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A TREATISE ON PULMONARY TUBERCULOSIS

In its

PATHOLOGICAL, CLINICAL, and SOCIOLOGICAL ASPECTS

By

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P R E F A C E

A treatise on phthisis needs no apology, unless it be for the faulty execution of an immense subject.

In the following pages, I have attempted the presentation of pulmonary tuberculosis, in its pathological, clinical, and sociological aspects. Such a work is naturally largely influenced by the teaching of undergraduate days, particularly by that of Professor Wyllie, Professor Hamilton, Dr Philip and Dr G. A. Gibson, whose teaching I have followed and in places reproduced. Such a treatise must also be reflective of the literature perused, and as the author has been mostly in contact with French writings, it has the interest of showing the opinions of the modern French School. There is also given the results of two pieces of clinical research; on the action of Tuberculin T.R. in phthisis, and on the trial of sulphur as a curative agent.

During recent years public opinion has been roused to the gravity of this disease, which stands highest on the death roll of every country in the civilised world, which claims over sixty thousand victims every year in Britain, meaning an annual loss to the State of over three million sterling per annum in wage earning power. Other diseases are nothing as compared to the ravages of this. Pneumonia, the second "Captain of the

Men of Death" has been well called by Osler "the old man's friend", but phthisis claims its victims in the hey-day of Youth, in the full bloom of Manhood and Womanhood, when hearts are true and hopes are high, with the expectations of the future about to be realised. It is then that the White Death enters the home to take away the noblest and the best. This was the cold hand that left unfinished "Weir of Hermiston" at the words "a wild convulsion of brute nature"; this was the Pale Shadow that stilled for ever the songs of Keats, but for which we might have heard yet again that Magic Voice,

"The same, perchance, that found a path
Through the sad heart of Ruth, when, sick for home,
She stood in tears amid the alien corn.
The voice that oft hath charmed
Magic casements, opening on the foam
Of perilous seas, in faerielands forlorn".

The enormity of the disease being realised, the people are asking for a cure. Like the Athenians of old, they are clamouring for a sign, but no sign shall be given them. A Science that is not fifty years old has found the cause and sources of the disease, and is ever preaching its prevention to an unheeding generation. A cure for phthisis would mean that the inevitable and eternal chain of consequences of selfishness and self-interest should be broken, and were Almighty God to clear the white plague from the world to-morrow, men and women would breed it before to-morrow's morn. While thousands are spent every year in easing the sufferings of those afflicted, year by year the children of the nation are

being poisoned by tuberculous milk, to say nothing of those born into the unspeakable environment of our slums. Against all this the State seems powerless to move in the face of class interests, yet Science has spoken in no uncertain voice. From the thinker in the library and the scientist in the quiet calm of the laboratory the Fiat has gone forth "Every bovine is suspect, every tuberculous animal is a danger to man."

HALLIDAY G. SUTHERLAND.

Tain,

April, 1908.

H. G. Sutherland



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Submaxillary Hyperthropic Osteo-arthropathy Sept. 1907.

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MORPHOLOGY: The bacillus of tuberculosis is rod-shaped, varying in length from 1 to 5 μm and 0.5 μm in breadth. It is straight, curved or sigmoid, the protoplasm being usually homogeneous, but may and does show colourless spheroidal bodies resembling spores arranged in single file.

PLACE IN NATURE: Hesse holds that the bacillus has developed phylogenetically from a bacillus of the group Streptothrix. He believes this to be the evolution of the germ in the past, but does not consider the changes occurring at the present time.

CHAPTER I.

THE AETIOLOGY OF PULMONARY TUBERCULOSIS

In dealing with the aetiology of a bacterial disease it is necessary to consider firstly, the nature of the parasite, secondly, its sources, and thirdly, the means whereby man is infected.

A. The nature of the Tubercle Bacillus.

The infective nature of tuberculosis was first experimentally proved by Villemin in 1865, who so firmly held this view that he believed the phthisis of which Laennec died to be due to the infection of a wound twenty one years prior to the appearance of the pulmonary disease, but it was not until 1882 that the brilliant discovery of Koch² established the bacterial nature of this disease.

MORPHOLOGY: The bacillus of tuberculosis is rod-shaped, varying in length from 3μ to 10μ and 0.5μ in breadth. It is straight, curved or sinous, the protoplasm being usually homogeneous, but now and then showing colourless spheroidal bodies resembling spores arranged in single file.

