chlorine in 1918. Pneumonia 1921 for three months. Since occasional dry cough. Signs of consolidation of left lower lobe on admission to hospital. Pneumonia subsided in two weeks, but dullness, with broncho-vesicular breath sounds and crepitations, persisted at left base. *Radiograph:* Cylindrical bronchiectasis of posterior branches of left descending bronchus. Also slight dilatation of some of anterior branches.

Case 18.—Male, aged 7. (L. H., 30436/1931.) Measles and bronchitis at 4. Pneumonia at 6. Since dry cough and shortness of breath. *Signs:* Persistent dullness, weak breath sounds, and scattered rhonchi at right base during one year's observation. *Radiograph:* Right basal bronchiectasis of smaller bronchi, especially posteriorly.

Case 19.—Female, aged 27. (Br. H., 5057.) Diphtheria requiring tracheotomy at 2 years. Scarlet fever at 12. Said to have had a cough "always," with occasional scanty sputum. Periodic haemoptysis and shortness of breath since 21. *Signs:* Moderate clubbing of fingers, dullness, and signs of excavation at left base. *Radiograph:* Considerable dilatation with sacculcation of branches of left descending bronchus. *Result:* This patient had a left phrenic evulsion done.

Case 20.—Female, aged 8. (L. H., 40250/1932.) Chicken-pox and measles as an infant. Attacks of winter bronchitis followed by pneumonia for three to four years. Came under observation during one of these attacks, which rapidly

* L. H. = London Hospital; Br. H. = Brompton Hospital.

subsided. Sputum negative for tubercle bacilli. Dry cough, and had failure to gain weight between these attacks. *Signs*. Undernourished, and dullness, with weak breath sounds and crepitations at right base, and weak breath sounds and crepitations at left base. *Radiograph*: Tubular bronchiectasis of the right lower bronchi (Fig. 4). *Result*: Bronchitis rapidly subsided. Discharged to convalescent home, with persistent slight dry cough. No change in physical signs.

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ENDOMETRIOMATA: THE FIVE CLINICAL TYPES*

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(With Special Plate)

I deal here with a condition which is very commonly found in women at or before operation, and one which is very rarely diagnosed or even thought of by practitioners. I refer to endometriomata, tumours which have only come into prominence during the last ten years. I have chosen this subject because my private and hospital records show that a state of endometriosis existed in 8.9 per cent. of my last consecutive 1,000 abdominal operation cases, which proves that this pathological condition is of sufficient frequency to warrant notice. It may be of interest to state that many of these had been treated for months as cases of chronic tubo-ovarian disease, others as slowly growing innocuous tumours of the uterus, others as cases of intractable dysmenorrhoea, and still others as dyspareunia, lumbago, appendicular colic, piles, and *B. coli* infections.

PATHOLOGY

In 1921 Sampson advanced the work of Cullen and Lockyer by demonstrating that tumours frequently found in the ovary, uterus, and broad ligament contained

cells exactly resembling those of the mucous membrane of the uterus. From this finding he evolved the theory that at times menstrual blood regurgitates through the tubes into the pelvic cavity. This retrogressing fluid carries with it particles of endometrium, which implant and graft themselves upon pelvic structures and then tend to invade particular viscera, setting up intense tissue reaction. These aberrant endometrial cells, subject to the influence of hormones from the hypophysis and ovary, retain their physiological capacity of cystic proliferation, secretion, and necrosis, with the result that cysts form, varying in size from a partridge's egg to that of a duck's egg, and surrounded by dense fibrous tissue. The cysts, small or large, contain tarry, chocolate-like material. Such an entertaining theory compels immediate criticism, for there would seem no reason why dying cells of the cast-off endometrium should thrive and multiply, though it must be admitted that such cells falling into the stoma of a recently ruptured Graafian follicle would have an ideal medium for tissue culture. So far, however, experiments along these lines in monkeys have failed to show that desquamating menstrual epithelium can grow upon the peritoneum; nor can such a theory explain how endometriomata can occur at the umbilicus or in the canal of Nuck, or why they are found upon the large intestine and not on the walls of the small gut.

I think I have seen and operated upon endometriomata in every site described above, and it is for that reason—though freely admitting the possibility of Sampson's theory—that I prefer to accept the assumption that these tumours originate from metaplasia of the cells of the peritoneum. The lining mucous membrane of all parts of the Müllerian canal (tubes, uterus, and vagina), as well as the germinal epithelium of the ovary and the pelvic peritoneum, is derived from the same parent tissue—that is, the coelomic epithelium; for it will be recalled that an invagination of this structure gives rise to the Müllerian mucous membrane, which undergoes further differentiation in its various segments from the fimbriae of the Fallopian tubes to the hymen. If such embryonic differentiation is possible, is it not probable that the germinal epithelium of the ovary and the cells of the pelvic peritoneum may come under the influence of certain stimuli (probably endocrinal) in adult life, and undergo or take part in similar changes, resulting in tumours large or small containing coelomic epithelium which has undergone metamorphosis and now has the structure and function of tubal or endometrial mucous membrane?

Whether to believe in the theory of Sampson or that of metaplasia, or, with others, in the assumption of lymphatic or embolic transference, is of little concern here, for this is a matter upon which, in the present state of our knowledge, it is impossible to be dogmatic.

CLASSIFICATION

Clinically my cases of endometriomata indicate that there are five distinct types:

1. That of a uterine tumour.
2. That of obstruction or invasion.
3. That of a tubo-ovarian mass.
4. The symptomatic type, where symptoms only predominate.
5. The post-operative scar type, which is very rare.

Though these variations are often very clearly differentiated, it is not uncommon to find one or more types present in the same individual.

Type 1.—These tumours grow slowly, and are soft and without a capsule. As a rule they are situated near one cornu, and are associated with profuse menorrhagia, but

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CECIL WALL AND J. CLIFFORD HOYLE: DRY BRONCHIECTASIS

FIG. 1.—Case 4. Cylindrical bronchiectasis of left descending bronch.

FIG. 2.—Case 7. Cylindrical bronchiectasis of left descending bronch.

FIG. 3.—Case 10. Sacular bronchiectasis of right descending bronch.

FIG. 4.—Case 20. Cylindrical bronchiectasis of right descending bronch.