THESIS FOR THE DEGREE OF M.D.

Observations on diseases of the chest
in ten years of general practice.

by

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Having been in practice now for over ten years on the North-east coast of England, one cannot help being struck by the numbers of patients suffering from diseases of the chest.

The commonest and therefore, the most important is undoubtedly Pulmonary Phthisis.

The first question that naturally arises in one's mind is: Why should Phthisis be so common? For the answer to this important question, we must first consider the Causes of Phthisis.

1 Heredity. By this is meant not that the disease itself is transmitted from parent to offspring, but that a certain condition of the tissues is transmitted which makes them liable at any time to start a Tuberculous process. Dr J.W. Moore in a paper read before the Section of Medicine, British Medical Association at Carlisle July 31st, 1896 says:— "Another point which has been removed from the pale of discussion by the discovery of the Bacillus Tuberculosis has reference to the influence of Heredity upon the transmission of the disease."

"That Phthisis propagates itself" writes Hirsch, "in many families from generation to generation is so much a matter of daily experience, that the severest critic can hardly venture to deny a hereditary element in the case." "Heredity in the narrower sense" - to borrow Hirsch's words - "which consists
in the transmission of a specific poison something like that of Syphilis, can no longer be regarded as an aetiological factor in the every-day forms of tuberculosis."

"Susceptibility" says Dr James E. Russell in his Report on the Prevention of Tuberculosis, (Glasgow; Robert Anderson, 1896, Page 18) "may be constitutional, and may be so great and so marked as to amount to a predisposition. This it is which passes by inheritance, and, until the discovery of the Bacillus, was regarded as hereditary tuberculosis. It is not the disease which is inherited. It is the predisposition the feeble constitution, the low vitality, the tout ensemble of conditions, some of which are recondite and imperfectly understood, some obvious and capable of specification and comprehension. The outcome is a soil, so congenial as to accept a delicate infection from which the majority of mankind emerges scatheless; a soil which sustains & propagates the bacillus so vigorously that to popular observation the disease seems to be inherited. In such a person a blow or a sprain, a cold or a sore throat, determines the local incidence."

This question of heredity is a most important one. In the large majority of cases there is no difficulty in tracing the taint in the family history. In fact so much is this the case, that one is almost tempted to ask, is it possible to get true Pulmonary Phthisis without some hereditary predisposition to the disease?
Dr Carter in his "Elements of Medicine" page 91 says "a predisposition on the part of the individual attacked is necessary, without which tubercular disease does not occur." Now that the infectious nature of Phthisis is all but universally acknowledged this statement may be considered erroneous.

Bad Ventilation and unhealthy sanitary surroundings have always been recognised as at least making people more susceptible to the disease.

Climate. That Phthisis is more common in a damp changeable climate such as we have on the North-east coast is an undisputed fact.

Syphilis. People who have had Syphilis are more prone to Phthisis on account of the debilitating influence it has on them.

Fevers. Especially those that are liable to be followed by lung complication, such as Measles & Influenza, which are often complicated by Bronchitis and Pneumonia.

Confinements and any debilitating influences.

Alcoholism. Not only does this act as a direct cause by making the tissues more susceptible to the tubercle bacillus, but also as an indirect one from the exposure to which any one under the influence of alcohol is liable.

Injuries. In 1890 I saw a young man (previously healthy) who had (whilst playing with a metal dart tipped with wool) instead of blowing the dart out of the straight tube in which it was placed
to aim at a target, drawn in his breath, and inhaled the dart. I saw him three months after the accident when he told me what had happened. He had then all the symptoms and signs of Phthisis, with elastic tissue and Bacilli in his sputum. The dart was coughed up, the wool being separated from the metal after being in his chest for about 4 months. He eventually died of Phthisis.

There are many other causes which might be enumerated such as dusty trades, but it is unnecessary even to mention them, because since the discovery of the Bacillus Tuberculosis by Koch in 1882 it is now, by the majority held that Phthisis is an infectious disease, & that the introduction of the Virus is the one exciting cause of the disease, the others being only predisposing causes by making the tissues a better nidus for the growth of the bacillus.

Although the consensus of opinion is in favor of the Infection or Contagion of Phthisis, such authorities as Sir Douglas Powell & Dr. C. Theodore Williams hold a contrary opinion.

Sir Douglas Powell in his book on diseases of the chest, page 348 says "My own personal experience & observations convince me, that, apart from artificial conditions, such as those brought about by experiment & in the ordinary circumstances of life phthisis is not an infectious malady."

Dr. Williams in Quain's Dictionary of Medicine page 400 says "The evidence of the Brompton and the
Victoria Park Hospitals negatives the idea of contagion such as is present in Small-pox or Scarlet fever, for it has been demonstrated that the percentage of acquired phthisis occurring among the resident staff of these institutions is less than that of most general hospitals; and even when any defective ventilation & overcrowding has given rise to evil consequence, these have shewn themselves in outbreaks of erysipelas and sore-throat & not in tuberculosis. Nevertheless Phthisis has been shewn to have been communicated by inhalation on some very rare occasions, under the following circumstances:

(1) Close intimacy with a consumptive patient in advanced disease, such as sleeping in the same bed or room.

(2) Activity of the tubercular process, either in the way of tuberculosis or excavation.

(3) Neglect of proper ventilation of the room occupied."

Dr Osier in his "Principles and Practice of Medicine" page 189 says "it has been fully proved that the expired air of tuberculous patients is not infective. On the other hand the virus is contained in enormous amounts in the sputum, which, when dried, is soon widely disseminated in the form of dust, & unless carefully sterilized constitutes a great medium of transmission.

A belief in the contagiousness of pulmonary tuberculosis has existed from the days of the early
Greek physicians and has persisted among the Latin races.

The investigations of Cornet afford conclusive proof that the dust of a room or other locality frequented by patients with pulmonary tuberculosis is infective. The bacilli are attached to fine particles of dust & in this way gain entrance to the system through the lungs. The following are some facts in favor of this view:—

(1) Primary tuberculous lesions are in a majority of all cases connected with the respiratory system. The frequency with which foci are met with in the lungs & the bronchial glands is extraordinary, and the statistics of the Paris Morgue show that a considerable proportion of all persons dying of accident or from suicide present evidences of disease in these parts.

The post-mortem statistics of hospitals show the same widespread prevalence of infection through the air passages. Biggs reports that more than 60% of his post-mortems showed lesions of pulmonary tuberculosis.

In 125 post-mortems at the Foundling Hospital New York, the Bronchial glands were tuberculous in every case.

In adults the bronchial glands may be infected while the individual is in good health.
H.P. Loomis found in 8 of 30 cases in which there were no signs of old or recent tuberculous lesions that the bronchial glands were infective to rabbits.

(2) The greater prevalence of tuberculosis in institutions in which the residents are confined and restricted in the matter of fresh air & a free open life condition which would favor, on the one hand, the presence of bacilli in the atmosphere, & on the other, lower the vital resistance of the individual.

The investigations of Cornet upon the death rate from consumption among certain religious orders devoted to nursing give some striking facts in illustration of this.

In a review of 38 cloisters, embracing the average number of 4028 residents, among 2099 deaths in the course of 25 years 1320 (62.88%) were from tuberculosis.

In some cloisters more than 4/5ths of the deaths are from this disease, & the mortality in all the residents, up to the 40th year, is greatly above the average, the increase being due entirely to the prevalence of tuberculosis.

It has been stated that nurses are not more prone to the disease than other individuals, but Cornet says that of 100 nurses deceased, 63 died of tuberculosis.

The more perfect the prophylaxis & hygienic
arrangements of an asylum or institution the lower the mortality from tuberculosis. The mortality in prisons has been shown by Baer to be 4 times as great as outside.

The death rate from phthisis is estimated at 15% of the total mortality, while in prisons it constitutes from 40% to 50% and in some countries, as Austria, over 60%.

Flick has studied the distribution of the deaths from tuberculosis in a single city ward in Philadelphia for 25 years. His researches go far to show that it is a house disease, about 33% of infected houses have had more than one case. Less than one-third of the houses of the ward became infected with tuberculosis during the 25 years prior to 1888. Yet more than one-half of the deaths from this disease during the year 1888 occurred in those houses.

There are, however, opposing facts. The statistics of the Brompton Consumption Hospital show that doctors, nurses & attendants are rarely attacked.

Dettweiler claims that no case of tuberculosis has been contracted among his nurses or attendants at Falkenstein.

(3) Special danger exists when the contact is very intimate, such as between man & wife.

On this point much difference of opinion exists, but the figures seem to indicate that under these circumstances the husband or wife is much more liable
subsequently to die of consumption.

Of 427 cases of Pulmonary tuberculosis at the Johns Hopkins Hospital, in 25 either husband or wife had been infected with it or had died of tuberculosis.

In response to a question as to contagion, asked by the Collective Investigation Committee of the British Medical Association, there were 261 replies in the affirmative, among which were 158 cases of supposed contagion through marriage.

Weber's cases are of special interest. One of his patients lost four wives in succession, one lost three, and four lost two each."

Dr Foot (Dictionary of Medicine Vol. 3 page 1136 & Dublin Journal of Medical science Vol. 64 Augt. 1877 page 162) recalls the fact that in the works of Aristotle is to be found the earliest notice of the opinion that phthisis is infectious.

In 1799 Laennec, while examining some tuberculous vertebrae slightly grazed the forefinger of his left hand by a stroke of the saw. The scratch was so slight that he paid no attention to it; but, on the following day it was slightly inflamed, & a small roundish tumour, apparently confined to the skin, formed on it almost without pain. At the end of eight days the epidermis cracked, displaying a small tumour within, yellowish, firm and in every respect like a crude, yellow tubercle.

Laennec cauterised it with the deliquescent
Chloride of Antimony. He felt no pain & in a few minutes after the fluid had permeated the tumour, he detached it by a gentle pressure. The part soon healed, & twenty years afterwards he had found no further effects from the accident.

Apparelly with his personal experience in mind, Laennec (quoted by Dr. Arthur Ransome in "The Treatment of Phthisis" page 41) in his "Traite de l'auscultation Mediate" expresses the opinion that "no consumptive succumbs to a first attack of the tuberculous affection". In 1822 he was obliged to give up his practice owing to pulmonary phthisis, of which he died in 1826.

Before discussing the infectiousness of Phthisis, it will be well to review the prevailing opinions as to the pathology of this affection during the first half of the present century.

Laennec's views were first published in 1811. He was the originator of the unity of Phthisis. All phthisis he regarded as "tubercular". He looked upon tubercle as an "accidental product" & the many modifications found in the lungs and other organs in the case of tuberculosis as caused by the evolutions of a unique, heterogeneous substance "tubercular matter".

