close to the surface of the lung, and is closely surrounded, as you will see, by sound lung tissue. There is no active tuberculosis in the wall or immediately around. This is a healed cavity, absolutely without action. Besides this old lesion there was another small cavity, as well as cicatrices and cretaceous matter in other parts of this lung, and there were cicatrices and cretaceous matter in the other lung. While this was the case, there were scattered throughout both lungs the lesions of a recent acute tuberculosis, without any softening or formation of cavities. Looking closely at the preparation you can see these scattered nodules on the cut surface. The explanation of this recent tuberculosis was to be found in a tuberculous ulceration of the larynx, and the case is illustrative of a class which I believe to be a numerous one. I am constantly meeting with cases in which an old and perhaps a healed tuberculosis of the lung is associated with a recent active tuberculosis without any intermediate stage. There are only these two widely separate stages of the disease. The explanation is generally to be found in a tuberculosis of the larynx.

My belief, as the result of considerable observation, is that a tuberculosis of the lungs, sometimes of inconsiderable extent, will often extend to the larynx, perhaps there also of little consequence for a time. The disease may be recovered from in the lung while it lingers on in the larynx. After a long course in the larynx—as long sometimes as four or five years—a reinfection of the lung may take place. This will occur when the laryngeal lesion becomes extensively ulcerated. The discharges from such ulcers are highly infective, they are loaded with bacilli. It is not to be supposed that the bacilli rise from the ulcers into the air as dust, and are either expired or inspired as such, but in the processes of respiration little fragments of mucus are liable to be carried in and planted in the finer bronchi. In consequence there is a sudden burst of acute tuberculosis to which the patient probably succumbs. In such cases the tuberculosis may be scattered somewhat homogeneously throughout the lungs, or it may be—as I have sometimes seen it—mainly in the central parts, involving the upper parts of the lower lobe and the lower parts of the upper. At any rate, there is no such proclivity to the apex as in ordinary phthisis.

The history of the case before us is illustrative of this class of cases. The patient, a man aged 42, was ill two years before death with cough, spit, and hæmoptysis. The illness was not regarded as serious, and it was accompanied by a bulging of the chest in the left front—a bulging which turned out to be from the cavity shown in the preparation. He recovered from this illness, and was apparently well till six months before death, when pronounced laryngeal symptoms occurred and acute pulmonary phthisis followed. Although there is here no record of laryngeal symptoms in the period between the two illnesses, I think we may infer that the tuberculosis lingered on, and that there was a hoarseness of the voice which the patient thought of no consequence.

The practical inference from this class of cases is that, after a pulmonary attack, even if of a slight nature, a remaining laryngeal disturbance should not be regarded as of trivial importance.

I ought to add that ulceration occurring in the trachea and larger bronchi may produce a somewhat similar reinfection of the lungs, giving rise to the same widely diverging stages of the disease.

What we find in the respiratory organs is sometimes visible elsewhere. We have seen how a tuberculosis of the testicle may travel to the kidneys, and while entirely retrograde in its original seat, may go on advancing in the more recent situation. This was the case in one of the preparations already shown (new preparation, No. 9).

In another of the preparations (IA, 25) we see a healing of the vertebrae, with collapse of two of the bodies, going along with considerable erosion of other bodies from without. The primary seat inside the bone has been evacuated by the emptying out of the necrosed structures, and the disease has been attacking neighbouring structures. In this case the tuberculosis had extended from the local seat to the membranes of the brain and to the blood, there being a tuberculous meningitis and general tuberculosis. The remarkable thing is that the tuberculosis of the vertebrae, which must have lasted for years, seems not to have been suspected during life.

Gentlemen, I have detained you too long. I trust that, as the result of my remarks, you will look more hopefully on tuberculosis as amenable to cure. If about a quarter of the people who die of other diseases show evidences of having been tuberculous, then recovery is not very uncommon.

In all my remarks I have assumed the standpoint of tuberculosis as an infectious disease. This is now the unalterable position of all pathologists. I must say that I see little trace as yet of this attitude on the part of the practitioner and the general public. As I have given expression to my views on this subject in a recent address at the Sanitary Congress in Edinburgh, I will not further expand them here. I should like, however, that the profession would seriously look into their practice in view of the discovery of the tubercle bacillus, and I should favour a discussion on the subject in this Society.

[The preparations referred to in the text are in the museum of the Western Infirmary and are described in the catalogue under the numbers quoted.]

REMARKS

ON

ARRESTED PULMONARY TUBERCULOSIS,¹

*In the Section of Pathology at the Annual Meeting of the
British Medical Association held at Bournemouth,
July, 1891.*

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WHEN I was invited to open a discussion in the Section of Pathology, the subject of tuberculous disease of the lungs seemed naturally to suggest itself as likely to be of interest to those who would meet at Bournemouth. From the programmes of the Sections of Medicine and Therapeutics, it is obvious that the same idea has occurred to others similarly situated; but in this I see no disadvantage; on the contrary, we may well hope that the discussion of so important a subject from various standpoints may serve to bring out clearly the present clinical and pathological conceptions of the disease generally known as "phthisis pulmonalis."

