Thesis on
"The Etiology of Ophthalmia 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, o the often doubtful advantage now of this side a word or that. Many pathologists to whom we habitually look for instruction have on this question behaved like veritable weather-cocks, so their opinions have shown a most ominous want of stability, being blown hither & thither by every blast of doctrine, until the mere outsider a looker-on came reluctantly to the conclusion that as cathedral 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, a meeting constantly — as one must do in this country — cases requiring immediate treatment.

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 and varying forms of each of those which we call by a generic name; beyond this, we need a more exact notion of what may be called analytic diagnosis: for there are few simple cases, it is those which are not simple we need to be able to discern all the components, and 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 must be compelled to remodel our thoughts."

our notions on the subject. The terms Pulmonary & Consumption have been used to include a number of lung diseases which, further inquiring may show to have connection with forms, such as that conditions 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 Pulmonic. Koch has given indisputable proof: he has found the germ in all the cases he has examined; he has cultivated it apart from its ordinary milieu; & 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 your climate, especially in Hospitals devoted to consumption, it follows that we would all be suffering from Pulmonic, unless there existed other factors in the production of the disease. We are thus brought to the question, are there any conditions of the body, or of parts of the body, conducive to the growth of germs? (Debat on Diabetes at Pathological Society of London.)


Speech by E. T. Williams at meeting of Medical Society of London Feb. 18, 1893. 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 shown 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 to which may be regarded as General or Local.

Of these conditions, the chief appears to be a deeneroused 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 of us. It is a condition in which we are easily "knocked over" or the numerous germs attack us without much opposition, storm a every citadel of the body. It is a condition which might be compared to the sleep of Sullivan, when he was overcome by the Lilliputians, who could easily be resisted or thrown off while he was awake. The germs will soon find out the weak points, or many cattle down in a line, 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 some toxicosis 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 develop.

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 and other animals he is certain that there is a special anatomical peculiarity in every one exhibiting phthisio 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, and 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, 1852
confinement. Inflammation (damage) is a necessary starting point, even in those predisposed. No inflammation, no phthisis. He also holds that the presence of the bacilli is secondary to the inflammation. With this amazing 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 atrophy or cachexia in which any imitation will set up inflammation of a lung sort. But the cases formed examined were all suffering from phthisis or carphula, so 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 mainly responds to slight stimuli, in which glands will in nature take on a chronic action has long been recognized; to that condition only should I think the term of squama, be used. If such marked changes as formed 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 recognized.
recognised. This point, Dr. Mohamed &
Francis Galton F.R.S. have settled by their method
of Composite Portraits. On examining patients
"with strong hereditary taint of Phtthisis" 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 phthisical (the
actual percentage was 14%) so that it is evident
that the delicate narrow ovoid face may
tend 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 character-
istic as representing a constitution certain
to develop phthisis, they recognised the
external signs of cedema, of which
formade has now described some of the
intimal

Composite Portraits of the Physiognomy of
Phtthisis, Guy's Hospital Reports 1907 (Vol 26: Series 3)
internal changes. At present, I have nothing to do with the other organs of the body, but may merely say that the presence in some of the bacillus tuberculosis may or may lead to different changes, to what occurs in the lung, on account no doubt of the different construction of the various parts. Although without confirmatory evidence from further investigation, I cannot believe that Forman is correct in saying that this condition is absolutely necessary for the growth of the germ; yet I admit that Serejala is one of the most suitable conditions of the body for the bacillus, so this is shown 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 to a germ, to a mucous membrane, the lymphatics connected therewith take on a local sort of inflammation of which the products are not readily absorbed and tend to linger in the tissues; yet the irritant being withdrawn the parts may recover. But let the exciting cause be the tubercle bacillus or let it be super-added to the already existing inflammation, then the disease becomes more chronic and tends to spread to other parts.

with more or less majority. If we consider that probably one of the causes of this 
stromous condition is Syphilis in a diluted form then we can understand that the 
inflammatory changes would be of a low type: or 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 Sarxula 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 similarly when we know more fully of the 
relations of the tubercle bacillus with disease, the meanings of such words as sarxula or 
phthisis are more definitely stated: thus at present 'sarxula' is as often used for the 
manifestation of the bacillus in the lymphatic system, as for the condition of the lungs suitable 
for germ growth: also as regards the 
term "tuberculois", that is perhaps the most 
desirable as any, although there are cases 
in which no tubercules are ever formed: 
and there are other cases having small nodes 
in several various organs without there 
being any connection with the tubercule 
bacillus. To return, however, that 
favorable condition for germ growth which
has been called sarcoplasm may result
from it. 1) Deficient supply of food so to quantity
and quality -- unadapted to age or
defective in certain elements.

3) 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
or having deficient food are very apt
to develop 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
sarcoplasm parents. These points seem to
have been in great measure understood by
Niemejfer as far as the knowledge of this time
allowed.

Now though the "sarcoplasm condition"
is a frequent fore-runner of pulmo-
ary tuberculosis there are other
conditions which will afford a congenial
home for the germs of in this view, I
think is am supported by Prof. Ziegler of Breslau.