Laennec saw identity of nature amidst apparent diversity of form & aspect, & he taught that isolated tubercle, grey granulations & tuberculous infiltrations grey & yellow, were all of the same pathological nature.
and origin. "The synthesis which he constructed of the different tuberculous processes has triumphed over all the attacks which it had to undergo, & has only now found its definite confirmation in the most brilliant conquests of modern technique (Herard et Cornil "Le Phtisie Pulmonaire")

Laennec also maintained that tubercle was not only a substance foreign to the organism, but that it led a special life; the idea of its specific nature was distinctly in the mind of this great observer.

These opinions of Laennec were violently attacked by Broussais (1826) whose views met with considerable favor in this country. Broussais maintained, in opposition to Laennec, that tubercle was purely and simply the result of inflammation, and that organs "irritated" to a certain degree & for a period more or less long ended by becoming tuberculous when they were so disposed. Tubercle, he said, was caused by chronic inflammation of the air passages and was not therefore in any sense "specific".

Broussais also included the pulmonary lymphatics in the tuberculous process. Tuberculisation, he affirmed, was attended by an inflammation of the lymphatic capillaries.

The two essential points in the doctrine of Broussais as to the nature of pulmonary tuberculosis - viz: irritation as the cause & a lymphatic product as the effect - have held up to quite recent times a
prominent place in the history of the pathology of Phthisis. Louis shared the views of Laennec, but Andral, Cruveillier, & Bouilland (1837) & many others held with Broussais that tubercle was a result of inflammation and therefore non-specific.

In 1844 Lebert attempted to demonstrate a specific histological structure in tubercle, but in this he signally failed, and his observations & conclusions were soon shown to be erroneous.

In 1850 Laennec's doctrine was vigorously attacked by Reinhardt, & later by Virchow, the great authority attaching to whose name no doubt shook unduly the confidence which, up to that time many had felt in the teaching of Laennec.

Reinhardt declared that most of the anatomical appearances recognised as tuberculous were essentially inspissated pus, together with epithelial cells, filling the pulmonary alveoli, & were the results of catarrhal pneumonia.

Virchow limited the application of the word "tubercle" to the grey granulation, and all the other forms of tuberculisation met with in phthisical lungs he declared to be the inspissated products of inflammation; and that phthisis, properly so called, was rather the result of caseous hepatisation than of tubercle.

He reserved the name "tuberculous" to acute military tuberculosis. The idea of the unity of phthisis as in all cases a tuberculous disease was
declared by him to be erroneous, and the greater part of pulmonary phthisis to be due to inflammation.

Thus we see that for Laennec and his followers there was but one kind of phthisis — i.e., tuberculous; and for Virchow & his followers there were at least two, one a caseous pneumonia, and the other a rarer form — i.e., the tuberculous.

It was at this time that Niemeyer's teaching became prominent, & he maintained that phthisis depended on the caseous degeneration of the products of simple inflammation; that when in a phthisical lung the grey granulations & pneumonia co-exist, the granulations were secondary & consecutive, and so arose his famous dictum that "the greatest danger to which a phthisical patient could be exposed was that he should become tubercular"; and so it happened that in this country, as in Germany, Laennec's doctrine was discredited & the belief in the duality of phthisis or in varieties of phthisis became very general, and modifications of the inflammatory process were considered to be the chief agents in its causation.

In France, however, the most distinguished pathologists sided with Laennec & maintained the unity of phthisis as a tuberculous disease; this was greatly due to the labor of Grancher, who by studying and comparing the tuberculous granulation with a fragment of caseous pneumonia, without any apparent granulation, was able to formulate the law that " a nodule of
caseous pneumonia has the same structure as the typical tuberculous granulation”.

This opinion soon secured the support of Reinfleish in Germany and of Wilson Fox in this country, and somewhat later it had the vigorous support of Charcot, who was then professor of pathological anatomy in the Paris Faculty.

We now come to the period of the discussion in the Pathological Society of London (Transactions of the Pathological Society of London 1873 Vol. 24 p.p. 284 et seq.).

But I will refer first to the year 1866 when Villemin claimed to have discovered the inoculability of tubercle; for this may be regarded as the initial step in connection with a vast amount of careful investigation, which led ultimately to the discovery, published some 16 years later, of the true nature of tubercle and the true pathology of pulmonary phthisis.

The sensational character of Koch’s discovery threw somewhat into the background the merits of the distinguished Frenchman whose investigations into the inoculability of tubercle started the labors of pathologists in the right direction towards the discovery of the truth.

Speaking of Villemin’s work Cohnheim says - "It was at this epoch (1860-1870) that a discovery was made in France, whence will date, if I am not mistaken, in the history of tuberculosis, not only an incomparable
progress, but also a complete transformation in our manner of regarding the disease. Few discoveries, indeed, were capable of stirring medical opinion to so great a degree as the demonstration by Villemin of the transmissibility of tuberculosis. 

Villemin's observations form so important a step in the modern advance towards a true pathology of phthisis that it may not be out of place for me to give his own account of his methods of procedure, more especially as it was to be soon after imitated in this country with the remarkable result of negating the true conclusions of Villemin, and so leading to a retrograde movement and to a retardation of the acceptance of the true pathology of phthisis. For Villemin's conclusions were correct, whereas the conclusions of his critics and imitators in this country were erroneous.

Villemin in describing his own procedure says:

"If we make in the ear of a rabbit, or in the groin or axilla of a dog, on a very narrow surface previously shaved, a subcutaneous wound, so small and shallow that not the smallest drop of blood shall appear, and if we insinuate securely & carefully a portion of tuberculous matter the size of a pin's head, taken from a cow or rabbit already rendered tuberculous; or, if on the other hand with a hypodermic syringe we instil under the skin of an animal a few drops of phthisical sputum, made more fluid by the addition of a little water, this is what we observe. The day after the operation the
most careful palpation finds no trace of the matter inoculated - the margins of the wound are united - then, after at most four or five days, a slight swelling is observed, accompanied sometimes by heat & redness, & we can watch the progressive development of a local tubercle, varying in size from that of a hemp seed, to that of a filbert.

For some time after the inoculation the animals present no appreciable alteration in their health. It is only at the end of from 15 to 30 days that they are seen to be losing flesh.

Some recover from this and put on flesh; others go on getting progressively weaker and fall into a state of marasmus, are often attacked with colloquative diarrhoea, and perish in a state of extreme emaciation.

On post mortem examination we find that the tubercles at the seat of inoculation are formed of a caseous mass around which are very often seen small yellowish granulations infiltrated for some distance in the intermuscular connective tissue.

The lymphatic glands communicating with the seat of inoculation are frequently swollen, present scattered granulations or tuberculous nodules, and sometimes end in complete caseous transformation.

We generally find tubercles in the lungs, but the eruption of tubercles is not limited to these organs. It appears more or less abundantly in the lymphatic glands, the intestines, the liver, the spleen, the kidneys etc.
These organs are often stuffed with tubercles. The serous membranes and notably the omentum and the mesentery, are sometimes riddled with innumerable granulations.

According to the lapse of time since the inoculation & the greater or less rapidity with which the eruption occurs, we find the tubercles either grey and transparent, or yellow, caseous & softened, or ulcerated and excavated. If the animals are killed before the 15th day it is rare to find tubercles in the organs.

There elapses between the time of inoculation and that of the tuberculous eruption a certain time, which has appeared to us to vary from ten to twenty days". (Herard et Cornil "Le Phtisie Pulmonaire, 2nd edit. Paris 1888, p.23).

It was found that caseous matter had the same infective properties as the grey granulation.

An important & influential academic commission confirmed the results announced by Villemin.

Objections to his conclusions, however, arose in various quarters, and in this country Wilson Fox and Burdon Sanderson sacrificed a great number of rodents with the object of testing the correctness of Villemin's observations and inductions, and the results they arrived at had the effect, they maintained, of negating his views. They stated that the results he had obtained were not peculiar to tubercle, but might be produced by almost any substance - pus, fragments of
bone, bits of cork, of india-rubber etc., but it has now long been admitted that these observations and experiments of Fox and Sanderson, which produced so great an effect on professional opinion in this country at the time, and which were thought by many to be fatal to Villemin's conclusions, were valueless on account of the absence of those antiseptic precautions which have since been shown to be needful in the conduct of such experiments.

Villemin, however, found an able defender in Chauveau, whose skilful experiments & observations on the transmissibility of tubercle in oxen & horses afforded ample corroboration of the conclusions of Villemin. While these facts were fresh in men's minds the discussion at the Pathological Society of London was instituted in 1873. Although it was nearly ten years later that the parasitic and infective nature of tubercle became incontrovertibly proved through the discovery by Koch of the bacillus of tubercle, the idea that tubercle was of this nature was prominent in many minds at the time of this discovery, & it is not a little remarkable that this idea should be almost ignored by those who took part in this debate. Indeed it is clear that the accuracy & conclusiveness of Wilson Fox and Sanderson's observations & experiments on the rodents are assumed by the debaters generally, and Villemin's labors & results are scarcely alluded to.
From brief extracts from this debate (Transactions of the Pathological Society of London 1873) one can form a good notion of the differences of opinion that existed amongst eminent pathologists as to the true pathology of phthisis.

Dr Burney Yeo in mentioning this debate in an address delivered before the Torquay Medical Society said, "It is exceedingly interesting in connection with this chapter of pathology to find the opener of this debate, evidently after much tribulation of mind & sorely against his preconceived views, forced to the conclusion from the results of his own elaborate and very careful observations that phthisis is, in all its forms, one and the same disease:-- i.e. a tuberculous one: so that with some hesitancy and here and there a little lack of clearness, the outcome of his researches was to re-establish the doctrine of Laennec of the unity of phthisis."

The opener of the debate, Dr Wilson Fox, says:-- "Fifteen years ago I came from Germany strongly impressed with the opinion that phthisis might be subdivided into many absolutely diverse diseases--diseases diverse in their essential nature as well as in their apparent origin. I had a great many terms at the end of my tongue--broncho-pneumonia, caseous pneumonia, and scrofulous pneumonia--but when I myself became a teacher I felt great doubt & difficulty in saying what was not tubercle, & still more in saying what was tubercle. I came at last to the conclusion
not to speak as I did in my earlier days, describing a lung as containing no tubercle because it did not contain what I imagined to be the only type of tubercle." And again he says: "It was my anxious wish to do something in my day & generation towards finding out a great many varieties of phthisis."

Dr. Bastian (page 317) emphatically asserted that phthisis was an affection "of which tubercle could no longer be considered as the pathological essence..... Laeencec's central idea had been shown to be erroneous ....... Phthisis had now been fully shown to be a non-tuberculous affection". He concluded that "the common varieties of pulmonary phthisis were to be regarded as due almost solely to various forms of chronic inflammatory changes in the lung; tubercle was to be regarded as a mere occasional and quasi-accidental complication; both phthisis and tubercle were robbed of their so-called "specific" attributes."