Recent events have certainly rendered it more than ever desirable that our views of the pathology of "phthisis" should be in accord with the results of scientific research, and that our knowledge of the natural history of pulmonary tuberculosis should be accurate, for a false pathology cannot but act as a bar to progress in therapeutics, and ignorance of the course of a disease may lead us to ascribe to remedies results which have been brought about by the unaided resources of Nature. The pathology of phthisis is a well worn subject, and I do not propose to occupy much of your time by its discussion, but some reference must be made to it as a preliminary to a consideration of the arrest of pulmonary tuberculosis, with which we are to-day more immediately concerned.

The discovery of the tubercle bacillus, followed by the proof of its pathogenic properties, has, I imagine, so far as most pathologists are concerned, set at rest the vexed question as to the nature of tubercle, and has at the same time gone far to determine their views as to the pathology of phthisis.

The position of those who hold that phthisis is but one disease, and that it is the result of a tuberculous infection of the lungs, has thus been materially strengthened, and the time has come when we may fairly ask those who still maintain the contrary opinion to bring forward the evidence on which their belief is founded.

One of the chief grounds urged against the acceptance of the above view is the great variety in the lesions of phthisis, and the undoubted fact that in the production of some of them inflammation plays an important part. It is true that

¹ The paper was illustrated by four series of cases; brief abstracts were presented to members attending the Section.

the process which leads to formation of a tubercle may be accompanied by an inflammatory exudation; this is nowhere better seen than in tuberculous infiltration of a serous membrane such as the pleura. We have no difficulty in distinguishing in such a case the specific from the simple inflammatory products. Fine miliary granulations stud the serous membrane, whilst the pleural sac contains serous fluid and thick membranous exudation adheres to each of its layers. Transfer these morbid products to the spongy lungs and it must be obvious that, just as the lymph upon the pleura often obscures the tubercles beneath, so the inflammatory changes therein will sometimes obliterate the specific products.

All will, at any rate, admit that there are a very large number of cases of destructive diseases of the lungs due to the presence of tubercle; with respect to these cases I have a proposition to make. It is simply that they should be termed cases of "pulmonary tuberculosis" or of "tubercle of the lungs," and that for such the term "phthisis" should no longer be used.

For cases, if such there be, of destructive disease of the lungs which can neither be recognised as due to the presence of tubercle, nor referred to any other well-known group, it is possible that "phthisis pulmonalis" may be the most appropriate name; on this point I express no opinion, as during the last six months, since I ceased to use that name for the tuberculous cases, I have not met with any such.

In asking you to discard such a name as "phthisis," sanctioned by long usage, one feels bound to indicate in what direction advantage may be looked for. In the first place, then, we shall bring our nomenclature of all tuberculous affections into line, and, as we now speak of a tuberculous meningitis, pleurisy, or peritonitis, so we shall designate the local manifestation of tuberculosis within the lungs by its proper name of "pulmonary tuberculosis." This change of name is the more to be desired in that, if I read aright "The Nomenclature of Diseases drawn up by the Joint Committee appointed by the Royal College of Physicians, 1885," the term phthisis, standing alone and unqualified, is no longer recognised. On reference to p. 83, Diseases of the Lungs, one finds: "356. Acute pneumonic phthisis. 357. Chronic pneumonic phthisis." "364. Millstone-maker's phthisis," but nowhere "phthisis." Tubercle is to be returned according to the variety, and the part of the body affected. It is true that in the Index, which it is stated on p. xxvi "is in some senses the real nomenclature," phthisis appears alone and in type which indicates that it may be so employed, but "see tubercle of lung" is affixed, indicating, as I read it, that there is but one such disease, and it is directed that the cases are to be registered under the head of tubercle.

I am glad to know that the Medical Registrar of the Middlesex Hospital intends in next year's report to give a separate place to tuberculous disease, as is now given to cancer, and under the heading to place the tuberculous affections of the various organs, as directed in the official nomenclature.

The term "fibroid phthisis" is also no longer officially recognised, "cirrhosis of the lung" having been substituted for it. As originally employed it included both tuberculous and non-tuberculous lesions; for the former I have a name to suggest which I will mention later; for the indurative affections of the lung not of tuberculous origin the terms "chronic pneumonia" or "chronic interstitial pneumonia" appear to me preferable to cirrhosis of the lung, as they do no violence to etymology; there is nothing "yellow" about an indurated lung.

The essentially tuberculous nature of the destructive pulmonary lesions induced by employment in various trades such as that of a knife-grinder, mason, miner, weaver, or potter has been demonstrated too often to need more than a passing reference.