PLACE IN NATURE: Hueppe³ holds that the bacillus has developed phylogenetically from saprophytes of the group Streptothrix. He believes this to be the evolution of the germ in the past, but does not consider the change is occurring at the present time. All tubercle bacilli

are of one species, their morphological and cultural differences being caused by adaption to various kinds of animals.

ZOOLOGICAL DISTRIBUTION: Tubercle bacilli occur in man the anthropoid apes, monkeys, bovines, pigs, birds, reptiles and fish.

STAINING PROPERTIES:- At body temperature, it readily takes the basic analine dyes, and retains these after treatment with acids, which property places it in the now large and well-known group of acid fast bacilli, of which the others best known are the Bacillus Leprosae, the Bacillus Xerosis, and the Bacillus Smegma. It is distinguishable from the leprosy bacillus by the fact that the latter always occurs in clumps and will not grow on artificial media, and from the other members of the group by its cultural peculiarities.

CULTURES: It grows at body temperature on blood serum, glycerine-agar, potato, and on bouillon. The colonies grow slowly, and appear at the end of the third week, as thin grayish scaly masses, varying according to whether the bacilli are human, bovine, avian or piscine.

DISTRIBUTION: The bacilli occur in all tuberculous lesions.

COMPOSITION: The composition and pathogenic properties of the bacillus will be considered in the Chapter on Pathology. (Chapter III.)

B. The Sources of the Tubercle Bacillus.

The scientific study of tuberculosis dates from Koch's discovery of the specific micro-organism, to which he held was due every manifestation of the disease, whether it occurred in man, bird, or beast. This was shortly followed by Ehrlich's discovery of the acid fast stain, which seemed to leave no doubt as to the identity of the organism, which caused such ravages among the human race and the bovines. Since then however, two great theories have been promulgated - the one, that bovine tuberculosis constitutes a danger to man - the other, that it is a distinct and, so far as human beings are concerned, an innocuous disease.

VIRCHOW'S BOVINE THEORY.

In 1889, Rudolf Virchow⁴, the founder of pathology, announced that the chief danger of tuberculosis to man was from the bovines, by the ingestion of the meat or milk of tuberculosis cattle. He also considered that pork constituted a danger, as pigs were greatly affected by tubercle of the cervical glands. This theory was obviously not based on experiment, but on the striking analogy that in the human race and in the bovines, tuberculosis is the most lethal malady, and that man for his nutrition consumes the flesh and milk of animals susceptible to a disease so fatal to himself. Other authorities have held and hold this view, but as Koch is associated with the denial of this theory, it is but fitting that the name of Virchow should stand as its protagonist.

BOVINE BACILLI IN HUMAN TUBERCULOSIS

KOCH'S HUMAN THEORY

In the very year that Koch asserted that the human

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family. In 1896 and later in 1898, Theobald Smith, drew attention to certain morphological differences between the tubercle bacilli of bovine and human sources. He showed that the bovine bacillus is short and straight, grows less vigourously on artificial media than does the human bacilli, but retains its characters through several sub-cultures, while on the other hand the human bacillus grows more quickly, and is long and slender.

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In 1901 Koch and Schutz, after a series of inoculation, feeding, and inhalation experiments, came to the conclusion that bovines are not susceptible to human bacilli, while they are readily affected by bovine bacilli and as a deduction from this Koch announced in London in 1901 "That (1) Tuberculosis of man and the cow were different, and that tuberculosis of the cow could not be conveyed to man; (2) That the regulations concerning milk, butter and meat, made against tuberculosis of animals and its transmission to mankind were not necessary."

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This theory is a deduction, based on two facts which Koch believed to be experimentally verified, that the bacilli of human and bovine tuberculosis are distinct species, and that bovines are not susceptible to human tubercle. In the intervening years a vast amount of experimental work has shown both these facts to be without foundation.

BOVINE BACILLI IN HUMAN TUBERCULOSIS

In the very year that Koch asserted that the human family was not susceptible to bacilli of bovine origin, ⁸ Ravenel was investigating the relations between bovine and human tubercle, and in June 1901, succeeded in isolating from the mesenteric glands of a child, a bacilli whose culture showed "every characteristic of the bovine tubercle bacillus, and must be regarded as having come directly from cattle." To Ravenel is the honour of having proved the bovine bacillus to be a causal organism of human tuberculosis.

In 1903, ⁹ Veszprémi investigated the comparative virulence of eight cultures of tubercle bacilli from human sources, and found the greatest variation, so that no standard of virulence for tubercle bacilli from human sources could be established.

The German Tuberculosis Commission ¹⁰ in 1904 examined thirty-nine specimens of tubercle bacilli from sputum, of which four were found virulent for cattle, these having come from lesions in children.