Douglas Powell (p.p. 336 & 337) maintained "that tubercle could not be regarded as the essential specific element in phthisis". He thought that "much of the inveteracy of phthisical lesions would be explained without the aid of any precedent deposit of tubercle".

Dr C.J.B. Williams (p. 342) said, "the consolidation of scrofulous pneumonia and similar low inflammations induce consumption of the lung in the same mode as tubercle" he was therefore opposed to the idea of unity.
Dr Green (p.345)said,"I wish to be understood to express the belief that the various anatomical changes met with in the lungs in phthisis are the result of inflammation,and that the differences in their anatomical characters,and in the subsequent history of the newly formed elements is mainly due to differences in the intensity and duration of the inflammatory process."

The idea of unity was strongly opposed by Dr Cayley(p.314)who said he would restrict the term tubercle to the grey granulations and thought the change in the lungs in many cases of phthisis as "entirely due to inflammatory processes" and that in some cases tubercles were developed secondarily to inflammatory changes which constituted the primary affection.

The late Dr Moxon was strongly in favor of the unity of phthisis,he believed all phthisis was tuberculous,but he thought the notion of tubercle as a specific growth had received a serious blow from the results of the inoculation experiments of Sanderson & Wilson Fox,and thus we see he was led astray by the prevailing antagonism to the idea of specificity and by his belief in the results of experiments which proved to be fallacious.

Wilson Fox observed at the end of his argument "There are no subjects in medicine which would,I think so dispel a spirit of dogmatism & exclusive adherence
to one's own opinions as the study of phthisis, on which such differences have existed, and do exist, among the greatest men of the past and present, as may well make any one doubt the accuracy of his own observations and the justness of his own conclusions.

The following is the manner in which he formulates the doctrine of unity: "The destructive changes in the lungs characterised by the term phthisis are almost invariably associated with, and in great measure caused by, a series of cell-growths which in their anatomical structure & vital characteristics are practically identical in all the forms of the disease.

Caseation he regarded as due to the destruction of the capillaries of the new formation, & he recognised that the new growth was capable also of undergoing a development into fibroid tissue. Tubercle he defined to be, "a lymphatic overgrowth produced by irritation under special circumstances of anatomical or constitutional predisposition ...... when you give rise to irritation in cases where lymphatic irritability is excessive, under these circumstances you may get tubercle. You may therefore get it from a blood state in which the relations of the lymphatics are abnormal, or you may get it locally by exciting inflammation."

"The anatomical and vital characters of those tubercular growths are distinctive, but they cannot be called specific, but cases where any close similarity exists between simple inflammation & tubercle are,
however, rare, & even then it is a superficial resemblance, rather than an identity of structure."

"In those acuter forms of phthisis — the so-called scrofulous pneumonia — the process is identical step by step with the majority of the processes that occur in acute tuberculisation, and differs from it only in the extent of pneumonic exudation."

"Chronic phthisis is distinguished by the characteristics of induration of the lung. ....the chronicity depending upon induration of those growths rather than on their acute destruction."  "Every indurated form of phthisis, most cases of so-called fibroid phthisis of some English authors .......come under the category of indurated tuberculosis."

He denied or doubted the origin of phthisis in catarrhal pneumonia, scrofulous bronchitis, and scrofulous pneumonia.

He pointed out that "the products of ordinary inflammation are in the lungs, as well as in the serous and mucous membranes, almost always found co-existing with tubercle .......the tuberculous growth is sometimes superadded to this, and gives to the pneumonia characteristic features ....anatomically pneumonia is not tuberculous unless that growth co-exists in the alveolar walls."

Wilson Fox's conclusions went far to re-establish the unity of nature and origin of phthisical processes — they went to disprove the then common belief that pulmonary phthisis could originate in
ordinary inflammation, and they disposed of much of the erroneous opinion then existing about "caseous pneumonia", "scrofulous pneumonia", "fibroid phthisis" and the like.

The idea of the infectivity & transmissibility of tubercle, so little dwelt on in this discussion, had however taken deep root in the minds of many Continental pathologists, & between the date of this discussion (1873) and the announcement by Koch of the discovery of the Bacillus of Tubercle in 1882, a great deal of good work was done in this direction with the effect of preparing men's minds for the great event.

Chauveau's observations corroborated those of Villemin. Observing that the infective virus was sometimes found in the fluids of the phthisical, he came to the conclusion that this consisted of very minute particles, "imperceptible even under the microscope, inert in appearance, but endowed, in reality, with a singular virulence."

Buhl in 1873 & Klebs in 1875 expressed their belief that bacteria were the cause of tubercle, & that they probably existed in the caseous masses.

In 1877 Klebs professed to have isolated and cultivated the "monas tuberculosa" but he failed to establish its specific character.

Toussaint (1881) Baumgarten & others also discovered microbes which they thought might be the active agents in tuberculosis.

Professor Bouchard in 1881 argued vigorously
On general grounds that phthisis must be classed amongst the infective diseases, & the next year (March 24th, 1882) R. Koch announced to the Physiological Society of Berlin that he had succeeded in isolating & cultivating the microbe of tuberculosis, & it was thus left to a German to give absolute demonstration of the causal unity of phthisis in all its various manifestations, as had been taught at the beginning of the century by Laennec.

It is not possible to over-estimate the importance of the results which followed this discovery.

The discovery of the true pathology of phthisis has been attended with practical advantages of the most remarkable kind; a flood of light has been thrown on its etiology, & the knowledge of the true nature & mode of propagation of this disease has led to a vast improvement in our methods of dealing with it, both in the way of prevention and cure.

This is proved by the striking reduction in the death rate from phthisis.

From 1858 to 1860 the death rate per million from phthisis was 2565; from 1889 to 1893 the death rate had sunk to 1512, a reduction of 41%.

The recognition of the infective & communicable character of the disease has doubtless had a great share in producing this admirable result.

At the present time phthisis is recognised by the great majority to be a communicable disease, and
many facts can be brought forward in proof of this.

Koch in his paper read before the Physiological Society of Berlin on March 24th, 1882, founded his claim for the recognition of the bacillus tuberculosis as associated with tuberculosis on the following facts.

He found the bacillus present in the following cases:

1st. in the human subject -
- 11 cases of miliary tuberculosis.
- 12 cases of "cheesy bronchitis" & pneumonia (in 6 of these cavities had formed)
- 1 case of tumour of brain, of the size of a hazel nut.
- 2 cases of freshly extirpated scrofulous glands.
- 2 cases of synovial degeneration of joints.

Twenty-eight cases in all.

2nd amongst the lower animals -
- 10 cases of perlsucht of the ordinary type.
- 1 case of caseous cervical gland in a pig.
- 1 case of a hen which died of tuberculosis.
- 3 cases of spontaneous tuberculosis in guinea-pigs.
- 7 cases of spontaneous tuberculosis in rabbits.

Thirty-one cases in all.

"Besides these cases of spontaneous tuberculosis, I examined" says Koch "172 guinea-pigs, 32 rabbits,"
5 cats, all of them infected with tubercle by the inoculation of the most varied tubercular substances, such as, grey and calcified tubercle of human lung, phthisical sputum, tuberculous masses from spontaneously diseased monkeys, rabbits & guinea-pigs, pieces of lung from cattle suffering from perlsucht, cheesy as well as calcified, & lastly, by inoculation from tubercular affections produced in animals by inoculation."

In all of these cases 268 in all, Koch found bacilli, & in some they were exceedingly numerous.

So much then, in proof of the statement that this particular bacillus is associated with tubercle.

It still remains, however, to see the evidence given by Koch that this bacillus, & this only is the cause of tubercle. To prove this he carried out a series of experiments, in which he took tuberculous particles from animals which had either died from tubercle, or having tubercle, had been killed for experimental purposes. These particles were about the size of millet-seeds, & having been removed from the body were placed on blood serum from the ox or sheep with every precaution necessary in making "cultivation experiments".

He had for his object in these experiments to obtain the bacillus free from taint, & this he claimed to have done.

After describing in his lecture how he planted the tuberculous particles on the serum, watched their slow growth, and noted their peculiarities, he makes the
following important statement. "The extremely slow growth, which alone is to be observed at breeding temperature, & the peculiar shovel-shaped, dry & firm condition of these colonies of bacilli, are not to be found in connection with any other known bacterium, so that the confounding of the culture of the tubercle bacillus with that of any other bacterium is impossible & already with only short experience, nothing is easier than to recognise at once accidental contamination of the culture."

These cultivation experiments were carried on for some time. After from 10 to 14 days some of the bacilli which had grown in the prepared serum, were removed to another test tube, and after another 10 days or so, some of this second crop was removed to a third test tube so that the cultivation must have been quite pure. Koch then inoculated certain animals with the pure bacillus obtained by cultivation, every precaution against contamination being taken. In each series of experiments several animals were used, including rats, mice, guinea-pigs, rabbits & a marmot, pigeons and frogs.

He thus sums up the results of these inoculations. "If one looks back upon these experiments, that a not inconsiderable number of animals were experimented upon, on which bacillus culture was brought to bear in very different ways - viz. through simply inoculating into the subcutaneous cellular tissues, through
injection into the abdomen or into the anterior chamber of the eye, or direct into the blood stream, without failing, even in one single instance, to develop tubercle; and there had formed in them not solitary nodules, but an extraordinary mass of tubercle corresponding with the large number of infecting germs introduced."

To counteract any possibility of mistake we must note that in all these experiments of Koch's - thirteen in all - a certain number of animals were not inoculated, but were kept under exactly similar circumstances to those inoculated, they were lodged the same and fed the same although they did not come in contact with the inoculated ones, but not one of them showed any sign of tubercle, either during life or post-mortem. It must be noted too that these inoculations were made with tubercle taken from various sources, some from the lungs, from mesenteric glands, and some from freshly extirpated scrofulous glands, as well as some taken from the lower animals, & that there was no difference in the effects produced nor in the appearance of the bacilli whatever the source of the tubercle inoculated.

Dr Koch makes some very striking observations about certain distinctions which he drew between tubercle occurring spontaneously in an animal, and tubercle that occurs after inoculation.

He bought & examined 100 guinea-pigs all of which he found healthy. Several of them were shut up
in a room with others which he had inoculated with tubercle. In three or four months, but never before that time, spontaneous tubercle began to show itself in the uninoculated guinea-pigs.

In them the bronchial glands were "always found unusually large & purulent, particularly, also in the lungs was to be found a large cheesy mass, with very far advanced breaking down in the centre, so that, sometimes, as in human beings, it had reached to actual cavity. The development of tubercle in the organs of the lower part of the body was very far behind that in the lungs.

