Thesis on
"The Etiology of Phthisis Pulmonalis"
composed
by
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Etiology of Phthisis Pulmonalis.

The reviewer of a book on the Pathology of Phthisis Pulmonalis a few years ago wrote thus:*
"Pulmonary Consumption & its relation to Tuberculosis has been for years past one of the chief battlegrounds of pathologists, and those who have vainly looked for some tangible result from so much discussion have had to content themselves with noise and smoke, & the often doubtful advantage now of this side & now of that. Many pathologists to whom we habitually look for instruction have on this question behaved like veritable weather-cocks, & their opinions have shown a most ominous want of stability, being blown hither & thither by every blast of doctrine, until the mere outsider & looker-on came reluctantly to the conclusion that ex cathedrâ utterances were, after all, not unfrequently the offspring of muddled brains." The discoveries of the last year, have advanced our knowledge considerably, but still there are difficulties in grasping definitely this most important subject, & meeting constantly — as one must do in this country — cases requiring immediate treatment.

* Review of 'The Pathology of Pulmonary Consumption by T. H. Green M.D. Cantab, 31 May 1879. p 775.

treatment I was forced to formulate for my own use, those ideas in which most authorities seemed to agree, so that I might have some basis upon which to construct a treatment, of which I might understand the rationale; for it appears that the various modes of treatment have been as varied as the pathological theories. In Phthisis, as much as to any other disease, the words of Sir James Paget will hold good; he says "We need not only the diagnosis between diseases essentially different, but that between the different & varying forms of each of those which we call by a generic name; & beyond this, we need a more exact power of what may be called analytic diagnosis; for there are few simple cases, & in those which are not simple we need to be able to discern all the components, & the proportions in which they are mingled or combined. Better treatment will follow better diagnosis, & better diagnosis will certainly follow a more exact pathology".

The contagiousness of Phthisis is no new theory, but as no definite proof could be given, it has hitherto remained a disputed point; now, that is all changed & though there are still some who deny that tubercle is in any way caused by a germ, I think we are must be compelled to remodel our

* "The Bradshawe Lecture" by Sir James Paget: vide Brit: Med: Journal 1882 Vol II p 1191

our notions on the subject. The terms Phthisis & Consumption have been used to include a number of lung-diseases which further inquiry may show to have no connection with germs, such as that condition which occurs in Diabetes, & which is really according to Addison & Wilks a "lung albuminisation" But it appears to me that there is no doubt that Koch's germ will produce Phthisis. †Koch has given indisputable proof:- he has found the germ in all the cases he has examin-ed; he has cultivated it apart from its ordinary nidus; & has demonstrated that an organism so cultivated is capable of generating the disease, when introduced into the system. Now, as the germs must be in great abundance, in various degrees of development, even under the unfavorable conditions of our climate, & especially in Hospitals devoted to ‡consumptives, it follows that we would all be suffering from phthisis, unless there existed other factors in the production of the disease. We are thus brought to the question Do there any conditions of the body, or of parts of the body, conducive to the growth of germs?

‡Debate on Diabetes at Pathological Society of London April 3. 1883. Reported Brit: Med: Journ: 1883 Vol I pp 664

†Summary in Articles in Brit: Med: Journ: 1882 Vol I pp 624-706

‡Speech by C. J. Williams at meeting of Medical Society of London Feb 12. 1883. Reported in Lancet.

From analogy, we say, yes, for we find that other forms of vegetable life require special soil in which alone they will thrive (of course with proper conditions of temperature etc) and it has also been shewn that all germs cannot be cultivated in the same medium. It seems to me that there are several conditions which allow the germ to become established in the system or in an organ & which may be regarded as General & Local.