The Royal Commission of 1901 ¹¹ have now demonstrated in clear terms that the bovine bacillus is virulent to the human family. On this point the Commissioners summarise as follows: "There can be no doubt but that in a certain number of cases the tuberculosis occurring in the human subject, especially in children, is the direct result of the introduction into the human body of the bacillus of bovine tuberculosis; and there also

can be no doubt that in the majority at least of these cases the bacillus is introduced through cow's milk. Cow's milk containing bovine tubercle bacilli is clearly a cause of tuberculosis and of fatal tuberculosis in man."

"Of the 60 cases of human tuberculosis investigated by us, 14 of the viruses belonged to Group 1, that is to say, contained the bovine bacillus. If, instead of taking all these 60 cases, we confine ourselves to cases of tuberculosis in which the bacilli were apparently introduced into the body by way of the alimentary canal, the proportion of Group 1 becomes very much larger. Of the total sixty cases investigated by us, twenty-eight possessed clinical histories indicating that in them the bacillus was introduced through the alimentary canal. Of these 13 belong to Group 1. Of the 9 cases in which cervical glands were studied by us, 3, and of the 19 cases in which the lesions of abdominal tuberculosis were studied by us 10 belong to Group 1.

"These facts indicate that a very large proportion of tuberculosis contracted by ingestion is due to bacilli of bovine source."

BACILLI OF HUMAN SOURCE ARE VIRULENT FOR BOVINES

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As early as 1901 Ravenel reported the infection of three cattle by means of intra-peritoneal injection of human sputum, and in the same year Lartingar isolated from a human being a bacillus excessively

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virulent for bovines. De Jong derived a tubercle bacillus from sputum, which killed a calf in nineteen days. The virulence of the bacillus was verified by Sturrman,¹⁵ who also believed it conformed with the bovine type.

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Klebs and Rievel in 1902 induced a fatal infection in a calf by making it inhale tubercle bacilli of human origin, and Delephine¹⁷ produced positive results in two cattle with the use of human sputum. Schottelius¹⁸ obtained positive results by feeding cattle with human sputum, and in each experiment the lesions were so general that they must have eventually killed the animal, had this not been done to determine the extent of the process. McFadyean¹⁹ in 1903 found that by feeding monkeys with bacilli, the animals died irrespective of the source of the bacillus - bovine and human being equally lethal.

Speaking of the Pathology of the lesions in cattle, caused by inoculation of bacilli from human sources,²⁰ Orth remarks that the lymph glands "exhibit the some rapid calcification which is so characteristic of perlsucht; that is to say, their condition does not resemble that common in human tuberculosis", but genuine perlsucht, although the infection was with bacilli derived from man."

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Mohler and Washburn have tested the virulence of bacilli of human origin on cattle by inoculation with the following result: "Of the 20 cattle submitted to the test, 3 failed to show any resulting tubercular lesion; 2 gave an extension of the micro-organisms no

further than to the prescapular or to the prescapular and retropharyngeal glands; the remaining 15 developed tuberculosis of a generalised character. Out of these 15 animals, 3 died from the infection, and another was killed when in a moribund condition. It is of further interest to note that all of these fatalities were brought about by the use of infectious material derived from human sources, 2 direct from the culture, and 2 from tissue emulsion, after passage through cats."

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The Royal Commission obtained similar results. The viruses of 60 cases of the disease in man were isolated and of these 14 were found to be extremely virulent to bovines. The Commissioners report: "We may say definitely and at once that these effects appear to us to be absolutely identical with the effects of the bacillus of bovine tuberculosis; we have wholly failed to discover any essential differences between the one and the other; both are equally virulent, that is, equally able to set up tuberculosis in bovines and other animals."

CONCLUSION

The results of these researches, which have extended over the past six years, make it absolutely clear that Koch's original facts are erroneous.

Firstly, although the cultural, morphological, and pathological peculiarities of the human and bovine bacillus are fairly constant, they are yet mutable, and the characteristics of either bacillus may be altered by residence in the other host.

Secondly, the fact that bacilli are obtained from human sources, which are virulent for bovines and resemble the type of bacillus most frequently found in bovines, must logically mean, either that cattle are susceptible to human tuberculosis or that bovine tuberculosis is virulent to man. Whether either or both of these deductions be accepted, they constitute a clear demonstration of the fact that bovine tuberculosis is transmissible to man.