The swelling of the bronchial glands, & the commencement of the development of tubercle in the organs of breathing, leave it beyond a doubt, that the tubercle of these animals was an inoculation tubercle, springing from a few, or possibly only one infectious germ, and, on that account, very slow in its progress."

Compare this description with what Koch says about inoculated tubercle: - "The place of inoculation was in the belly of the animal near the inguinal glands.

The first sign of the success of the inoculation was the appearance at the end of a week, of a nodule over the site of the puncture. About the end of the second week, the inguinal glands, beside the wound, began to swell, & sometimes also the axillary glands. From that time the animals grew quickly thinner, and died in from four to six weeks, with marked
tubercular affection of the liver & spleen, those organs having been but slightly affected, as compared with the lungs, in the cases of spontaneous tubercle."

Koch, again, inoculated several animals with certain substances, such as swollen gland, synovial membrane, portion of monkey's lung, which did not contain tubercle, & in no instances did the animals experimented on show signs of tubercle either during life or post-mortem.

Several experiments with sputum from tuberculous individuals are mentioned in Koch's lecture. The sputum was allowed to dry, as it may dry in a room occupied by a patient.

This dried sputum was found to be as fatal when inoculated as is a cultivation when inoculated.

Koch thus sums up & gives the conclusions arrived at by his work:— He says: "All these facts taken together justify the conclusion that the bacilli present in tubercular substances are not merely the associates of the tubercular process, but the cause of it, and that we have before us, in bacilli, the actual tubercle virus.

It is also possible, by this means, to draw the boundary of those diseases regarded as tubercular, which, hitherto, could not be done with certainty.

A decided test for tubercle is wanting, and one man considers miliary tubercle, phthisis, scrofula, perlsocht etc to be tubercle; another man holds, perhaps with equal right, that all these processes of
disease are different.

In the future it will not be difficult to decide what is tuberculous & what is not tuberculous. Not the peculiar structure of tubercle, not its non-vascularity, not the presence of giant-cells will decide the question, but the presence of tubercle bacilli - be it in the tissues by the colour test, or be it through culture on prepared blood-serum.

This criterion taken as a guide, must, according to my researches, stamp miliary tubercle, cheesy pneumonia, "cheesy bronchitis" tubercle of glands, and of the intestines, perlsucht in cattle, inoculated and spontaneous tubercle as identical".

The evidence of the communicability of phthisis is of two-fold origin.

Experimental evidence, which is completely given by Koch in his paper read before the Physiological Society of Berlin on March 24th, 1882.

A record of cases published by eminent and trustworthy medical men.

The following case is recorded in the Annals de Dermatologie et de Syphiligraphie, Vol. 5, 1884 pp. 362, 363 & 364.

A man 70 years old with neither personal nor hereditary tendency to tubercle, had a whitlow on his left thumb. The abscess was opened with a bistoury. Later on, ulceration set in, a little above the left wrist, on its radial side. The ulceration measured 11 centimetres in length, and 1 1/2 to 4 centimetres in
breadth.

M. Hanot detected the tubercle bacillus in pus taken from the ulceration. One year later this man showed signs of pulmonary tubercle. He died, and post-mortem examination showed the presence of tubercle in his lungs. The axillary glands were sound. It is surely not unreasonable to suppose that this man's whitlow was either itself the result of tuberculous inoculation, or was opened by a knife which carried the infection. It seems also reasonable to suppose, that this lung tubercle was caused by infection from the site of the whitlow.

In Vol. 6 of the same Annals p. 658, Dr Tscherning of Copenhagen, reports the case of a woman, 24 years of age, in good health, robust, without hereditary predisposition to tubercle, and showing no evidence of herself having had tubercular disease, who was cook in the house of Professor H.

He died in 1884 of pulmonary consumption, which had lasted about six months. During the last days of his illness, his expectoration was a "pure cultivation of tubercle bacilli". A few days before his death, the glass vessel into which he used to spit was broken, & this woman pricked her finger with one of the fragments. Fourteen days afterwards she consulted Dr Tscherning, for the first time. She then showed symptoms of whitlow of the injured finger. Carbolic acid applications relieved her symptoms. Eight days afterwards there was no trace of suppuration, but there was felt,
in the subcutaneous cellular tissue, a little hard lump about the size of a pea. It was painful & surrounded with a zone of oedema. About one month afterwards Dr Tscherning cut out this nodule, & found it composed of granulation tissue. It lay between the skin & the tendon. The wound rapidly healed under a dressing of Iodoform and Perchloride of Mercury.

A little more than a week later, the patient again presented herself with a considerable swelling affecting the same finger, but now encroaching on the palm of her hand, & interfering with the movement of flexion. The disease got worse. About ten weeks after the first operation, through the oedematous skin, a thickening in the sheath of the tendon could be easily felt. At this time two swollen glands in the forearm & two others in the axilla, were noticed. In other respects the woman was in perfect health, & the lungs were sound. About eleven weeks after the incision of the nodule, Prof. Studsgaard cut out the axillary and forearm glands, & removed the finger at the metacarpophalangeal articulation. At the same time through an incision in the palm of the hand, he removed, up to that joint the tendon & its sheath. He also scraped away all subcutaneous granulations. In eleven days the wound healed completely, beneath an antiseptic dressing. Under the microscope the diseased tissues were found studded with typical tubercles, & tubercle bacilli were found. The patient was in good health when seen two months after the operation.
In the France Medicale of July 1887, Dr Cartaz relates that he has collected 17 cases from various authors, besides a case of his own, in which tubercle affected the nose. The tubercle showed itself in one of two forms, ulceration, or small tumours. These two forms sometimes co-existed in the same individual. The seat of the disease was either close to, or just within the entrance of the nostrils. Sometimes it spread down the lips. The ulcerations co-existed, almost always, with grave lung lesions, undoubtedly tubercular in character. It is reasonable to suppose that these were cases of auto-infection by tubercle.

Bennett in the British Medical Journal October 11th, 1884 records the following:—"A healthy young military officer, without hereditary taint, whose wife was in the last stage of consumption, took with her, a sea voyage, lasting four months. On account of bad weather they were confined very much to their cabin, which was small & badly ventilated. On their arrival in London, the wife died, and the husband showed the first signs of consumption.

But it may be said, some of the instances just recorded, are instances of the well known truth, that tubercle, in all its forms, is an inoculable disease. The great point is, that without the tubercle bacillus or its spores, inoculation of tubercle would be impossible. Cases such as these bring forcibly before one's mind the fact, that phthisis, may & does arise from inoculation, in the every day sense of the word.
The disease is due to a germ, and this germ must find a suitable soil for itself, before it can grow & multiply, and although it is much easier & more evident to us that tubercle is caused in a naked wound by inoculation, still it is equally easy to suppose the same inoculation taking place in the lungs, when we find as we so often do the germs floating about in the air of rooms inhabited by consumptives.

On this point it is well to note the observations resulting from Dr Cornet's two years' work in the Berlin Hygienic Institute, & published in pamphlet form by Messrs Richter of Berlin.

Dr Cornet's experiments were conducted with a view to ascertaining whether, in places occupied by consumptives - such as hospital wards & private residences - tubercle bacilli were present in such numbers as to be a cause of material danger to animal life. He sterilized sponges, and with them washed the walls & floors, or whatever part of the room, or its furniture he wished to examine. Then broth was inoculated by these sponges. If the sponges, in washing the walls or furniture, took up tubercle bacilli, those organisms would, of course, grow in the broth, and it being injected into healthy guinea-pigs, these animals, if they showed symptoms of tuberculosis after receiving injections of the broth-culture, gave clear evidence that tubercle bacilli had gained access to their bodies. When these experiments
had been sufficiently multiplied & controlled, the conclusions warranted by the facts were, that tubercle bacilli were often present where consumptives lived, & that the organisms were frequently sufficiently numerous in those places, to be a source of great danger to animal life. The guinea-pigs used for these observations were killed 40 days after their inoculation with the broth, & the condition of their bodies was carefully ascertained by post-mortem examination. Of twenty one hospital wards, where the majority of the patients were consumptives, fifteen of all the wards yielded dust which, in the way mentioned, produced tuberculosis.

In the same way, it was shown that dust from walls in lunatic asylums was often found infected with tubercle, so also were the walls & floors of rooms in private houses where consumptives had lived.

The following are some of Cornet’s experiments-

In a hotel room, where there was living a woman who was tuberculous tubercle bacilli were found on the bedstead & picture frames. In the case of a young man, who had been ill for 9 months from consumption of lungs and larynx, & whose habit it was to spit upon the floor, the tubercle bacilli were found upon the walls beside the sofa. This man’s room & his person, Cornet says, were kept scrupulously clean. Taking that for granted, the case shows, as Cornet pointed out, how, even when cleanliness is prevalent, the cause of tubercular disease may remain in the room which has been occupied by a
consumptive, especially if he be given to the filthy habit of spitting about his room, as this man was.

The examination of this room turned out to be specially interesting, because, three & a half months after it had been completed, the brother of the occupant consulted Cornet, & was found to be suffering from commencing consumption. He had previously made no complaint of illness, & Cornet says he regarded him as quite well when the examination of the room was being carried out.

In the workshop of a tailor, & of a corset maker, where, in the former case, the master, & in the latter a workman, were ill of consumption, tubercle bacilli were found upon the walls of the workrooms. In the case of the corset-maker the examination of the room was undertaken because of the illness of a young man, who had developed consumption while employed in the room where the other consumptive was.

Cornet records the fact, that he did not find tubercle bacilli in dust taken from all the rooms in which lived patients who were suffering from consumption.

He did not once find it in dust taken from the rooms of those consumptives who expectorated only into spittoons, never spitting upon the floor, nor into handkerchiefs.

In these cases, specially careful search was made for the bacilli, but in no instance were they found.
This observation of Cornet's is of the utmost importance, giving us one way by which we can limit the spread of the tubercle bacilli. The chief source of the tubercle bacillus is the sputum of phthisical patients, & next in importance is milk from tuberculous cows, & the flesh of tuberculous animals when used as human food. It is not in the breath of phthisical patients that we find the bacilli, but in the sputum, if it is allowed to dry about houses & in the streets.

It is not my intention to write on all the symptoms, complications & treatment of phthisis, but only to mention such points as seem to me, from their utility & interest, to be deserving of more notice than is generally given to them in the various works on the subject I have had the opportunity of reading.

Cases are frequently seen in which some of the earlier symptoms of phthisis,—such as cough, expectoration, loss of flesh— are present, but physical examination fails to detect any of the ordinary physical signs, & although one may be very suspicious that the disease does exist, still there is a certain call amount of doubt, till a sudden comes to go & see the patient who has had a Haemoptysis, more or less profuse, & always alarming, especially to the friends, for although in itself it is rarely fatal in the earlier stages, it always causes a great deal of alarm & anxiety to the patient & his friends.