Of these conditions, the chief appears to be a depressed state of the health in which any troubles, whether affecting the mind or the body, are not repelled or thrown off as in health. It is not perhaps a very well defined state, yet one which is common enough to be familiar to most us. It is a condition in which we are easily "knocked over"; & the numerous germ-troops can without much opposition, storm & carry the citadel of the body. It is a condition which might be compared to the sleep of Gulliver, when he was overcome by the Liliputians, who could easily be resisted & thrown off while he was awake. The germs will soon find out the weak points, & many settle down in a lung, in lymphatic glands or other parts, or they may be disseminated all over the body with so much

much constitutional disturbance as to early produce a fatal result, while the local action may be very limited. This would be a parallel instance, to what sometimes occurs in other diseases as Scarlet Fever, in which a person may die rapidly from the severity of the poison before many of the pathognomonic symptoms had time to develope

For the production of this general condition of "vulnerability", it is possible to make out several causes, but I would first refer to statements made in a paper by Formad, of * Philadelphia U.S.A. He asserts that "from an examination of many hundred bodies of man & other animals he is certain that there is a special anatomical peculiarity in every one exhibiting phthisis or scrofula whether the diathesis be inherited or acquired. The organs supposed to be concerned in the production of white blood corpuscles are disproportionately large to the size of the animals, & have narrowness of the lymph spaces which are partially obliterated by cellular elements. If any inflammation (damage) occurs the germs at once obtain a hold. This condition may be acquired by mal-nutrition and confinement

* Philadelphia Med: Times Nov: 18. 1882

confinement. Inflammation (damage) is a necessary starting point even in those predisposed. No inflammation" says Formad "no tuberculosis". He also holds that the presence of the bacilli is secondary to the inflammation. With this sweeping statement I cannot agree. In many cases of phthisis what he describes will be found, but the changes are very probably what is usually known as struma or scrofula in which any irritation will set up inflammation of a low sort. But the cases Formad examined were all suffering from phthisis or scrofula & as we hold that the diseased action in each is most commonly the manifestation of the Bacillus tuberculosis, his work goes but to show the changes, produced in the lymphatic system by the bacilli. That there is a condition of constitution which readily responds to slight stimuli, & in which glands will inflame & take on a chronic action has long been recognised; & to that condition only should, I think the term of <u>scrofula</u>, be used. & If such marked changes as Formad describes, were present throughout the body in all persons suffering from phthisis, then there would be some external signs by which this wide-spread internal change could be recognised

recognised. This point Dr Mahomed & Francis Galton F.R.S. have settled by their method of Composite Portraits. On examining patients "with strong hereditary taint of phthisis" they found that the average face gives more delicate features, an apparently lighter lower jaw & an altogether narrower face than the average in other diseases; while the average face was so, the narrow ovoid was present in only 14·3 per cent of the phthisical cases, it was present in almost as many cases other than phthisis, (the actual percentage was 14%) so that it is evident that the delicate narrow ovoid face may mean liability to disease of any sort. But when phthisis has become well developed they were able to get a typical face viz:- Large projecting ears, narrow mouth & a short small chin, a small narrow lower jaw with perhaps prominent upper teeth. The faces might be divided into two groups in one of which the narrow ovoid predominated, & in the other, blunt thick features. Thus while not finding any special characteristic as representing a constitution certain to develope phthisis, they recognised the external signs of scrofula, of which formado has now described some of the internal

* Composite Portraits of the Physiognomy of Phthisis, Guy's Hosp: Reports 1887. (Vol 2 5th Series)

internal changes. At present, I have nothing to do with the other organs of the body, but may merely say that the presence in them of the Bacillus tuberculosis may or does lead to different changes, to what occur in the lungs, on account no doubt of the different construction of the various parts. Although without confirmatory evidence from further investigation, I cannot believe that Formad is correct in saying that this condition is absolutely necessary for the growth of the germ; yet I admit that Scrofula is one of the most suitable conditions of the body for the bacillus, & this is shewn by the frequency with which persons having this 'temperament', become affected with the Bacillus tuberculosis in one part or another. Let such a person have some slight irritation other than of a germ, to a mucous ~~mem~~ surface, immediately the lymphatics connected therewith take on a low sort of inflammation of which the products are not readily absorbed & tend to linger in the tissues[*]: yet the irritation being withdrawn the parts may recover: but let the exciting cause be the tubercle bacillus or let it be superadded to the already existing inflammation, then the disease becomes more chronic and tends to spread to other parts.