Much has been written on the subject of syphilitic phthisis, but I confess that the existence of a destructive disease of the lung, due to syphilis, has never appeared to me as proved, and a careful examination of many lungs has not, in my case, been repaid by an ability to recognise with certainty a syphilitic lesion. I can, in fact, only recall one case to which the term syphilitic phthisis was even remotely applicable; it was one in which a gumma in one of the main bronchi, whilst in process of softening, opened into a large

branch of the pulmonary artery and caused death by hæmorrhage.

The pathological anatomy of syphilitic lesions of the trachea and main bronchi is too well known to need description.

Another advantage would be the necessary disappearance of the term "the stages of phthisis." Few expressions have done more than this to confuse the minds of students, and to prevent the unfortunate sufferers from the disease from acquiring such an understanding of their malady as their knowledge will allow. It is hardly necessary to insist that the so-called "stages of phthisis" are stages in a pathological process which may be arrested in any one of them, and that they have no necessary connection with the general advance of the disease. But it is difficult to make a patient who has been told that he has a cavity, a condition which he knows to represent the third stage of the disease, believe that he is better off than many who are still in the first or second, whereas we all know that arrest in the stage of cavity may be complete and its duration indefinitely prolonged.

It is never easy for a long word to displace a short one in nomenclature, and it is more than twice as difficult for two, but I am not without hope that this may happen, and that the name which clearly indicates the pathological nature and clinical relationships of the affection may ultimately be adopted in preference to one derived from a symptom which, if it apply to the body, is shared by this disease with many others, and one which has been differently interpreted with each changing phase of pathological opinion.

There are certain varieties of pulmonary tuberculosis for which distinctive names appear to be necessary—variations from the common type which can be recognised clinically as well as pathologically.

1. The term *Pulmonary Tuberculosis* is itself sufficient for the ordinary cases of chronic phthisis, in which, although all the various changes to which tuberculous lesions are liable may be, and often are, represented, no single one is present in such a marked degree as to impress a special character upon the case.

2. *Miliary Tuberculosis of the Lungs* may occur as part of a general tuberculous infiltration, having its starting point in the lungs or elsewhere, or it may be limited to those organs. One of the chief dangers which beset the subjects of "arrested tuberculosis" is a liability to a sudden dissemination of the virus, followed by a rapid formation of miliary tubercle. This is also a very common mode of termination of chronic cases of lung tuberculosis.

3. *Caseous Tuberculosis of the Lungs*.—The special tendency of tuberculous products to undergo caseation is admitted by all, and its occurrence appears to be a measure of the resisting power of the individual, which seems to be at a minimum in those very acute cases termed "florid phthisis" or "galloping consumption." Such cases are characterised by a tendency to rapid caseation and softening of the tuberculous and inflammatory products, and to them the name "caseous tuberculosis of the lungs" is especially applicable. This variety includes cases in which either a whole lobe or the greater part of it is affected (lobar caseous tuberculosis), and others in which the caseous and softening lesions are disseminated throughout one or both lungs (disseminated caseous tuberculosis). The former are very likely to be mistaken for croupous pneumonia, until the unusual degree of emaciation or the absence of a crisis suggests an examination of the sputa, when the discovery of tubercle bacilli reveals the true nature of the disease.

4. *Fibroid Tuberculosis of the Lungs*.—This group includes the tuberculous cases of fibroid phthisis in which there is a special tendency on the part of the tubercles to undergo fibroid transformation. Single granulations, or groups of tubercles, or extensive areas, are found black or slate grey in colour, and of a dense fibroid consistence. The larger masses do not break down, but in their centre fibrous tissue develops, and the special elements atrophy, so that it may be that separate tubercles can with the naked eye be distinguished at the margin only. Around the contracting fibrous lesions the tissue is emphysematous, a compensatory change; a few caseous nodules may be present, but softening and cavity formation are often conspicuously absent. In the indurated areas the pulmonary tissue shows atrophy and fibrous changes. Occasionally this form of lesion appears to result

from the tuberculous infiltration of lung tissue already emphysematous, but more often the latter change is, as above stated, secondary and compensatory.

Fibroid tuberculosis occurs with especial frequency in men of good muscular development, but often with flat chests. Such cases are marked by recurrent hæmorrhages, by their prolonged duration, and by the frequency with which the process undergoes arrest.

I do not include under this heading the very chronic cases characterised by slow destruction, contraction of cavities, and extreme pleural thickening. Such cases I regard as ordinary cases of so-called chronic phthisis, for which the term "pulmonary tuberculosis" appears to be adequate. One not uncommonly sees in the *post-mortem* room lungs presenting in the upper lobe the above appearances, whilst in the lower lobe the mode of formation of such lesions is clearly shown by the presence of tuberculous infiltration, of softening and cavity formation. Such an appearance is shown in Fig. 1.