The shorter the interval since the haemorrhage the more difficult is it to locate the spot from which
it comes.

A sign which I have noticed within an hour of the commencement of the bleeding, is absence of breath sounds over the part from which the blood comes, with fine crepitations all round, when the patient is told to breathe very quietly. When the patient again breathes naturally sounds can again be heard over the area.

The following is a case in point:— J.K. age 40, sent for me about 9 a.m. on the 10th April 1894. I saw him within an hour of the commencement of the attack, & found that he had expectorated about half a pint of bright red blood. On physical examination of his chest I could detect nothing, but on telling him to breathe quietly, I noticed an absence of breath sounds, with fine crepitations round it, over a small area in the left apex.

He recovered completely from the effects of the haemorrhage, but subsequently developed phthisis from which he died at the beginning of 1897.

The explanation of this sign, I think, is that the alveoli round the vessel which has ruptured becomes filled with fluid blood, which on ordinary breathing is not enough to prevent the entrance of air, but when the patient breathes quietly the air is stopped by the blood, & hence one hears no breath sounds over that area.

On reading over my notes on cases, I find I have record of a condition in phthisis which I have never been able to find described in any book.
I have only seen this in two cases, but both of them were exactly similar, & each terminated fatally in a short time of this condition presenting itself. The first case I saw in 1889, & the other was the case of J.K. previously mentioned.

The following is the record in my note book—

I had been attending, for some months, a young girl of 17 for phthisis from January 1889, she went on fairly well till towards the end of March 1889, when on the evening of the 24th about six o'clock I was suddenly sent for & found her in the following peculiar condition. She was quite conscious & able to speak, and told me that when the attack first began she felt numb down the whole of the left side, & that she had a peculiar prickly sensation down the same side. This lasted for some time, when the whole body became affected in the same way, & she was then quite unable to speak although she understood everything that was said to her.

Slight facial paralysis was also noticed. This feeling lasted for about an hour & then passed off. On the two following days she had similar attacks, but not so severe as the first one.

The question was, what were these attacks due to? I was somewhat at a loss at first to answer this natural question of her parents, as I could find no mention of such a condition in any of the text books, & as I had never previously seen such a condition. At first sight it looked somewhat like some commencing
organic mischief at the base of the brain, but from the rapid recovery she made from the attack, it is much more probable it was a congestive condition of the brain & cord, and in favor of this view is the fact that her "period" was some days overdue & did not come on, although she had been regular for some months previously. She died on April 27th, but I could not get permission to make a post-mortem.

In the treatment of phthisis, there can be no doubt whatever that Creasote treatment is the one on which we can place most reliance. In my experience, there are very few patients both young & old who cannot take Creasote.

The best form is Beechwood Creasote & I invariably give it in Inf. Gent. Co. to be taken in a wineglassful of hot milk immediately after meals.

The tolerance which patients exhibit for the drug given in this way is surprising. I have even got children of a few years old to take it for weeks at a time without any ill effects on the stomach.

For an adult the dose recommended in text books, is from 1 to 3 minims, but to do any good at all this is far too small.

The object of treatment being to get the patient's tissues saturated with the drug, our aim ought to be to get him to take, as speedily as possible, large doses.

I have frequently had patients taking ten minims three times a day, several times I have given 15 minims, and on one occasion I was able to get a
patient to take 20 minims thrice daily for weeks at a time, only having a few days rest from the drug, as soon as I noticed his stomach getting deranged. This was soon, & easily put right, when he would commence his 20 minim doses again.

Creasote when given by the mouth, is excreted by all the excretory organs - the skin, kidneys, bowels, and more especially by the lungs. The latter being the most essential in the treatment of phthisis.

This being the case, we can conclude that the larger the doses given the more will be excreted by the lungs, and therefore the more effect the Creasote will have on the Tubercle Bacilli.

Antiseptic inhalations are often of great service in the treatment of phthisis, many substances have been used, such as Creasote, Carbolic acid etc., but on account of their disagreeable odour not many patients will persevere with them for any length of time.

A very useful plan is to sprinkle a piece of absorbent cotton wool, hung round the patient's neck, with a few drops of a mixture of Eucalyptus & Menthol, but the inhalation I have found most effective is, that by Cinnamon oil.

The effects of this are often very satisfactory, & on account of its pleasant odour & non-irritating properties, patients will use it for any length of time. Its effects are soon noticed, it eases the cough, lessens the expectoration, & materially diminishes if not
entirely stops the night sweats, which are so disagreeable & weakening to the patients.

Patients can even be got to go to sleep with the inhalers on, and so inhale Cinnamon oil which is a powerful antiseptic. This treatment by Cinnamon oil was, I believe, first advocated by Dr J. Histon Thompson in the British Medical Journal for Nov. 7th, 1896.
Pneumonia. Until comparatively recent years Pneumonia & Inflammation of the lung were considered synonymous terms, but at the present time most authorities regard pneumonia as a specific fever, with the inflammation of the lung simply as the local manifestation of a general disease.

Niemeyer in his Practical Medicine (8th edition 1883) makes no mention of Pneumonia being anything but Inflammation of the lung. Although he has evidently had some doubt in his mind that it is always a simple inflammation.

In Vol. 1 page 167 he says - "Its exciting causes are generally unknown. At times pneumonia becomes of very frequent occurrence. While croup, acute articular rheumatism, erysipelas, & other inflammatory disorders prevail at the same time, attacking their victims without any obvious provocatives. This prevalence of acute inflammatory disorders through the operation of unknown atmospheric & telluric agencies is generally spoken of as inflammatory epidemic influence".

Dr William Osler in his "Principles and Practice of Medicine" 1892, page 511, defines Pneumonia as "An infectious disease characterised by inflammation of the lungs & constitutional disturbance of varying intensity."

Again on page 512 he says, "Change of opinion has of late taken place as to the nature of pneumonia, which is now almost universally regarded as a specific
infectious disease, depending upon a micro-organism. Among general circumstances favoring this view is the occurrence of pneumonia in epidemic form, a fact recognised by Laennec & by Grisola.

Many house epidemics have been described during the past twenty years. On several occasions I have known two, three & even four persons admitted to hospital from the same house.

In 1887, I saw with Graham of Toronto, a local outbreak in which three members of a family were consecutively attacked with the most malignant pneumonia.

There are instances on record in which as many as ten residents in one house have been attacked.

Of late years many epidemics in towns have been reported. Still more striking are the epidemics which have been described in prisons & garrisons, of which one of the most remarkable is that reported by W.B. Rodman of Frankfort, Kentucky.

In one year there occurred in a prison population of 735, 118 cases with 25 deaths. The prison was much overcrowded at the time.

Sir R. Douglas Powell in his "Diseases of the Lungs and Pleurae" 4th edition 1893, page 214, says - "It is true in a sense that Pneumonia & Inflammation of the lungs are synonymous terms, but the latter expression by no means covers the whole pathology of the disease; indeed, a careful examination of the aetiology & clinical features of pneumonia, would suggest
its being placed in our nosology amongst such diseases as acute rheumatism, erysipelas, quinsy, influenza, the inflammatory phenomena in each of these diseases being but the local expressions of a general state, & only exceptionally proceeding beyond hyperaemia & simple exudation."

We thus see that it is pretty well agreed that pneumonia is not simply a local affection, but a "fever". And since the discoveries by Friedlander in 1882 of a bacillus which he supposed was the cause of pneumonia, the infectious nature of the disease has taken a firm hold on many observers' minds.

Sir Douglas Powell on page 217 says "There is also evidence to show that under certain circumstances not completely known, pneumonia may be communicated by one person to another ....... The difficulty is of course the usual one, of separating several persons being attacked in consequence of exposure to a common cause, from those in which the first attacked has transmitted the disease to others".

Several striking instances of the apparent origin of pneumonia by contagion are quoted by Dr Sturge and Coupland from various authors (Pneumonia 2nd edition, 1890, page 329, et seq.) but on analysing the evidence they come to the conclusion that "there is less clinical evidence of the occurrence of pneumonia by contagion than there is of its origin from exposure, from sewer gas, from want of food, even from mental or
bodily depression, & if it be granted that any of these agents may possibly provoke pneumonia, it may be doubted whether it is further necessary, in order to satisfy the facts, to make any large appeal to this origin by contagion."

Again Sir Douglas Powell in a paper read before the British Medical Association in London & published in the Lancet August 10th, 1895, page 355, after expressing much regret that he could not record any practical abatement in the mortality of the disease, emphasised the fact that probably 20% of the cases were attributable to surface chills, contracted through hygienic imprudence, especially amongst those past middle life, & this imprudence was likely to be encouraged by the fatalistic & inadequate view that the reception of a microbe was alone sufficient to cause the disease.

After having reviewed other conditions besides chills, such as depressed vitality, privation, the uraemic state, rheumatic conditions, diabetes etc, Sir Douglas passed on to the question: How far was the reception & culture of a specific organism essential? Friedlander's, Fraenkel's and Klein's microbes were contending for the honor of disseminating pneumonia, the pneumo-coccus of Fraenkel being most in favor.

The speaker's view was, that undoubtedly pneumonia was a specific germinal, & therefore a microbic disease, but the pneumo-coccus could not as yet be altogether accepted as the germ of pneumonia.
On page 220 "Diseases of the lungs" he says:— Some time must even yet elapse before the exact position of micro-organisms with regard to disease can be finally determined; & before any particular micro-organism can be unreservedly accepted as the spore of pneumonia i.e. as the only means by which that disease can be originated or propagated — there are many difficulties to be surmounted.

The climatic conditions in which the disease most occurs, its common origin in exposure to surface chill, the rareness with which infective propagation is observed, & the not yet demonstrated virulence of the coccus, under cultivation, are circumstances requiring to be explained by the exclusive germ theorists of pneumonia. It is a point worthy of note that the diseases already enumerated as those with which pneumonia can be best compared, are equally remarkable for their apparently double mode of origin, — viz. from exposure to chill, & from exposure to poison influences. One might be tempted to say that each of the two diseases, ordinary & pythogenic pneumonia, or idiopathic & wound erysipelas, a simple & virulent catarrh, is quite as distinct from the other as typhus is from enteric fever.

To take a simple instance, a slight dampness of feet will, with almost unerring certainty, give to many persons a severe catarrh, with the usual symptoms of malaise, chilliness, coryza etc. The catarrh thus acquired is virulent, & will run through the household.
The composite catarrhal products expelled from the air passages of the first subject who acquired the catarrh may demonstrably contain micro-organisms regarded as specifically infectious, to account for their contagiousness, yet the onset of the disease can scarcely be attributable to the reception of such organisms but must rather be regarded as some form of vaso-motor disturbance bringing about changes, possibly favorable to the development of the ever present germs. In considering these difficult, but interesting and important problems, one must not confound what may be the carriers of the disease with the disease itself, nor look upon what may be only epiphytes as genuine disease spores."