[*] Vide Lectures on Pathology of Scrofulous Lymphatic (with) Glands by Mr Treves: Brit: Med: Jour: 1877 Vol I p 678.

with more or less rapidity. If we consider that probably one of the causes of this strumous condition is Syphilis in a diluted form then we can understand that the inflammatory changes would be of a low type: & it is of interest to note that in Syphilis there is a phthisis-like affection of the lung without the presence of Koch's germ. But Scrofula may be brought about by causes other than Syphilis, (which is a probable cause). And here I might draw attention to the confusion in the use of terms which is likely to arise unless, when we know more fully of the relations of the tubercle bacillus with disease, the meanings of such words as scrofula & phthisis are more definitely stated: thus at present 'scrofula' is often used for the manifestation of the bacillus in the lymphatic system, as for the condition of the part to suitable for germ growth: so also as regards the term "tuberculosis", that is perhaps a name as suitable as any, although there are cases in which no tubercles are ever formed: & there are other cases having small nodes in parts of various organs without there being any connection with the tubercle bacillus). To return, however; that favorable condition for germ-growth which has

has been called Scrofula, may result from it" (1) Deficient supply of food as to quantity & quality. – unsuited to age or defective in certain elements.

(2) Bad air; consuming of breathed air.

(3) Want of exercise. Persons more or less confined for long periods in close ill-ventilated rooms taking little exercise & having deficient food are very apt to develope this temperament."

But although it may thus be acquired, it may not be transmitted to offspring. The condition is often present at birth, but it is not necessarily ~~the result~~ derived from scrofulous parents. These points seem to have been in great measure understood by Niemeyer so far as the knowledge of his time allowed.

Now though the "scrofulous condition" is a frequent fore-runner of pulmonary tuberculosis there are other conditions which will afford a congenial home for the germs & in this wise, I think I am supported by Prof: Ziegler & Dr. Macalister.

* From notes of Lectures by Prof: Errainger Stewart during sessions 1878-79-80. Ed: Univ:

† Sydenham Society's Translation of Niemeyer's Lectures on Pulmonary Consumption: Section on Aetiology.

‡ A Text book of Pathological Anatomy & Pathogenesis 1883 Pt I Chap: on 'Granulomata'.

Mac Alister: & these favorable conditions for the growth of the bacillus, are brought about by inhalation of dust in various forms, or result from other diseases as measles, whooping-cough, croupous pneumonia & bronchitis, or may be induced by influences which weaken the body (without the production of the strumous condition), such as defective food, ventilation & exercise, debauchery etc: Why should want of exercise, bad food or debilitating diseases & make a person subject to tubercle of the lungs? Refer first: the tissues of the body generally are incapable to resist disease, & 2nd: a favorable local condition is produced in the lung. Referring again to Ziegler, we find that "The germs are difficult to cultivate & in the body only grow when they reach a spot not subject to much mechanical disturbance or displacement." If that is so, then one part of the lung is more at rest than another, there will be the usual starting place of this disease: and so we find it, the apex is the most immobile part of the lung, & is "almost invariably the seat of early phthisis." †

"The apex movements are limited by their position & by the frequent cohesion of the

‡ Op: cit: p 10

† Jas. E. Pollock M.D. Harveian Lectures vide Brit: Med: Journ: 1887 Vol I p 39

the pleura at that part, while mechanically it is somewhat difficult to empty the upper portion of the lung". Again less blood circulates in the apices, & as the lung is fixed at its root, the apices being at a higher level, the expanding influence of the diaphragm is less felt there. Thus any additional agencies acting with the natural ones will produce a loss of tone in the lung apex, & thus* "a state of things is induced which although not inflammation, is so immediately related to it, that out of it without any intervening process of development, inflammation at once declares itself". The circulation is retarded in the apex, & to quote Prof: Burdon Sanderson further, † "retardation of the stream particularly in the veins is a condition sine quâ non of emigration". "The process of inflammation is essentially a terminable process - i.e. one which has no tendency to spread or last beyond the limits of the proximate cause". Now applying

* For discussion of other causes supposed to conduce to apex disease Gulstonian Lectures on Pulmonary Cavities by Wm Ewart M.D. reported in Brit: Med: Journal 1882 Vol I p 369

† Lumleian Lectures on Inflammation by Burdon Sanderson M.D. Lect II: Brit: Med Jour: 1882 Vol I p 492

‡ Ibid p 450.

applying this to the lung, there are cases* in which there appears to be some slight change at the apex, but which rally speedily as after an ordinary localised inflammation: but there are many others which go on when the cause of the inflammation has subsided, & there must consequently be some other source of irritation: given the presence of a germ & we see why the disease is kept up. But