AVIAN TUBERCULOSIS

It is probable that avian tuberculosis plays but little part in the infection of man, but the question of the morphology of the avian bacillus is of extreme interest, as throwing a light on the mutability of the tubercle bacillus. All variety of birds are liable to be affected, and in them it appears that infection is solely through the alimentary canal, either from the ingestion of polluted faeces, or from the eating of rats and mice. It was previously supposed that pulmonary tuberculosis was of rare occurrence in birds, but in 1904 Rabinowitsch²² found that 60% of tuberculous birds suffered from the disease in the lungs.

That transition between the avian and the human type is possible has been proved by Pansini²³, who isolated an avian strain from a phthisical patient, and by Phisalix²⁴ who found that the culture derived from a tuberculous emu developed on different plates both the characters of avian

and of human bacilli, which were extremely virulent to the guinea pig, so that this observer considers it represented a transition stage, showing the characteristics of the other types. Eberlein ²⁵ has shown that the bacilli of tuberculosis in the parrot is identical microscopically, culturally and pathologically, with the human type. As he further succeeded in infecting parrots with tubercle from man, it is probable that these birds are susceptible to human tuberculosis, and derive their infection from this source.

These investigations justify the belief that avian and human tuberculosis are not separate diseases, but that both bacilli are but varieties of the common species, being modified by the tissues of the different hosts.

C. The means whereby Man is infected.

Man may become infected with tuberculosis by:

1. Congenital Infection.
2. Inoculation.
3. Inhalation of bacillus
4. Ingestion of bacillus.
5. Absorption of bacillus by the tonsil .

1. Congenital infection is possible, but rare, and will be considered under "Phthisis and Marriage".

2. Inoculation is also extremely rare. There is no proof of the infection ever having been carried through calf lymph, but the case is recorded of the tuberculous rabbit who caused tuberculosis of a circumcision wound.

3. Aerogenous infection by inhalation of the bacilli. The earliest view as to the means of tuberculous

infection was that the bacillus entered the body in the milk of diseased animals, but when Still called attention to the preponderance of lesions in the bronchial glands, this was generally accepted as an indication of aerogenous infection, which was held as proved when Koch in 1901 announced the duality of human and bovine tuberculosis. rapidly loses its virulence. He holds that

The aerogenous theory has been based on four factors

(a) Koch's assertion that bovine tuberculosis was a separate affection made it probable that the disease in man was caused by inhalation. This assertion has since been disproved. (b) The sputum of phthisical patients contains 'the virus', and such patients are therefore, during the course of their disease, disseminating tubercle bacilli. Nuttall²⁶ calculated that a phthisical patient throws off from one and a half to four and a third billion bacilli every twenty-four hours. As dried sputum is rapidly reduced to dust, the bacilli are widely distributed. Cornet²⁷ collected 118 dust samples from hospital wards and the rooms of consumptives, and of these 40 were found to be virulent to susceptible animals. The dust of a room in which a phthisical patient died was found to be infected six weeks afterwards. Strauss²⁸ found tubercle bacilli in the nostrils of 9 out of 29 assistants, nurses and attendants at the Charité Hôpital, Paris.

It is facts such as these which have given such support to the aerogenous theory, but for their proper consideration certain qualifications must be noted.

In the first place all the positive results cited above were obtained by the inoculation of susceptible animals, and it is generally recognised to be a matter of extreme difficulty to induce tuberculosis in an animal by the inhalation of tuberculous dust. Cadéac²⁹ has shown that sputum dries slowly, is pulverised with difficulty and rapidly loses its virulence. He holds that the transmission of tuberculosis by inhalation of dried sputum has not been proved. The oft quoted experiment of Tappeiner³⁰ was done by keeping the animals in a closed chamber into which he sprayed sputum in large quantities. A boastful attendant who entered this chamber succumbed to generalised tubercle in fourteen weeks. On account of the large quantities of moist sputum which was used, it is quite possible that this was an ingestion and not an inhalation experiment.

Further, Sangman³¹ has shown that in phthisis even a slight cough will bring up large quantities of droplets containing the bacilli, and that in laryngeal tuberculosis even greater quantities are thrown around, so that the physician, his instruments, and the patient's surroundings are constantly bespattered with droplets containing tubercle bacilli; which must obviously be inhaled in large numbers by anyone in the immediate vicinity of the patient. Yet in spite of this, he finds that among 238 previously healthy lung specialists and laryngologists, exposed for five and a half years to the risks of inhalation, only two have developed tuberculosis. From these facts he deduces that "inhalation by

healthy adults of droplets containing tubercle bacilli has no or almost no significance in the dissemination of tuberculosis." (c) It was Still who first pointed to the preponderance of lesions of the bronchial glands in pulmonary tuberculosis, and deduced from this that inhalation constituted the most common form of infection.³² His observation has since been confirmed by Holt³², who found the lungs affected in 99% and the bronchial glands in 96% of cases of phthisis, and by Hamburger and Sluka³³, who found that of thoracic tuberculosis in children, the lungs were affected in 50% and the bronchial glands in 98%.