Dr Arthur Whitelegge in "A system of Medicine" edited by Dr Clifford Allbutt, Vol. 1 page 655 says:- "apart from its occurrence as a sequela of certain zymotic diseases, croupous pneumonia is met with not only in sporadic cases, but also in groups, and occasionally in more or less extensive epidemics, with clear evidence of infection from person to person. The proof of infection is strengthened by the discovery that the tissues and sputa of pneumonic patients contain specific micro-organisms which can be cultivated, and have the power of reproducing the disease when inoculated into lower animals; moreover Dr Klein has found that animals so inoculated become centres of infection to others kept near them. Pneumonia of this kind is to be regarded not as an affection of the lung simply,
but rather as a constitutional infection - a "pneumonic fever" - of which pulmonary & pleural lesions are only the local & not necessarily constant manifestation."

On page 658 he says - "At least three different microbes have been regarded, on apparently conclusive evidence, as pathogenic in infections pneumonia; an oval capsulated micrococcus, described by Friedlander, a capsulated diplococcus, by Frankel & Weichselbaum; and a bacillus found by Klein in the Middlesbrough & Scotter epidemics. Each of these can be cultivated & inoculated on lower animals."

Many other published opinions could be quoted showing, or tending to show, that there is an infectious form of pneumonia, but that every case of the disease must be regarded as infectious is at the present time very uncertain.

In the Medical Annual for 1898 (published by John Wright & Coy, Bristol) page 436 Dr F. de Haviland Hall writing on pneumonia says: - "The evidence of the microbic origin of pneumonia is now so strong that it is time that the whole question of the symptoms & treatment of this disease should be re-considered from the standpoint of our present knowledge."

As to the age at which pneumonia is most prevalent, the majority of writers state that it is most common between the ages of 20 & 40, & that it is a rare disease before five years of age. My own experience is contrary to this, from the numbers of
cases I have seen during the last ten years, in young children, I am forced to the conclusion that it is amongst the commonest acute diseases of childhood.

Sir Douglas Powell in "Diseases of the Lungs" page 214 says:— "No age is exempt from pneumonia, but the disease is more prevalent at that period, between 20 and 40 years, when persons are more exposed in the active struggle of life."

On the other hand Dr Francis Minot in Keating's Cyclopaedia of the Diseases of Children, Vol. 2 part 2 p. 587 says:— "It was formerly believed that lobar pneumonia was rarely met with among young children; but more extended observation has shown that it is, on the contrary, one of the most common of the severe diseases of childhood."

The symptoms & physical signs of pneumonia are so well known that it is unnecessary for me to dilate on them here, but there is a physical sign, which I should like to draw attention to, & which I ventured to describe in the Lancet in 1896 as a new physical sign for the early diagnosis of Croupous Pneumonia. The following is a copy of the short notice I then sent to the Lancet:—

"Croupous Pneumonia is I believe, one of the commonest of the acute chest diseases of children which a practitioner is called on to treat. This statement is based upon a careful examination of many cases commonly called Bronchitis & Broncho-pneumonia which accurate records of temperature & after progress
have proved to be Croupous Pneumonia.

The various authors I have consulted are agreed that the earliest physical signs of Pneumonia in both adults & children are impaired resonance on percussion or fine crepitation on auscultation and that in children especially there is no definite physical sign till the disease is well advanced.

The early diagnosis is thus as difficult as it is important.

Dr Francis Minot in Keating's Cyclopaedia of Diseases of Children (Vol. 2 part 2 page 591) says:--

"In not a few cases, moreover, no satisfactory results are obtained by auscultation & percussion until a comparatively late period, owing to the limited extent of the affected region, and its position in the centre of a lobe, surrounded by healthy lung tissue."

In several cases recently seen where the general symptoms of pneumonia were present but none of the ordinary physical signs, I have discovered what I am venturing to describe as a new physical sign.

On careful auscultation of the chest I have heard jerky expiration over a limited area & on noting the position of this area have found developed in it subsequently the usual signs of pneumonia. This jerky expiration is the first physical sign developed, & can be heard soon, if not immediately, after the rigor before dulness or crepitations appear.

It is much more distinct in children, but I
have also heard it in adults.

The following cases sufficiently illustrate what I wish to point out, though I have notes of several others:

Case 1. Nelly S. age 4 years.
First seen April 29th, 1893.

History (given by Mother) seized with a shivering fit about 12.30 on the 28th (previously quite well) Hands cold, drowsy & thirsty. Pain in limbs in afternoon. Breathing short. Became delirious about 9 p.m.

First seen by me about 4 p.m. on the 29th. Breathing very rapid, slight cough, very quick pulse.
Temperature 104 deg.

Physical examination. Except that expiration all over the right base was markedly jerky nothing abnormal was found in the chest.

30th. Temperature 102. Physical signs the same.

May 1st. Slight dulness on percussion & fine crepitation over right base.

The pneumonia ran a normal course, the patient made a good recovery.


Complaint, pain in right side & shortness of breath.

History - On April 1st got very heated playing football & during the following night had a rigor.

First seen by me on April 3rd.
Respiration rapid, pulse very quick.
Temperature 103.4 deg.
Except jerky expiration at right base posteriorly nothing abnormal on examination of the chest.

Diagnosis. Pneumonia at right base.

This was subsequently verified by ordinary physical signs.

The disease ran a typical course, although convalescence was somewhat prolonged.

Dr Douglas Powell speaking of Pneumonia in his book on diseases of the Lungs page 226 says:

"The breath sound at one base has, however, at a very early period a peculiar rough, harsh quality, very like that of exaggerated breathing & slight crepitation may be audible."

The explanation of this jerky expiration I am not prepared to offer, but suggest that it may possibly be due, either to the primary congestion interfering with the elasticity of the lung, thus making the expiration puffy or jerky, or to a propagation of the heart beat through a more readily conducting medium than the healthy lung.

I am more inclined to think that the former explanation is the correct one, because it is difficult to see how the heart beat could so much affect the air leaving the right lung, & I have been unable to satisfy myself that the rhythm of the jerks correspond with the cardiac impulse.

Further observation will show how much value
is to be attached to this sign, but I note it with the hope that it may prove as useful to others as it has to me."

Since publishing the above note in the Lancet in 1896, fuller observation & more extended experience, have fully convinced me of the correctness of the observation.

I have notes of many cases both in children & adults, where I have based the diagnosis of pneumonia on this physical sign, before the ordinary signs have had time to develop.

To those in general practice this physical sign is more useful than it is to hospital Physicians & Consultants, because as a rule neither of the latter see a case of pneumonia till the disease is fully developed, unless it be that the pneumonia is about to start as a complication of some other disease, such as Typhoid fever or Influenza.

From this physical sign, too, I have been enabled to say that an existing pneumonia was about to spread, because the same jerky expiration can frequently be heard round a patch of fully developed pneumonia.

A form of pneumonia which up to the present has not received much attention, is that produced sometimes by the administration of Ether.

A discussion on this subject was recently introduced by Dr Drummond at a meeting of the Northumberland & Durham Medical Society. Notes of some
eight cases, and two post-mortems were referred to. I, however, only saw one of the cases, & it terminated fatally about the fifth day.

The cases were chiefly in operations within the abdomen, & as a rule the patients were under Ether for a lengthened period.

Symptoms. The temperature rises the day following the operation, & as a rule is elevated for 6 or 8 days, though in some cases for as much as a fortnight or three weeks.

The Physical Signs are those of Bronchitis with more or less marked dulness, generally to one base.

Tubular breathing seldom pronounced, in places muco-crepitant rales can be heard, & in others there is absence of respiration, or again a distant tubular expiration could be detected. Vocal fremitus usually impaired.

The signs varied from day to day, & in this way differed from ordinary Croupous Pneumonia, depending apparently upon whether the air tubes were patent or not.

Copious muco-purulent expectoration was a marked feature, with little or no tendency to rusty discoloration.

Two cases died, one on the eighth day & one on the fifth.

Post-mortem. In both marked consolidation of one base, of a patchy character suggestive of Catarrhal pneumonia, but with much more haemorrhagic appearance
than is usual in that condition.

Microscopical examination showed the tubes blocked with large quantities of thick mucus containing proliferating columnar epithelial cells, the alveoli filled with large catarrhal cells & many contained red blood corpuscles in quantity.

The view adopted in explanation of this condition was that an abdominal operation prevents the patient from coughing & thus the lungs become blocked with the abundant secretion of mucus.

The next subject I will mention is Empyema, I have notes of some thirty cases, some of which were my brother's, some my own, but I have had the privilege of following all the cases throughout.