*The following seems to be an example of such cases:—
I.V. female aged 20 years, healthy till now, with no family-history of phthisis, had been studying hard for an examination for nearly a year, during which she lost flesh & suffered much from dyspepsia with its usual accompaniments. At the beginning of Dec: 1882 she began to have a dry cough with pains at the upper part of the chest on the left side. On examination:- At the left apex there was slight dulness in the sub-clavicular region: no flattening: Auscultation, above & below clavicle showed inspiratory sounds to be prolonged: the expiratory short & occasionally interrupted, with fine crepitations. Temperature varied from normal to 101°.5 F. but there was no delirium. The treatment consisted in regulating stomach & bowels; giving a saline cough mixture; & applying mustard over apex, followed by linseed poultices. The fever diminished, & the dulness slowly disappeared with improvement in the breathing. There was little expectoration; & in the only examination I was able to make of it no Bacilli were detected. By the end of January 1883 Miss I.V. was able to go about, was eating & digesting well & gaining weight. She was then taking Hy pophosphites. (She is now well Ap. 20. 83)

But there is a further stage of acute inflammation of the lung in which the exudation breaks down; that condition is favorable for the growth of the tubercle-bacillus, & especially so when the pneumonia involves the apex either primarily or by extension. That this does happen, is shewn by the number of apex-pneumonia cases ending in Phthisis.

Another source from which an infective tuberculosis may spring is to be found in connection with Haemorrhage into pulmonary tissue. While some have held that haemorrhage into the lungs is a certain fore-runner of phthisis, others are - or were, of opinion that tubercle was the cause of the haemorrhage‡. Now however we recognise, that there are cases free from tubercle in which blood is effused into the lungs without any phthisis resulting, and that there are cases of the disease in which haemorrhage never occurs. The best authority on this subject, so far as I am aware, is Dr. Reginald E. Thompson who has specially studied† "The causes & results of Pulmonary Haemorrhage"

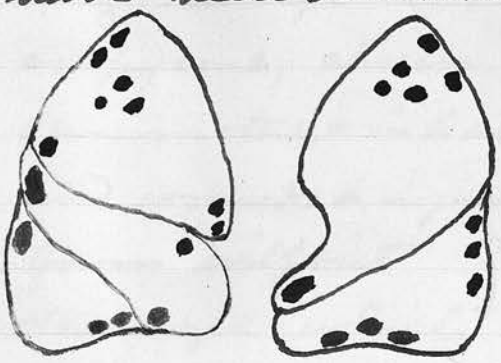
‡ Laënnec, "Traité de l'Auscultation mediate".

† London 1879: see also in Transactions of the Roy: Med: & Chir: Soc. London. Vol. 61 "On Pathological Traces of Pulmonary Haemorrhage". & J. E. Pollock M.D. on Haemoptysis &c Brit: Med: Jour: 1881 Vol I p 109

Haemorrhage". He points out that there are three sources of bleeding connected with the disease: (1) First as a result of confirmed Phthisis: Second(2) As an early complication of the congestive forms of the disease: Third (3) As an event not dependent upon pre-existing phthisis, but capable of producing the latter. Of the first form it is unnecessary to speak here while the second comes under the head of congestion & inflammation which we have already recognised as a preliminary cause of phthisis. In the third form phthisis is an event subsequent to the local disablement of the lung, but as we have said there are cases in which phthisis does not follow. The fibrinous clots of blood may become hard & gradually shrink & contract, giving no further trouble; or they may break down & be expectorated leaving a cavity which may contract under favorable circumstances; but if the bacillus effects an entrance into the damaged tissue tuberculosis will soon follow. The germ was the link in the chain, which would have shown Laënnec that it was possible "to understand how haem- optysis could lead to tuberculosis" & that in supposing "that phthisis was always caused by a new growth" he was nearer the truth than he himself knew.

vide Meinert p 20-21 (Sydenham translation) & Laënnec Op: cit: Tom. II p 118 etc.