Fig. 1.

You will observe over the upper lobe the thickened pleura, due to the presence beneath of contracting cavities, which are smooth-walled and in communication with dilated bronchi; the intervening fibrous septa surround the obliterated vessels and bronchi, and represent the remains of atrophied, collapsed, and fibrosed lung tissue; in addition, the interlobar septum is much thickened. In the lower lobe the advancing margin of the disease clearly indicates its tuberculous nature, and the appearances are such as are constantly met with.

It may be useful here to enumerate briefly the various changes which tuberculous products may undergo.

1. If the life of the tubercle be short, it may remain a grey miliary granulation and may be so found on necropsy.
2. It may undergo caseation, either separately or after having coalesced with neighbouring granulations.
3. The caseous mass may soften and be discharged, a vomica being the result.
4. The caseous mass may become surrounded by a fibrous capsule, and may so remain during the life of the individual, who may die of some non-tuberculous disease.
5. Such a mass may undergo cretification.
6. After a varying period local changes may lead to the breaking down of the capsule and the softening and discharge of the contained matter, if still caseous, or its discharge as a calcareous mass, a cavity remaining in either case.
7. In the process of softening, the caseous mass may open up a communication with a blood or lymphatic vessel and absorption of the virus may be followed by acute general or pulmonary tuberculosis. Or in the process of discharge the virus may be inhaled into other portions of the lungs and may there cause a local tuberculosis.
8. The tubercle may undergo fibroid transformation, followed by pigmentation, a process which often spreads to the neighbouring pulmonary tissue. This change may affect a few granulations or by its extent may form a marked feature in the case, in which case the term "fibroid tuberculosis" becomes applicable. The consecutive lesions have already been referred to.

Since the time of Laennec it has been recognised that tuberculous disease of the lung may undergo arrest, although I think that few who have not had extended experience in the *post-mortem* room can have any conception how frequently such an event takes place.

Probably all whose duty it becomes to superintend many necropsies are struck by the common occurrence of lesions at the apices of the lungs, often apart from any disease elsewhere in those organs. Such lesions have been frequently described, and are I believe regarded by most pathologists as evidence of previous tuberculous disease which has undergone arrest. This view, however, is not accepted by some eminent authorities whose opinions are to be treated with great respect, and it is to this point that I desire now especially to direct your attention.

These lesions are various and require a brief enumeration. We meet with:—

1. Pigmented tubercles which have undergone fibrosis.
 2. Fibroid induration, puckering and scarring of the apex, with or without obvious tubercles.
 3. Areas of caseation, often surrounded by a fibrous capsule and deeply-pigmented tissue, the latter sometimes presenting obvious tubercles.
 4. Cretified masses similarly situated.
 5. Cavities of the size of a hazel nut with smooth walls, filled with pigmented material of caseous consistence and not in communication with a bronchus. Larger thick-walled cavities are generally obviously tuberculous in origin.
- With the above lesions certain others are generally associated and are clearly consecutive to them. Such are:—
6. Scarring and puckering of the pleura.
 7. Adhesion of the pleura, with areas of extreme thickening, clearly the result of the separation of the visceral and parietal layers by the contraction of an intrapulmonary lesion.

This change will be met with in various stages: (a) Fine filamentous adhesions, with many vessels passing between the two layers of the serous membrane. (b) Contraction commencing, separation of the surfaces, and serous exudation into the interstices of the adhesions; this fluid can be squeezed out after section. (c) The exudation undergoing fibrillation and no longer fluid. (d) The exudation organised and of a varying degree of consistence according to its age, ultimately becoming almost cartilaginous in density, and forming a wedge with its apex towards the lung.

The cases in which the apex of the lung or the whole upper lobe is covered by a dense and thick fibrous investment are usually so obviously tuberculous that no question can arise as to their nature.

8. Thickening of the interlobar septa, from contraction of neighbouring disease within the lung.
9. Dilatation of the bronchi. This is in nearly all cases a compensatory change.
10. Surface emphysema, bullæ of various sizes being situated around the contracted lesions.
11. Compensatory enlargement of other parts of the same lung or of the opposite lung.
12. Obliteration of the intercostal spaces.
13. Displacement of organs.
14. Caseation of bronchial glands.