An examination of the following table of thirty cases of empyema which have come under my notice, shows the greater prevalence of the disease in childhood. Four cases only occurred in adults, the remaining twenty-six in children, their ages varying from seven months to nine years.
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age</th>
<th>Cause</th>
<th>Operation</th>
<th>Result</th>
<th>Duration of Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A.C.</td>
<td>6½</td>
<td>Pneumonia</td>
<td>Incision</td>
<td>Recovery</td>
<td>49 days</td>
</tr>
<tr>
<td>2</td>
<td>J.W.</td>
<td>8</td>
<td>do</td>
<td>Repeated</td>
<td>aspiration</td>
<td>do</td>
</tr>
<tr>
<td>3</td>
<td>C.S.</td>
<td>5</td>
<td>do</td>
<td>Tubercular</td>
<td>Injection</td>
<td>do</td>
</tr>
<tr>
<td>4</td>
<td>G.B.</td>
<td>4½</td>
<td>{Tubercular}</td>
<td>Incision</td>
<td>do</td>
<td>do</td>
</tr>
<tr>
<td>5</td>
<td>J.M.</td>
<td>2</td>
<td>Pneumonia</td>
<td>do</td>
<td>do</td>
<td>39</td>
</tr>
<tr>
<td>6</td>
<td>C.D.</td>
<td>6</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>22</td>
</tr>
<tr>
<td>7</td>
<td>C.O.</td>
<td>8½</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>61</td>
</tr>
<tr>
<td>8</td>
<td>M.L.</td>
<td>23</td>
<td>Pleurisy</td>
<td>do</td>
<td>do</td>
<td>34</td>
</tr>
<tr>
<td>9</td>
<td>C.W.</td>
<td>4</td>
<td>Pneumonia</td>
<td>do</td>
<td>do</td>
<td>35</td>
</tr>
<tr>
<td>10</td>
<td>E.G.</td>
<td>4</td>
<td>Pleurisy</td>
<td>do</td>
<td>do</td>
<td>46</td>
</tr>
<tr>
<td>11</td>
<td>J.M.</td>
<td>30</td>
<td>Pneumonia</td>
<td>do</td>
<td>do</td>
<td>35</td>
</tr>
<tr>
<td>12</td>
<td>T.B.</td>
<td>9</td>
<td>Pleurisy</td>
<td>Incision</td>
<td>&amp; afterwards</td>
<td>Thoracoplasty</td>
</tr>
<tr>
<td>13</td>
<td>W.E.</td>
<td>3½</td>
<td>Pneumonia</td>
<td>Incision</td>
<td>do</td>
<td>21</td>
</tr>
<tr>
<td>14</td>
<td>J.S.</td>
<td>22</td>
<td>Phthisis</td>
<td>do</td>
<td>do</td>
<td>46</td>
</tr>
<tr>
<td>15</td>
<td>C.H.</td>
<td>4</td>
<td>Pneumonia</td>
<td>Thoraco-</td>
<td>plastic</td>
<td>do</td>
</tr>
<tr>
<td>16</td>
<td>J.S.</td>
<td>2½</td>
<td>do</td>
<td>Incision</td>
<td>do</td>
<td>23</td>
</tr>
<tr>
<td>17</td>
<td>W.P.</td>
<td>9</td>
<td>do</td>
<td>Refused</td>
<td>operation</td>
<td>25</td>
</tr>
<tr>
<td>18</td>
<td>A.L.</td>
<td>8</td>
<td>do</td>
<td>Refused</td>
<td>operation</td>
<td>1 year</td>
</tr>
<tr>
<td>19</td>
<td>W.M.</td>
<td>6</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>21 days</td>
</tr>
<tr>
<td>20</td>
<td>A.S.</td>
<td>3</td>
<td>do</td>
<td>do</td>
<td>do pus</td>
<td>(Long convalescence)</td>
</tr>
<tr>
<td>21</td>
<td>M.O.</td>
<td>2½</td>
<td>do</td>
<td>Incision</td>
<td>do</td>
<td>36 days</td>
</tr>
<tr>
<td>22</td>
<td>C.P.</td>
<td>7½</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>30</td>
</tr>
<tr>
<td>23</td>
<td>J.W.</td>
<td>4</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>21</td>
</tr>
<tr>
<td>24</td>
<td>W.W.</td>
<td>6</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>30</td>
</tr>
<tr>
<td>25</td>
<td>G.S.</td>
<td>6</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>25</td>
</tr>
<tr>
<td>26</td>
<td>A.H.</td>
<td>2½</td>
<td>do</td>
<td>do</td>
<td>Died</td>
<td>3 days</td>
</tr>
<tr>
<td>27</td>
<td>G.L.</td>
<td>4½</td>
<td>do</td>
<td>do</td>
<td>Recovery</td>
<td>29</td>
</tr>
<tr>
<td>28</td>
<td>J.C.</td>
<td>4½</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>31</td>
</tr>
<tr>
<td>29</td>
<td>M.L.</td>
<td>6</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>26</td>
</tr>
<tr>
<td>30</td>
<td>G.C.</td>
<td>10m.</td>
<td>do</td>
<td>do</td>
<td>do</td>
<td>27</td>
</tr>
</tbody>
</table>
CAUSES. (1) Pleuro-pneumonia is by far the most common cause. In 76.6 per cent of my cases the history left no doubt of the truth of this important observation which I believe was first pointed out by Dr. Drummond of Newcastle.

The rich network of lymphatics immediately under the pulmonary pleura, the almost invariable association of pleurisy with pneumonia, the constant presence of the pneumococcus in pneumonic lungs, & the recognition of the fact that the pneumococcus is capable of causing suppuration, prepare one to receive an explanation supported by experience & the teaching of anatomy & pathology.

(2) Pleuritis. The following quotation from a recent writer would show that the old belief that pleurisy is the most common cause of empyema is still held by some authorities:— "In opening up the question of the treatment of empyema, our first thought is that it is often but making the best of a bad business - stepping in to patch up a 'case' that would have been so much better prevented; for we feel that, with more careful observation on the part of those by whom the case brought to hospital with an empyema have been first attended, many a simple pleuritic effusion could have been nipped in the bud, & even if severe, cured before suppuration, by the timely use of the aspirator. It seems certain that in many, if not most such cases, it is neglect of early use of the aspirator
rather than its misuse, which is responsible for a serous effusion becoming purulent."

Only ten per cent of these cases appear to be probably owing to this cause. The number of ordinary cases of pleuritic effusion seen which never suppurate is remarkable, if this be a frequent result. If an ordinary serous effusion becomes purulent, a dirty needle or aspirator is the cause in the majority of cases; some would say always.

In the following case all precautions were taken to render the patient's side & the needle aseptic before aspiration:-


Twenty ounces of clear serum removed by aspirator on March 20th. On March 28th again aspirated, and obtained seventeen ounces of floculent serum. On May 7th again aspirated & fourteen ounces of pus withdrawn. May 18th chest incised, & one pint of pus escaped.

(3) Tubercular disease of the lungs, with signs of cavity, occasioned empyema in one case (i.e. 3.3 per cent of my cases). The purulent pleurisy followed a pneumothorax as is the usual course in tubercular cases.

(4) Tubercular disease of the lower lobe of one lung, without signs of cavity, was followed by empyema in one patient (i.e. 3.3 per cent of my cases).
G.B., age 43, has had a cavity in his left apex for some years. Seven weeks ago he had an attack of acute pneumonia at his left base. His present complaints are of a cough, with profuse expectoration, shortness of breath & loss of flesh.

From being a stout man he has been reduced to a very thin one. The cough he says, comes on at intervals, & at these times a large quantity of yellow expectoration is expelled by it. Relief is obtained for a time; then the cough & expectoration are repeated.

Physical examination shows an apical cavity, apparently about four inches in length. Posteriorly & laterally the left base up to the lower angle of the scapula is dull on percussion, the vocal fremitus is unaltered, & crepitation & increased vocal resonance are heard over the dull area.

An aspirator needle introduced on June 23rd between the sixth & seventh ribs, posteriorly, proved the presence of pus. The chest was incised at this spot, & nine ounces of pus escaped. The immediate result was most satisfactory, all his symptoms being relieved. The tube was removed in three months, & the sinus closed, but crepitation continued still at the left base.

(5) Abscess of any neighbouring organ opening into the pleura, e.g., abscess of the liver, of which a case came under my notice recently.
Diagnosis. In the ordinary case, a child is being attended for pneumonia, & presents the usual symptoms & physical signs of that disease. The acute illness runs its course, & convalescence seems assured. Then it is noticed that the child does not recover as was expected. It becomes listless, has a poor or fitful appetite, loses flesh, & sweats at night, especially about the head.

Vomiting is not an infrequent symptom, & has appeared to me more common when the right side is affected. The child has a short dry troublesome cough and a febrile temperature. A peculiar ashiness of complexion may usually be observed. With symptoms of this kind, a history such as has been described, & physical signs of fluid or a consolidated lung, the exploring needle should find pus.

In several cases I have seen the diagnosis has been difficult from the misleading character of the history & symptoms, & has only been arrived at by a complete examination & a confidence in physical signs. The difficulty arises from the curious simulation of an abdominal ailment by certain of these cases. The illness commences acutely with vomiting, pain in, distension, tenderness & hardness of the abdomen. The cough is not an aggressive feature, & it is easy to regard the case as one of peritonitis, which it closely resembles. In some cases of simple pleurisy, the same abdominal tenderness, resistance & pain are seen, & the explanation is obvious if the distribution of the
intercostal nerves is remembered; the 6th terminating in the epigastric region, the 10th in the umbilical region. The pleuritic pain is referred to the ends of the irritated nerves; the skin supplied by them is tender, & the abdominal muscles innervated by them are contracted. When, too, it is remembered that acute idiopathic peritonitis is one of the rarest of ailments such a mistake is not likely to happen. The physical signs are those of fluid in the pleura; the side is dull on percussion, vocal fremitus is absent or weak, the breath sounds are commonly tubular in character. (It is quite rare to have absence of breath sounds in these cases). The heart is altered in position. In some cases the side is hyper-resonant, or the percussion note little altered, especially when the effusion is on the left side. In such a case the tympanitic area of resonance, usually occupied by the stomach laterally, will be dull, and the dulness extends over as far as the epigastric region. These signs are due to the diaphragm being pushed down by the fluid in the pleural cavity, & displacing downwards the abdominal contents. On the right side the dulness of fluid in the chest may be mistaken for the normal liver dulness if the lower edge of the displaced liver is not recognised.

In all doubtful cases the exploring needle should be employed. It may be used with perfect safety if the skin round & over the proposed puncture
be previously purified & the needle rendered aseptic. In some cases several punctures are necessary before the result is certain, and even though the needle goes into pus, this may fail to be withdrawn. In every case where nothing comes through the needle, it should be suspected of being blocked.

These difficulties arise in cases of localised empyema, and where pus is inspissated or flakey.

**Prognosis.**

1 Case 20 proves & case 9 suggests, that pus in the pleural cavity is capable of absorption. In case 20 the presence of pus was diagnosed by physical signs, & verified by the needle; but the parents of the child refused operation. Recovery followed without expectoration or external discharge of the pus. In case 9 operative interference was refused for six weeks after the diagnosis of empyema was made, & on opening the chest only four ounces of very thick sticky pus came away. It looked as if the pus were in process of absorption.

2 Spontaneous evacuation through a bronchus. In case 19 recovery followed this event without an operation. In five cases pus in such quantity as to make it certain that an opening had formed in the lung, was expectorated prior to operation. After operation these cases pursued a completely aseptic course, and complete recovery, without damage to the lung, followed in all. Most of these cases came to me with a diagnosis of consumption.
3 In some cases the pus is discharged into the oesophagus, stomach, & other adjacent organs. This can only take place where the accumulation is great, & the course of the disease is prolonged & unaided. I have no experience bearing on it.

4 Spontaneous evacuation through the chest wall is of all forms of spontaneous evacuation the most unsatisfactory. It usually occurs through the fifth interspace, & oedema of the chest wall is a common signal. In a few cases recovery follows, but the majority die of exhaustion or waxy disease.

5 Surgical treatment offers the best results, immediate and remote. My cases prove that uncomplicated empyema treated with proper surgical care, & in children, is not a serious disease, for of twenty-six cases all but one recovered. The death I have to record is that of case 26, of which the following is a note:

A.H., 2½ years, suffering for three weeks from broncho-pneumonia. The case was diagnosed empyema & I saw the child on November 5th, 1891. The pus reached as high as the scapula on the left side. All over the right side were heard bronchitic rales, & the child was suffering from great dyspnoea. On November 6th the chest was opened, giving exit to about a pint of pus. The breathing was relieved but the bronchitis increased and the patient died on the third day after operation from this complication.