The positions into which the blood gets have been described by Dr R. J. Thompson



* These are:- the summit & middle part of upper lobe, the middle axillary region close to the pleura, the anterior inferior border and the middle part of the base corresponding to the summit of the arch of the diaphragm. A glance at the above diagram at once shows that other parts of the lung suffer at first quite as much as the apex does: but although excavations are caused in the base of the lung by the softening of the haemorrhagic nodules yet it is extremely rare that tubercular disease attacks that part - except by extension when the upper portion of lung is seriously damaged by the disease. So that while the upper lobe of the lung afforded facilities for the production of phthisis, which the other parts do not possess, the ~~excavations~~ cavities left by the softened nodules (absorbed or expectorated) would heal as readily there, as in the other more mobile portions of the lung. These facilities I have already noticed (page 11). The

* From diagram in Brit: M. Jour: 1882 Vol I p 416. Dr Ewart there discusses Haemorrhage in regard to the Causation of Pulmonary Cavities: & I have followed in this part of the subject in somewhat similar lines.

The result is very similar in regard to lung-damage by other means such as, empyema, abscess of the lung, hydatids, gangrene, gummata, & inhalation of foreign bodies, and wounds & injuries of the lung inflicted from without. The base recovers from damage which in the apex would surely be followed by tubercle.

The subject of extension of the tubercular disease from other parts of the body is discussed below under the question, "How the germ reaches the Lungs."

From what has now been said I think we may conclude that however the germ reaches the lung, in order that tuberculosis result there must be some damage or weakness in the part, probably one or more of the factors already described. The division of Phthisis into Inflammatory, Tubercular & mixed forms does not seem to be so very far wrong; including in the term "Phthisis" or 'Consumption' of the Lungs the various destructive diseases which are now getting differentiated, specific tuberculosis stands out clearly as either a primary disease, or one superadded to another already existing; just as whooping-cough may attack a child de novo, or may follow in the wake of some other disease such as measles. The number of disorders upon which the (bacillus)

bacillus of tubercle may success fully develope, shows what diversity must exist in the modes of treatment after the germ has been got rid of: still once the lungs are clear of the germs, they are placed in a position to recover, if the general & "local" "tone" can be improved, (this, of course is generally being attempted while the extermination of the germs is going on): if the original conditions are left pretty much in the same as when the bacilli first found them germs will very soon find their way back again. As examples of this might be taken those cases in which the disease becomes quiescent after residence at High Altitudes or under anti-septic treatment, but returns or breaks out again on the withdrawal of an a-septic or anti-septic atmosphere.

Diagnosis is becoming more & more scientific & exact, & it is well that it is so, but it should always be kept in mind that the aim & object of being correct in diagnosis is not to obtain the satisfaction of a "post-mortem" confirmation of it, but is to lead to a proper treatment for the possible relief & cure of the sufferer, consequently in a disease such as we are now considering to be able early to recognise those conditions likely to result in tuberculosis with a view to rectification thus

thus to prevention of this cruel disease, is of decided importance.

We now come to the question:- "How does the tubercle Bacillus enter the system?" Koch has shewn in his experiments that results occur with the greatest rapidity when the inoculation or injection of the germ was directly into the veins or into the abdominal cavity. In such cases then of general tuberculosis we would expect to find the germ in the blood but D^r C. Theodore Williams informs me that D^r Percy Kidd, the Pathologist at Brompton Hospital, or he himself, have examined the blood in a number of such cases without finding the bacillus; but Prof: Weigert of Leipzig has described how the bacilli may pass directly out of the chyle, in absence of intestinal ulceration, into the thoracic duct (according to Prof: Ponfick) & so into the veins, with the production of tubercles in the walls; therefore when the inoculation is in that manner germs must be found at one time or another in the blood but there

* vide summary of Koch's paper in Brit: Med. Journ. 1882 Vol 2 p 707.