Patients in whom lesions such as the above are found may have died of a disease which has not the most remote connection with tubercle, or they may have succumbed to acute tuberculosis or to a non-pulmonary tuberculous disease, or the disease in the lungs may have again become active, and death have been due to the gradual destruction of those organs. I have searched the *post-mortem* reports of the Middlesex Hospital from 1879 to 1886, during which period I held the appointment of pathological registrar, and have extracted from them records of many such cases. It is difficult to place the evidence satisfactorily before you except in print, and I have therefore distributed copies of the abstracts of the *post-mortem* notes. The cases are arranged in various series and in the order of their occurrence. At the head of each case is an abstract of the general pathological conditions present, then follows an account of those appearances in the lungs which I regard as evidence of previous tuberculosis. In some cases I have added a few remarks. The descriptions have necessarily been abbreviated but no other alteration has been made in the wording. Some of the examina-

tions were made by my colleagues Drs. Coupland, Finlay, Pringle, Pasteur, the late Dr. Lyall, Mr. W. Roger Williams, Mr. J. B. Sutton, and Mr. C. L. Hudson; to such cases I have appended their initials.

TABLE I.—*Middlesex Hospital, 1879-1886.*

Number of necropsies, 1,943: obsolete tubercle in lungs, 177=9 per cent.

Males	110	Right lung	35
Females	67	Left lung	36
		Both lungs	106
Cause of Death:			
Cancer	73		
Sarcoma	5		
Tuberculous affections, excluding pulmonary tuberculosis	Not included		
Empysema and bronchitis	See Series II & III		
Pneumonia	8		
Pulmonary oedema	7		
Specific fevers	1		
Caries and necrosis of bones	3		
Wounds, injuries, poisons	2		
Diseases of the brain and membranes	14		
" " spinal cord and membranes	13		
" " heart vessels	2		
" " liver	14		
" " alimentary canal	4		
" " kidneys, bladder, and genital organs	6		
Various diseases	16		
	9		
177			

TABLE II.—*Institute of Pathological Anatomy, Vienna, 1869-1879 (Heitler).*

Number of necropsies, 16,562: obsolete tubercle in lungs, 789=4.7 per cent.

Males	509	Right lung	69
Females	280	Left lung	65
		Both lungs	655
Cause of death:			
Cancer	107		
Sarcoma	3		
Tuberculous affections, excluding pulmonary tuberculosis	106		
Empysema and bronchitis	60		
Pneumonia	46		
Pleurisy	15		
Bronchiectasis	7		
Pulmonary oedema	1		
Peritonitis	31		
Specific fevers	25		
Puerperal fevers	6		
Amyloid disease	10		
Caries and necrosis of bones	23		
Wounds, injuries, and poisons	23		
Diseases of the brain and membranes	56		
" " spinal cord and membranes	7		
" " heart and vessels	55		
" " liver	52		
" " alimentary canal	15		
" " kidney, bladder, and generative organs	93		
Various diseases (not more than 1 of any one affection)	58		
789			

On Table I, the number of necropsies of which reports were made, the number of cases in which obsolete tubercle was present in the lung, the sex of the patient and the lung affected, and the cause of death are recorded. In the *Wiener Klinik* for 1879 there will be found a paper by Heitler "On the Curability of Phthisis, and the Combination of Tuberculosis with other Disease," in which an account is given of the results obtained from the records of the Institute of Pathological Anatomy of Vienna from 1869 to 1879; these are similarly tabulated (see Table II), and the correspondence between the two is sufficiently close to afford a strong presumption of truth.

The percentage of cases of obsolete tubercle in the Middlesex Hospital table is almost twice as large as in the Vienna (9 to 4.7 per cent.), and it would have been considerably increased had the cases dying from acute tuberculous and non-pulmonary tuberculous affections been included. These I have put into a separate series, and also those in which death occurred from "phthisis" and chronic phthisis. These records were not written with a view to publication, and, as already stated, the examinations were not all made by myself. They constitute, I think, strong evidence that pulmonary tuberculosis frequently undergoes arrest, and

would, I believe, be confirmed by the records of any general hospital. The cases in Series I require a brief analysis.

Age.—The average age of the adult patients was almost exactly 50 years, but arrest may take place at any age.

Sex.—It will be seen that in both tables the number of males is largely in excess, but before concluding that arrest is more common in men than women, we require other data which are not at present to hand.

Place of Residence.—It is probable that the large majority of the individuals who appear in this series lived in London or its neighbourhood, many of them, doubtless, amid surroundings of the most unfavourable nature. That arrest may take place under such conditions is a fact which is abundantly confirmed by one's clinical experience at the Brompton Hospital. I hope, however, that it will not be said that I recommend tuberculous patients to winter in a London slum; such places are already overcrowded.

The Lung Affected.—One result, perhaps somewhat unexpected, but confirmed by Heitler's table, is that the process appeared to be more often arrested when both lungs have been affected than whilst it is limited to one. It is contrary to clinical experience to find the disease simultaneously attacking both apices and equally distributed through both upper lobes; it is therefore probable that in such cases one apex is affected after the other, and possibly in some cases after arrest has taken place there. It will be seen that there is practically no difference between the two lungs as regards liability to infiltration.