That antiseptic precautions are all important in such cases was forcibly conveyed to me by experience
S.B., age 9. On May 18th, 1888 his chest was incised & about a pint of pus removed. Owing to infrequent dressing the case became septic, & in spite of a variety of measures, the patient grew worse. Eight months after, when the patient had become very reduced, portions of his fifth, sixth & seventh ribs were excised. Immediate improvement followed, & two months afterwards he was healed and well.

If the saving of life is satisfactory, the perfect recovery of these cases is more satisfactory still. I have measured & examined the chests of most of them months after operation, and, except for the scar, it is impossible to say which side has been affected.

Empyema has, never to my knowledge, been followed by tubercular lung disease. Recovery has been complete & permanent in all uncomplicated cases. In case 17 the right lung was compressed for six months before an operation was allowed by the parents of the child, & a marked lateral curvature was present. Two months after the closure of the wound the right half of the chest measured the same in circumference as the left, & all trace of spinal curvature had disappeared. Such a result can surely only be predicted if the irritation of putrefaction or irrigation be avoided. Either causes chronic thickening, induration, & subsequent contraction of the inflamed pleura, with deformity of the chest & spine. The average duration of treatment from the date of incision to the completion of
healing was thirtythree days. The shortest period was twentyone days in case 25; the longest sixtyone days in case 7.

Judging from my limited experience in adult patients, four in number, I think in uncomplicated cases as good a result should be obtained in them as in children, though more empyemata in the former are due to serious and incurable disease.

Treatment. There is no medicinal treatment which will cure empyema. It requires surgical measures as an ordinary abscess does. Occasional recoveries follow aspiration, & in my earlier cases I tried it; but very seldom with a good result. In a large percentage the pus re-accumulates rapidly, & I soon concluded against further trial of the aspirator. Aspiration is, however, of service in one class of case in patients who are suffering from dyspnoea, due to a large fluid collection, or in whom some complication renders operative interference inadvisable. In such cases it is to be resorted to only to tide the patient over a trying time, & as soon as the urgent symptoms disappear the chest should be opened & drained.

In the following case this method was employed with a satisfactory result:

C.C., age 10 months. First seen November 14th, 1892. A history of "bronchitis" for a fortnight. Left chest full of fluid which the needle proved to be pus. There was capillary bronchitis on the right side, with great dyspnoea. The aspirator withdrew one
pint of pus from the left chest with marked improvement to the respiration. The child's condition improved & on November 20th the left chest was opened & drained of one pint of pus. Convalescence was rapid.

The operation. The patient, surgeon, instruments & assistants are prepared as for an antiseptic operation with the most scrupulous care. An anaesthetic in the case of children is always advisable, though in the case of adults with a very full chest the operation can be done, & with much greater safety, without anaesthesia, or with the local use of cocaine. Chloroform is the best anaesthetic for the purpose, but must be administered with special care, & during the whole operation the patient should lie on the back. I have seen the most serious effects produced by turning the patient over to the sound side during the operation. This may be avoided by drawing the patient to the edge of the table, so that the side to be incised projects over the edge.

The situation of the incision - in all cases it is advisable before incising the chest wall to explore with a needle to ascertain the position of the pus. The point, I select, when possible, is in the sixth interspace, just in front of the posterior axillary line. A vertical incision is first made through the skin, then a horizontal one through the muscles along the upper border of the seventh rib into the pleural cavity. The handle of the knife is then introduced into the wound, or a pair of sinus forceps guided by it into the
pleura, by opening & withdrawing which the wound is dilated.

Before all the pus has escaped an India rubber drainage tube, quarter of an inch in diameter & two inches long, is inserted. This tube is perforated for the inner one third of its length only, for if there are any openings in the outer part of the tube it frequently becomes blocked by granulations growing into them. A safety pin passed through the outer end prevents the drainage tube from slipping in - an accident apparently of not infrequent occurrence. As soon as the discharge has become small in quantity half the tube may be cut off, & the remainder retained for a few days. For the first day after removing the tube I am always anxious, & if the cough returns, with some rise of temperature or vomiting, I explore the wound with a probe, lest the outer opening should have closed too quickly.

In cases of long standing empyema the diaphragm may be wounded in re-introducing the tube if care is not taken. The collapsed lung allows the diaphragm to ascend into the chest, & it lies closely opposed to the chest wall. Being softened, too, it is easily torn, & the abdominal cavity may be opened through it.

The wound is dressed with antiseptic dressing - corrosive gauze & corrosive wood-wool wadding being those I always employ. The dressings are kept in place with a domette bandage fixed by braces over the shoulders, & prevented from rising to let air pass.
underneath by three or four turns of elastic webbing bandage. It is in all cases advisable to dress the wound two or three times during the first twentyfour hours, in which the discharge is most profuse.

The dressings are after this changed daily till the second week, when every second day is usually sufficient. The guide to frequency of dressing is the amount of discharge. The bandage should never be allowed to be stained longer than can be avoided. During the third week, in a large proportion of cases, the tube can be removed & the wound rapidly closes.

The discharge should be serous after the third or fourth day. If it is purulent, this indicates either that the tube is not acting efficiently, or that the wound has become septic.

Irrigation is unnecessary & hurtful. Even the most fetid cases become sweet after a few days good drainage & careful dressing. As an aid to assisting the lung to expand, immediately the patient is out of bed he should (if old enough) be encouraged in the use of gymnastic appliances, & making respiratory efforts such as are required in running upstairs, or in playing wind instruments.

In two cases Eslander's operation was performed & the notes sufficiently indicate the class of cases in which this operation is useful. In both cases the wound had been allowed to become septic. In one, excessive purulent discharge for eight months, in the
other for two & a half months, had reduced the patients to a weak & emaciated condition. The side was contracted in each, with ribs close together, & the lung appeared bound down. A long vertical incision was made near the posterior axillary line, & the soft parts dissected off the ribs. An incision was then made through the periosteum covering the fifth, sixth and seventh ribs for about two inches, the periosteum detached, & this amount of the three ribs excised. A free opening was then made through the pleura, & a counter opening posteriorly. A drainage tube was then passed from one opening to the other, & in shortening the tube it was left in the posterior opening. Both cases did remarkably well, the more chronic being entirely healed in two months, the less chronic in one month. In both slight deformity remains.

My conclusions are:-

That pleuro-pneumonia is the most frequent cause of empyema.

That empyema rarely follows pleurisy with effusion.

That in adults empyema is more likely than in children, to be secondary to serious lung disease.

That the diagnosis of empyema is not always easy, for it may be ushered in by symptoms pointing more to the abdominal than the thoracic cavity, & the physical signs resemble closely those of consolidation.

That the exploring needle should be used in every doubtful case, and, if needful, in more than one place.
That there is no danger from using the needle with proper care.

That the immediate prognosis of an uncomplicated empyema treated by incision & drainage is good. It is worst when allowed to burst through the chest wall.

That the remote prognosis is excellent. No deformity follows recovery, & no sequelae are to be anticipated.

That the best prognosis can only be given in cases where an aseptic condition of the pleural cavity can be assured by antiseptic operation, & antiseptic after-treatment.

That the anaesthetic should be carefully administered, & the patient not turned over during its administration.

That in adults it may be safer to do the operation without general anaesthesia.

That aspiration is disappointing as a curative agent, but useful in some cases temporarily.

That incision & drainage with antiseptic precautions may be trusted to cure all cases of curable empyema.

That irrigation is unnecessary & dangerous.

That rib re-section is needful in some neglected cases, but in the generality is only meddlesome surgery.

In connection with the above cases of empyema it may be instructive to record one case of Pyo-pericarditis which has come under my notice.

A.B., age 24. First seen March 5th, 1888, complaining of severe pain in left side of twelve hours duration.
History. Patient was quite well until the evening of the 4th, when he was seized with a severe shivering which was soon followed by a feeling of feverishness accompanied by pain in the left side. Patient had got wet through the day previously.

On examination the temperature was 104.5, pulse 116, & respirations 24 short & painful. He has a cough but tries to hold it back owing to the pain in his side. There is on examination mild pleuritic friction over the whole of the lower lobe of the left lung, more especially in the axillary region & fine crepitation of a commencing Pneumonia.

The heart sounds are normal.

The Pneumonia developed during the next 24 hours & the lung became consolidated, with dulness, tubular breathing, & increased vocal resonance.

For the next five days patient progressed favorably, the temperature varying between 101-105, but on the seventh day it was noticed that the patient was slightly cyanosed, breathing more embarrassed & that the dulness extended over the praecordial region and was specially well marked in the epigastrium.

On March 13th (8th day) the patient’s temperature was 102.6 the cyanosis more marked, pulse 126 small and feeble, & he had passed a bad night owing to the dyspnoea.

Dulness over the praecordia & in the epigastrium was noticeable, the heart sounds muffled & weak.

An exploring needle was thrust into the pleural cavity in the mid-axillary line, but with a
negative result.

As I felt sure that the symptoms & signs pointed to fluid in the pericardium I passed the needle of an exploring syringe into the fourth interspace about one inch from the edge of the sternum, this must have passed into the right auricle for a syringleful of dark venous blood was drawn off.

The needle was then inserted through the sixth interspace, about the same distance from the sternum, & a syringeful of pus was withdrawn.

About three ounces of sero-sanguineous pus was withdrawn with the exploring syringe as a temporary expedient, seeing that the patient lived some distance from my house, & arrangements could not be made immediately to treat the case antiseptically.

Patient was very much relieved & immediately fell asleep until early morning, when the dyspnoea again became troublesome.

Operation - March 13th. Owing to the condition of the patient no anaesthetic could be given. The skin having been previously purified, an incision two inches long, with its centre over the sixth interspace close to the left edge of the sternum was made through the skin. The intercostal muscles were incised transversely & a pair of dressing forceps thrust into the pericardium gave rise to a discharge of about two pints of sero-purulent fluid. A large rubber drainage tube was inserted & antiseptic dressing applied.
Patient bore the operation well & his pulse & breathing improved after it.

4 p.m. Has slept for short intervals. Pulse 160 Temperature 100.4. Wound dressed; discharge very profuse.

11 p.m. Patient has great discomfort from palpitation. Pulse is almost uncountable. Ordered Ammon. Carb.grs 20 Tr.Digitalis minims 10, every four hours, & a suppository containing ½gr. Morphia.

Dressed; a great deal of discharge.

14th. Had a fairly good night. Temperature 100. Pulse still uncountable. Increased the dose of medicine to double quantity, & added Tr.Opii minims 20.

3 p.m. Feeling better. Palpitation not so severe, though pulse is still very rapid. Dressed. Ordered to lie on face. Taking nourishment well.


Unfortunately the person who was nursing him instead of giving him one tablespoonful as ordered on the label gave him the whole contents of the bottle, a dose of 200 minims of Liq.Morph. At 2 a.m. I was sent for & found the patient was suffering from Morphia poisoning. In spite of all that could be done he became more & more unconscious and died.