From Notes & Lectures by Prof. Cooninger S. Stewart during
Sessions 1875-76-78. Phil. Univ.

The American Society's Translation of Niemeijer's Lectures
on Pulmonary Consumption; Section on Aetiology.

A text book of Pathological Anatomy and Patho-
genesis 1883 H. T. Chase: on Granuloma.
Mae Allister: "there favorable conditions for the growth of the bacilli are brought about by inhalation of dust in various forms, or result from other diseases as measles, whooping-cough, whooping pneumonia, bronchitis, or may be induced by influences which weaken the body (without the production of the atrophic condition), such as defective food, ventilation or exercise, debility, etc. Why should want of exercise, bad food or debilitating diseases or make a person subject to tubercle of the lungs? Referring first the tissues of the body generally are capable to resist disease, it with 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 if one part of the lung is more at rest than another, there will be the usual starting place of this disease; and as we find it, the apex is the most immobile part of the lung, i.e. almost invariably the seat of early phthisis." The apex movements are limited by their position or by the frequent cohesion of the pleura: cit. p. 10.
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 apexes 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, so that "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, so to quote Prof. Burton Sanderson further, "retardation of the stream particularly in the veins is a condition sine qua 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. Noting for discussion of other causes supposed to coincide to apex disease Gulstonian Lectures on Pulmonary Cavities by W. Stewart M.D., reported in Brit. Med. Journal 1872 Vol. I p. 369.

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

The following seems to be an example of such cases:—

J.W. female aged 27 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 and suffered much from dyspepsia with its usual accompaniments. At the beginning of Dec. 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; percussion above and below clavicle showed inspiratory sounds to be prolonged; the expiratory short, occasional, with fine crepitations.

Temperature varied from normal to 101.5° b ut there was no delirium. The treatment consisted in regulating the stomach and 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. Examinations three months later showed no evacuations. The patient was able to work, not no Raeille were detected. By the end of January 1833 Miss J.W. was able to go about, was eating and digesting well on a varying weight. She was then taken on for further study (She is now well Aug. 10 34).
But there is a further stage of acinar pleurisy, inflammation of the lung in which the exudation breaks down; that condition is favorable for the growth of the tubercle bacillus, especially as when the pneumonia involves the apex either primarily or by extension. That this does happen is shown 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 forerunner 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 espoused into the lungs without any phthisic results, 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 J. Thompson who has specially studied "The causes & results of Pulmonary Haemorrhage."

Haemorrhage." He points out that there are three sources of bleeding connected with the disease: (1) First, as a result of confirmed phthisis; (2) Second, as an early complication of the congestive forms of the disease; and (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 clot of blood may become hard and gradually shrink and contract, giving no further trouble; or they may break down and 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 form was the link in the chain which would have shown Learm that it was possible "to understand both haemoptysis could lead to tuberculosis" and that in supposing "that phthisis was always caused by a new growth," he was nearer the truth than he himself knew.

The notes of the Leipzig at the time of the translation.

+ Reiner: op. cit. Tom. ii p. 118 et seq.
The positions into which the blood goes have been described by R. S. Thompson. These are: the summit of the middle part of upper lobe, the middle axillary region, 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 as much as the apex does; but although excavations are caused in the base of the lung by the coagulation of the haemorrhagic fluid, 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 unlike the upper lobe of the lung afforded facilities for the production of phthisis, which the other parts do not possess, the coagulated blood left by the coagulated modules (abscesses, &c.) would heal as readily there, as in the other more mobile portions of the lung. These facilities have already noticed (page 11). The above explanation is given in order to the causative Pulmonary cysts which 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, pneumonitis, inhalation of foreign bodies, and wounds of injuries of the lung inflicted from without. The case recovers from damage which in the apex would surely be followed by tuberculosis.

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 Phtisis into Inflammatory, Tubercular, or mixed forms does not seem to be so very far wrong; including in the term "Phtisis" or "Consumption" of the lungs the various destructive diseases which are now getting differentiated as specific tuberculosics. stands out clearly as either a primary disease, or one superadded to another already existing; just as whooping-cough may attack a child it may have, or may follow in the wake of some other disease such as measles. The number of disorders upon which the (bacteria)
bacilli of tuberculosis may succeed fully develop to the extent that diversity must exist in the modes of treatment after the germ has been got rid of; still once the lungs are clear of the germ, they are placed in a position to recover, if the general and local "tone" can be improved, (this of course is generally being attempted while the extermination of the germ is going on); if the original conditions are left pretty much in the same as when the bacilli first found the thing begins, will very soon find their way back again. An example of this might be taken those cases in which the disease becomes quiescent after residence at high altitudes or under anti-septic treatment, best returns or breaks out again on the withdrawal of an aseptic or anti-septic atmosphere.