It will be noted that in some of the cases the lower lobe was already affected, showing, as I have pointed out elsewhere, that this often occurs at an early period. The fact that the lower lobe (about its posterior apex) is involved is therefore no bar to arrest of the disease.

Stage of the Morbid Process.—In the majority of cases the lesion was fibrous or caseous, and in only 43 cases out of the 177 in Series I is it stated that a cavity was present. It does not, I think, follow that the cavity found *post mortem* was a clinically recognisable cavity, or indeed that any cavity at all existed during the period of activity of the disease. I believe that in many cases it is a false appearance of excavation, produced by the growth of a fibrous capsule, which becomes smooth within, around a caseous mass. This is often described as a cavity with caseous (often blackish grey) contents. My own pathological experience gives little support to the view which is, I believe, held by some that a cavity, recognisable as such by physical signs, may become completely obliterated. If one is to believe the stories of patients (which, by the way, it is generally better not to do) some authorities appear to have arrived at such an "altitude" of diagnosis that they can trace (on paper) the gradual shrinking of a cavity up to its final obliteration. This I confess to be beyond my powers. One frequently sees a thick-walled cavity which has both contracted and altered its shape, but I cannot recall ever having observed a cicatrix which appeared to represent what was at one time a clinically recognisable cavity.

Cause of Death.—The cause of death in Heitler's cases and in those at the Middlesex Hospital is tabulated above, and the two series correspond fairly. It will be observed that in both cases the number of deaths from cancer is large. The proportional excess in the Middlesex Hospital table is due to the existence there of a special cancer foundation.

Cancer and tubercle are very often found in association, although it is rare to find both processes active in the same case, such an event being perhaps most common in cases of epithelioma of the mouth and tongue. Another possible cause for the association of cancer and tubercle is that it is more common to find in cancerous patients a family history of tuberculosis than of cancer. Both diseases are evidence of a lowered resisting power on the part of the tissues.

It is impossible to state how long in these cases the arrest of tuberculosis preceded the appearance of the cancerous growth. The association also may have been accidental or perhaps in some of the cases the arrest was an example of the antagonism of morbid processes, of which medicine affords many examples, the tuberculous infiltration ceasing on the appearance of a predominant process. The character of the pulmonary lesions in these cases negatives the view that the tuberculous process was grafted on to the cancerous, although

the possibility of such an occurrence cannot be denied. If it occurs it will probably be found where the primary growth is situated in the mouth, pharynx, or larynx.

We have now to consider what proof there is that these lesions represent the arrest of a tuberculous infiltration.

I would first direct your attention to Series II in the papers before you. This includes nineteen cases in which, lesions similar to the above being present at the apices of the lungs, death occurred from acute tuberculosis. In some of these the tuberculous infiltration was general, whilst in others it was limited to the lungs.

A case, not included in this series, which I saw in consultation with Mr. H. R. Fuller, of Curzon Street, and afterwards with Sir Andrew Clark, affords a striking proof of the tuberculous nature of the old caseous lesions.

The patient was a gentleman, aged 53, who, at the age of 13, had an illness which was said to be "consumption." He recovered and enjoyed good health, with the exception that he had an attack of pleurisy in 1885 whilst in Egypt. He was suddenly seized with hæmoptysis on February 1st, 1888, and some days later was found to have crackling *râles* at the left apex. The diagnosis then made was that an old tuberculous lesion at the left apex had broken down, and that tuberculous infiltration was occurring in the surrounding lung. The temperature continued high, and the area over which the *râles* were present gradually extended until the greater part of the left lung had become involved. The right lung was subsequently invaded, and nothing stayed the progress of the disease, the patient dying on the twenty-eighth day after the attack of hæmoptysis.

The condition present on *post-mortem* examination is represented in this diagram. (Fig. 2). At the left apex there was a

Fig. 2.

caseous mass the size of a large olive; this had been firmly encapsuled, but local changes in the neighbouring bronchus had apparently caused destruction of the capsule at one point. The caseous mass had undergone softening and about half of it had been discharged; it presented a ragged edge. The bronchus which led directly into the cavity thus formed was ulcerated at several points. The left lung was extensively infiltrated with recent miliary tubercle. The right showed similar infiltration, but over a smaller area. A point of great interest in the case was the discovery at the apex of the right lung of a firmly encapsuled caseous mass almost exactly similar to that on the opposite side. The capsule was intact at all points and no local changes were found in the neighbouring tissues. Of the presence of this lesion there were no physical signs during life, nor from its size, nature, and situation were they to be expected.

The next point in the evidence is the existence of such cases as are recorded in:

Series III. Cases presenting lesions similar to those in Series I, in which death occurred from a non-pulmonary tuberculous disease, excluding acute tuberculosis.

This series of six cases includes such diseases as laryngeal stenosis, secondary to tuberculous ulceration (caries of the lumbar spine, suppuration of caseous glands, and scrofulous nephritis.