† Prof: Weigert: On Tubercles of Veins. Virchow's Archiv: May 1882: vide Med. Times & Gazette 7 Oct 1882 & American Journ: of the Med: Sciences Jan: 1883 p 264.

there are other means by which the bacilli may reach the lung: & these have lately been specially noticed by Mr Watson Cheyne, of whose rising reputation Edinburgh graduates may well be proud ‡. He comes to the conclusion that the bacilli may enter the lungs directly by the bronchi & may at once attack the alveolar epi- -thelium, the disease spreading by continu- -ity to other alveoli: this, of course, occurs when the local conditions are favorable, but † Dr Theodore Williams thinks that "it is possible, if "(the germs be)" in great numbers & under specially favorable opportunities for multiplication and development such as are to be found in the hot climates of the South Pacific Islands that even individuals not predisposed may be attacked". More frequently however the entry of the germ is more insidious. Mr Watson Cheyne shows that "the bacilli" (having obtained admission to the veins & lymphatics) "escape from them & pass into the alveolar epithelium where they grow, causing multiplication of the epithelial cells". The

‡ Report on the Relation of Micro-organisms to Tuber- -culosis. Practitioner April 1883: & B.M.J. 1883 Vol I p 507

† Speech made at meeting of the med: Soc: of London Feb: 12. 1883, & reported in Lancet.

The mode of entry into the lung evidently is another factor in determining the form which the disease may take. Thus through the blood-vessels the lungs may be affected either by a general tuberculosis, or by the submucous alveolar tissue being attacked; through the lymphatics, this latter form may also be produced: while by the bronchi either directly or by extension from the bronchial epithelial cells an inter-alveolar form may occur. Probably the various forms exist more or less in all but the acute general tuberculosis form; & when the cases become chronic the fibrous tissue of the part gets increased in quantity.

Besides the manner of getting into the abdominal lymphatics & so to the thoracic duct, there are other ways in which the bacillus may reach the lymphatics ~~in other parts of the body~~ of the lung. These ways are (1) by extension from disease existing in some other part of the body, when it is easily understood that the germs may be conveyed to the lung, examples of this are quite common. and

(2) entrance to the pulmonary lymphatics may, I think, be given by those small openings the pseudo-stomata of the bronchi. I have no where seen this mode

mode described, but on considering the matter I was led to think of cases of Anthracosis & the manner in which the particles of coal dust enter the lung tissue. If coal in pieces often large enough to show distinctive microscopic characters can enter by these pseudo stomata, what is there to hinder the entrance of a germ, only one third of the diameter of a red blood corpuscle in length? Entrance through these openings would then bring the bacilli into the peri-bronchial lymphatics & thence into the peri-vascular set: (from description given by Prof: D.J. Hamilton in Practical Pathology class) These vessels run outwards & form a dense plexus in the inner layer of the pleura, on which is so often affected when phthisis is present. The cell elements in the peri vascular sheathes proliferate forming little round swellings which press into the lumen of the vessel which they either obliterate, or burst into: so from the other set - as well as from the peri-vascular - the germs may escape into the alveolar epithelium. Watson Cheyne says that "probably the cells filling the alveolus are derived from the epithelioid lining of blood & lymphatic vessels": & that would certainly be possible if the mode of entrance is as I have stated. If the tubercles in the lymph spaces surrounding bronchi & do not (break)

break through into the alveoli then the sub-mucous form of the disease will pre-dominate; but in actual practice that & the inter-alveolar affection are found to be closely united. Recently* a paper has been published by Spina of Vienna, in which he states that in his opinion the mode of entry of the germs through the lymphatic or blood vessels," is a purely gratuitous assumption as the bacilli easily find their way into the bronchi with the atmospheric air". So they might! much more easily than through the tissues, but if the entrance of dust in Anthracosis is at all analogous, then the micro-organism will enter either way, probably often in both ways. But Dr Spina goes much further & denies even the existence of the tubercle bacillus: ~~Dr~~ Prof: Toussaint of Toulouse has been shown by Watson Cheyne to have erred on this point as Spina although an assistant in Prof: Stricker's Institute may also have fallen into mistakes. If the germ be not a cause of tubercle then we should expect to find absent sometimes from cases of tuberculosis; and sometimes at least, present in cases of other diseases (but)

* Brit: Med: Journ: 1883 Vol I p. 473. Sketch of paper in Wien: Allgem: Med: Zeitung. by Spina 1883. Feb 13.

but numerous observers *† have come to the conclusion that this germ does not occur in diseases other than tubercular; and that in tubercular disease of the lung is, with few exceptions (which can be explained) always to be found. The general appearance of the Bacillus † & the various methods of staining & demonstrating it are now well known; but some of the failures in