Diagnosis is becoming more and more scientific exact, it is well that it is so, but it should always be kept in mind that the aim of 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 of cure of the sufferer, consequently, in a disease such as we are now considering to be able early to recognize 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 shown 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 Dr. C. Theodore Williams informs me that Dr. Percy Kidd, the Pathologist at Bromsgrove Hospital, &c., 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. Penfick) & so into the veins, with the production of tubercles in the walls; therefore when the inoculation is in that manner certain must be found at one time or another in the blood but there


there are other means by which the bacilli may reach the lungs: 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 and may at once attack the alveolar epithelium, the disease spreading by continuity to other alveoli: this, of course, occurs when the local conditions are favorable, but Dr. Theodore Williams thinks that it is possible, if "(the germ is)" in great number and under especially 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 pre disposed 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 or lymphatics "escape from them a space into the alveolar epithelium where they grow, causing multiplication of the epithelial cells." The "Report on the Relation of Micro-organisms to Tuberculosis," Practitioner, April 1883, p. 141. [1883 Oct 12] Speech made at meeting of the Medical Society of London Feb. 12, 1883, and 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 tuberculous, or by the submucous alveolar tissue being attacked; through the lymphatics, this latter form may also be produced; while by the bronchii 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 tuberculous 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 of the lung of the body. These ways are (1) by extension from disease existing in some other part of the body, when it is easily understood that the same may be conveyed to the lung, examples of this are quite common. &

(2) entrance to the pulmonary lymphatics may, I think, be given by those small openings the pseudo-atomata of the bronchi. I have no where seen this mode
mode described, but on considering the matter
swas led to think of cases of Anthracosis and
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
preconceptions, what is the 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
capillaries into the peri-bronchial lymphatics
and thence into the peri-vascular act. (from
description given by Prof. D. J. Hamilton in Practical
Pathology, class) These vessels run outwards to
form a dense plexus in the inner layers of the
pleurisy which is as often affected when
pulmonary is present. The cell elements of the
peri-vascular sheaths proliferate forming
little round swellings which press into the
lumen of the vessel which they either
obliterate, or burst into as from the other
act - 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 or lymphatic
vessels"; so that would certainly be
possible if the mode of entrance was
I have stated. If the tributaries 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 whereby the germs through the lymphatic or blood vessels, "is a purely gratuitous assumption as the bacilli easily find their way into the bronchial 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 O. Spina goes much further. Denies even the existence of the tubercle bacillus: said Prof. Touraixaint of Toulouse has been shown by Watson Cupple to have erred on this point as Spina, although an assistant in Prof. Stricker's Institute may also have fallen into mistakes. If the germs 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. (Ibid.)

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

Pfeiffer: Berliner Klinische Wochenscrlift Jan 16 1883
Balmer & Fränkel. Ibid: No 44 1882
Enttmann. Ibid: No 52 1882
Dettweitgen & Meissner. Ibid No 7 1883
Le Fosse: Revue Médicale de la Suisse Romande Dec: 1882
Lichtenstein: Fortschritte der Medizin Vol I 1873
Licht: Deutsche Med: Wochen: No 5 1883
Hillis: Ibid: No 47 1882
Dunschield: Brit Med: Journal 1883 Vol V p 206
Marchiafava & Celli: Gaz: de psi: Oct 15 1883 Oct 29
Williams: speech quoted (Feb 1883): among others were:
Ziegler & Macalister. Op cit. Art 122
Ehle: B. M. J. 1882 Vol I p 916 Vol II p 725
Reid: Glasgow 13 B. M. J. 1882 Vol II p 142
Hannay: Celtic: B. M. J. 1882 Vol II p 788
Nigral: B. M. J. 1882 Vol II p 826
Bannister: B. M. J. 1882 Vol II p 1096
Negri & Pinolini: Lond Med: Record Jan 15 1873 p 26: describe five forms which are probably the Bacillus in different stages of development.
in well-marked cases of the disease, to demonstrate the bacilli are due to the methods used for collecting the sputum for examination. The best time for obtaining the sputum is the morning after the patient has fasted for some hours; the mouth should be washed out before quitting, to remove the Lepthotrichus Brocalis; the sputum, received in a bottle previously washed in carbolic acid, should be covered with alcohol as soon as possible; the preparation is then ready for storing or examination whenever it may be required. It is best to examine the sputum several days in succession.

A simple method of examining the breath for germs has been described by R.C. Smith, M.D., 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 respiration. Dr. W. Roberts of Manchester has shown to the Royal Society a method of examining the breath; Dr. Ramsom and Mr. W. Williams have also devised plans for the examination of the air in rooms, hospital wards, ventilating shafts, etc. But it would not be necessary

†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 these parts of 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 drainage 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 infected from it. He points out that a simple chronic laryngitis may develop into tubercular. So, a tubercular, or "suspicious" bronchitis may also lead on to the similar disease of the lungs. That so-called "neglected cold" may lead to tubercle germs 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 casual Jones numbers of Phthisis Pulmonalis but the subject is a wide one and I feel that there is a great deal of information needed before we can fully understand all the facts. "Phthisis of the larynx" by Dr. Med. Journal Nov. 22, 1883. 18. 94.
the relations existing between the germ and the body. But aside 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 or strengthening of the constitution, or thus enabling it to withstand the attacks of the bacillus, will be found most effective in the long run, though I would not exclude anti-septic 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. Or—
“Educate your eye to distinguish what is said, and thus render yourself, in some measure independent of this failing organ. And thus it may be you will make a valuable provision for the future.”