Evidence of the tuberculous nature of these lesions is also afforded by the cases contained in:

Series IV. Cases headed phthisis or chronic phthisis in the reports showing clear evidence of a period of arrest, the lesions being in most cases similar to those in Series I.

Eighteen cases are included in this series.

It would be possible to extract from the reports of most of the cases of chronic phthisis some evidence of the disease having shown periods of quiescence or arrest; but I have been careful to select only such cases as presented these appearances in a marked degree.

The Site of the Lesions.—It will be noted that in nearly all the cases recorded the apices of the lungs are the parts affected. This corresponds with the habit of tuberculous lesions, but with none other that I am acquainted with. There is also the further point that the occurrence at the apices of lesions unlike these, and not suggesting a tuberculous origin, is extremely rare.

Absence of any other Adequate Cause.—It is out of the question to suppose that these lesions are due to syphilis, and I shall not stop to argue the point. That they are not the result of simple inflammation is proved by the presence in many of the cases of tubercles visible to the naked eye, and by the infective properties of the caseous masses.

It will doubtless occur to you to ask whether, in any case, tubercle bacilli were found in the obsolete lesions. Such a discovery would have made the evidence complete, and I regret that I cannot record it; many of the cases belong to a period prior to that of the bacillus, and none of them were recorded with a view to future use. Bacilli have, however, as you doubtless are aware been found in similar lesions and even in cretaceous masses from the apices of the lungs, and my friend and successor, as pathologist at Middlesex Hospital, Dr. Sidney Martin, will, I believe, be able to lay before you important evidence, which I have long been wanting, of the effects following on the inoculation of caseous material from such lesions.

For the foregoing reasons, then, I am of opinion that such lesions as I have described, situated at the apex of a lung, indicate the arrest of a tuberculous process.

The pathological process by which the arrest of tuberculous disease within the lung is effected is similar to that which Nature employs to separate living tissues from dead, and to isolate foreign bodies. By the formation of a barrier of inflammatory tissue the progressive spread of the disease is checked, and if the increased resisting power of the individual, which such a change implies, can be long enough maintained a firm fibrous capsule will be formed—around a caseous mass, for example—and arrest will be complete, and may possibly be permanent. But such a condition implies possibilities of danger in the future, as the disease, although encapsuled, is not eliminated.

Or, again, the reactive force of the tissues may not be able to assert itself until the disease products have softened and been eliminated; in such cases the fibrous barrier appears in the form of the thickened cavity wall.

In some rare cases a calcified mass may be discharged with its surrounding fibrous capsule and portions of lung tissue. This specimen illustrates such an occurrence; it was expectorated by a patient under my care who was undergoing treatment by the injection of tuberculin. The calcareous mass came away first, and shortly afterwards the pigmented fibrous capsule which surrounds it. I thought at the time that the specimen was unique; but I have here another of the same kind, only rather smaller, which has been kindly lent to me by Dr. Delépine, who has met with two other cases of a like nature.

I fear, however, that it is not to remedies which effect the elimination of tuberculous deposits that we must look forward with hope in our battle with pulmonary tuberculosis, but rather to such as increase the resisting power of the individual and enable his tissues to stop the progress of the disease. This they are ever striving to do, even when the destructive process is most acute, and it is only necessary that the local or constitutional conditions should turn in their favour for them to commence the construction of the fibrous wall which will repel the invader.

What it was in the cases which I have related that turned the scale in favour of the patient I cannot say. The reports

are pathological and not clinical, but it is improbable that the medical histories would have repaid an examination. In the great majority there would certainly have been nothing to suggest the presence of a tuberculous lesion of the lungs. The patients belonged to a class which does not, as a rule, regard slight ailments, and is obliged to work as long as strength holds out.

The affection, whilst in an early stage, may have been arrested by some intercurrent disease, for I have a firm belief in the antagonism of certain morbid processes. It may be that in some cases the resisting power of the individual was lowered by unfavourable surroundings, and that a condition of the tissues was established which allowed the bacillus to obtain a lodgment, and that this was followed by an increase of vitality, which prevented further extension of the morbid process, and shut off the disease products from the organism. In some cases the increase in vitality appears to come too late to arrest the local lesion, but early enough to prevent the generalisation of the disease. In such cases a whole lung may very slowly undergo destruction, whilst compensatory changes in its fellow provide for the respiratory needs of the body, death occurring only after many years, and then possibly from some other affection.

On reference to Series IV, it will be noted that a cavity was present in nearly all the cases. But in estimating the probability of arrest occurring in any given case, the extent of lung affected is of greater importance than the presence of a cavity, although widespread infiltration is naturally often associated with excavation about the site of the primary lesion. Arrest may be hoped for in cases of fibroid tuberculosis with a larger area of infiltration than in any other form of the disease. It may, however be stated that if the cavity can be recognised clinically the probabilities are that at some later period there will be an extension of the tuberculous process, and that death will occur from a tuberculous lesion.