*† Heron: Lancet 1883 Vol I Feb 3: p 188: B.M.J: 1883 Vol I p 186
Pfeiffer: Berliner Klinische Wochenschrift Jan: 15: 1883
Balmer & Fränkel. Ibid: No. 45. 1882.
Guttmann. Ibid: No. 52 1882.
Dettweitzen & Meissen. Ibid No 7 1883.
D'Espine. Revue Medicale de la Suisse Romande: Dec: 1882.
Lichtheim. Fortschritte der Medicin No. I 1883
Licht. Deutsche Med: Wochen: No. 5. 1883
Hillis Ibid No 47 1882
Broadfield. Brit: Med: Journal 1883 Vol I p 205
Marchiafava & Celli. Gazz: degli Osp: 1883 Oct 7.
Williams: speech quoted (Feb 1883): & many others than mentioned
† Ziegler & Macalister. Op cit. Art 127.
Ehrlich. B.M.J. 82. Vol I p 916 Vol II p 735.
Reid Fleisch B.M.J. 1882 Vol II p 142.
Hanaye Gibbs B.M.J. 1882 Vol II pp 735 & 788.
Vignal B.M.J. 1882 Vol II p 826.
Baumgarten B.M.J 1882 Vol II p 1096.
Negri & Pinolini: Lond: Med: Record Jan 15. 1883 p. 25. describe five forms which are probably the Bacillus in different stages of development.

in well marked cases of the disease, to demonstrate the Bacillus are due to the methods used for collecting the sputa for examination. The best time for obtaining the sputa is the morning after the patient has fasted for some hours; the mouth should be washed out before spitting, to remove the Leptothrix buccalis: the sputa, received in a bottle previously washed in carbolic acid, should be covered with alcohol as soon as possible; the preparation is then ready for staining & examination whenever it may be required. It is best to examine the sputa several days in succession.

A simple method of examining the breath for germs has been described by R. C. Smith M.D. D.Sc. U. of Manchester: but if there are many germs in the surrounding air at the time, germs will enter the bronchi by the nose unless it is also included in the respirator. Dr. W. Roberts of Manchester † has shewn to the Royal Society a method of examining the breath: & Drs Ransom & Theodore Williams have also devised plans for the examination of the air in rooms, hospital wards, ventilating shafts etc. But it would not be necessarily Phthisis pulmonalis from which a person would

‡ Brit: Med: Journal 1883 Vol I p.105-

† Midland Med: Miscellany 1883 p.27

would be suffering, were tubercle germs found in his breath; as there might be present a tubercular condition of the larynx or bronchi. The relation between the disease in those parts & the lung is interesting. Already we have described one way in which the germ may spread from the bronchi to the pulmonary lymphatics, but we must not forget that the germ may pass from larynx or bronchi directly to the alveoli, or the disease of those parts may extend into the lung cells. Dr. Hunter MacKenzie *) states that as a rule the larynx more often infects the lung than is ~~it~~ infected from it: & he points out that a simple chronic laryngitis may develope into tubercular. So, a tubercular, or "consumptive" bronchitis may also lead on to the similar disease of the lungs. That so called "neglected colds" may lead to tubercle-germ growth is important, as they are often treated as if they were of little consequence.

In the preceding pages I have stated what seem to be usual forerunners of Phthisis Pulmonalis but the subject is a wide one & I feel that there is a great deal of information wanted before we can fully understand all the

*) "Phthisis of the larynx" Edin: Med: Journal No. 331 Jan. 1883 p. 594

the relations existing between the germ & the body. But I think we may draw some general conclusions as to treatment from what has been said without going into the question of diagnosis which would require a Thesis itself and I cannot do better than quote the words of Dr. Williams at the end of the speech already referred to, as they embody what I wish to say. "Measures directed to the fortifying & strengthening of the constitutions & thus enabling it to withstand the attacks of the bacillus, will be found most effective in the long run, though I would not exclude antiseptic treatment especially in the form of pure air, pure food & abundant exercise in mountain climates, which induce more complete development of the organs of respiration".

Francis J. Allan.

to the eye, because her
music is shut out from
the ear; so ——

"Educate your eye
to distinguish what
is said, and thus
render yourself,
to some extent independent
of the failing organ
And thus it may be
you will make a
valuable provision
for the future".