But to this rule many cases are exceptions; many recognised and probably many which escape observation. I saw lately a lady, aged over 60, who was complaining of slight bronchial catarrh, and on examination of the chest I discovered a cavity at the left apex, which was proved with almost absolute certainty from the patient's history to have existed for forty-seven years.

The relation of the presence of the bacilli to the question of arrest is of great interest. So long as they continue in the sputa, the lesion cannot be encapsuled; and it is only in the fibrous barrier that safety lies. But their presence does not, in my opinion, necessarily imply that the area of infiltration is extending, and it is certainly compatible with the complete absence of symptoms and signs, and the enjoyment of what is often described as perfect health. I am acquainted with cases in which they have been present in the sputa whenever looked for since the discovery of the micro-organism, notwithstanding that the patient has generally enjoyed good health, and they may be the only thing of which he complains.

It is very unwise to allow the patient to concentrate his attention on the bacillus, as such a condition of mind may mean the destruction of hope—a factor of the utmost importance in the treatment of the disease.

It has possibly appeared to some of my audience that the pathological experience which I have related represents the arrest of pulmonary tuberculosis as of more frequent occurrence than their own clinical observations would support.

Many writers on the subject of "phthisis" refer to the few cases of complete cure which they have observed, but it must be remembered that their conception of the disease is clinical rather than pathological, that many of the cases when first recognised are fairly advanced, and that they leave out of reckoning altogether the cases which have been diagnosed as "catarrh of the apex"—"congestion of the apex," "pneumonia of the apex," a diagnosis which is often held to have been correct from the subsequent complete disappearance of the physical signs, etc., and also the cases of hæmoptysis, in which the blood was said to have come from the throat or elsewhere merely because there were no physical signs of disease discoverable in the lungs.

The well-known case of Sir Astley Cooper may be quoted

as well illustrating this point. He left directions that his body was to be examined after death with particular regard to certain points, one of which was "some suspected indications of phthisis in his youth." He is believed to have had an attack of hæmoptysis for which he bled himself. The result was that "at the superior and posterior part of the right lung was a small depressed and somewhat contracted surface about the size of a sixpence, a section of which exposed a calcareous mass very uneven upon its surface, and about equal to the size of a small pea. It was placed about three-lines distant from the pleura."

There can be no doubt that such lesions as I have referred to represent a period of ill-health, and it is highly probable that they might during their active period have been recognised by physical signs. Pulmonary tuberculosis in this early stage has been frequently overlooked in the past, but now that in the examination of the sputum we possess a test for the presence of the disease, it is possible to make a certain diagnosis at a much earlier period, and for this reason it becomes of great importance that we should be aware how frequently the process is arrested.

NOTES ON

"HEALED" OR RETROGRADE TUBERCLE.

Read in the Section of Pathology at the Annual Meeting of the British Medical Association, held in Bournemouth, July, 1891.

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In patients dying from diseases other than tubercle, tuberculous lesions, old or recent, are not infrequently found. The old tuberculous lesions are usually in the form of what is called retrograde tubercle, that is, tubercle which is no longer active, which no longer tends to generalise itself over the body. As a rule such old lesions are found at or near the apices of the lungs, in rarer cases in the mesenteric glands with or without healed tuberculous ulcers of the intestine. Retrograde tubercle in the lungs occurs in two forms, one of which may be called "calcareo-caseous tubercle," and the other "fibroid and pigmented tubercle."

In the first case, the healed tuberculous lesion is in the form of calcareo-caseous nodules, from $\frac{1}{8}$ to $\frac{1}{2}$ inch in diameter, and surrounded by dense and pigmented fibrous tissue; there are not infrequently nodules of fibroid and pigmented miliary tubercle near these calcareo-caseous masses. The area of lung affected is, as a rule, small, not more than 2 inches in diameter; not infrequently, however, there are only one or two small nodules present. In the majority of cases no other tuberculous lesion is found in the body.

In the second case, there are no calcareo-caseous nodules, but the miliary granulations are completely transformed into dense and pigmented fibrous tissue. In some cases only a small area of the apex of the lung is affected; in other cases the area is much larger, and extends over the posterior half of the upper lobes.

The great difference between these two varieties of healed tubercle is this: whereas the fibroid and pigmented miliary tubercle never contains any tubercle bacilli, the calcareo-caseous tubercle almost constantly does. The bacilli are not commonly very numerous. They are of normal shape, and occur singly or in groups of five to twelve.

For some time past I have noted all the cases of retrograde tubercle which have been observed in the *post-mortem* room at the Middlesex Hospital, and will shortly state the results of these observations. In 445 consecutive *post-mortem* examinations, 31 cases of calcareo-caseous tubercle were observed,