It is by no means certain, however, that lesions in the bronchial glands are due to inhalation of bacilli by the lungs. On the contrary, Calmette, Guérin, Deléarde and Vallée, to whose work further reference will presently be made, have shown that in young bovines fed with food containing bovine bacilli, the bacilli will pass through the intact intestine without causing injury, will remain in the mesenteric glands without the formation of a tuberculous lesion, but will later cause tuberculous changes in the bronchial and retropharyngeal glands, with or without lesions in the lungs.³⁴ (d) Comby maintains that the infant mortality due to tuberculosis is in favour of the aerogenous theory. He points out that the infant mortality is slight in the first six months of life, but increases slightly up to the age of 1, then greatly increases during the second year, after which it steadily mounts up to the tenth year. It is

argued that the infants environment being at first so restricted, there is little chance of infection from contact with tuberculosis patients, hence the few deaths from phthisis in the first six months of life, but that as it goes about, the chance of infection, and with it the mortality ^{from} ~~of~~ tuberculosis, steadily rises.

These deductions appear to the writer to be absolutely unsound. It is true that the mortality from tubercle is low in the first year of life, but when the mortality does rise it is due to tabes mesenterica and meningitis, forms of tuberculosis generally admitted to be due to the ingestion of bacilli. It is most probable that the early low mortality is due to the fact that the child is fed at the breast, and that the later rise is caused by the consumption of cow's milk. This argument will be further developed in considering the Ingestion Theory of Infection.

4. The Ingestion Theory. As we have seen the aerogenous theory of tuberculous infection is by no means based on so solid a foundation as was first supposed, and with the waning belief in this means of contagion there is a growing opinion that the chief danger of pulmonary tuberculosis lies in the ingestion of the bacillus in contaminated food. This view is founded on four circumstances, in support of which a vast amount of evidence is rapidly accruing. It has been experimentally shown that in animals the ingestion of tubercle bacilli is the simplest and quickest method of infecting the lung. Secondly it is known that bovine tuberculosis is

capable of infecting man, and if in addition to these facts, we consider the amount of tuberculosis in childhood, and the amount of diseased milk, meat, butter and cheese which is placed on the market, it is clear that the ingestion theory is well founded.

(a) Calmette and Guérin^{35,36} have shown that in calves, goats, and adult bovines, when bovine tubercle bacilli are introduced into the alimentary canal by means of an oesophageal sound (to prevent any possibility of contaminating the air passages) there invariably follows from one such ingestion tuberculosis of the thorax. They find that animals will thus contract tuberculosis by absorption from the intestine without visible lesions in the digestive tube. In young animals the bacilli are arrested in the mesenteric glands, where they remain a longer or shorter time without causing a lesion, but in adult animals the defensive action of these glands is much less active and the bacilli enter the main lymph stream, from whence they are carried to the lung by the pulmonary artery. There they cause tubercles at the end of 30 to 45 days, the lesions being situated chiefly beneath the pleura at the upper and anterior borders of the lungs. In its passage along the lymph channels, the bacillus causes lesions of the bronchial and retropharyngeal glands.

(b) As the bovine bacillus is now proved to be virulent to man, it follows that the ingestion of diseased food must be attended by grave risks of pulmonary tuberculosis. At the same time, it has never been

tuberculosis in childhood, as compared with tuberculosis in maintained, although some of the opponents of the Ingestion Theory would have it so, that the sputum of phthical patients does not also constitute a risk. A grave risk is undoubtedly present, but it is not the risk of inhalation. A tubercle bacillus from infected persons, whether it exist in the air in the form of a fine spray or among dried particles of dust, is first arrested in the upper air passages, and is later drawn or drops into the mouth, to be swallowed, absorbed by the intestine, and hence reaches the lung by the lymph channels. It is also certain that food may be contaminated in its handling or preparation by phthical patients.

(c) A consideration of the incidence and variety of tuberculosis in infancy and childhood gives striking support to the Ingestion theory. Thus Heubner³⁷ found that of 800 infants, none were tuberculous under the age of 1, but that 26 were affected between the ages of 3 and 4. The difference of the environment of an infant under 1, and of a child of 3 or 4 is quite insufficient to explain the great increase of tuberculosis, for the environment of all such children is that of their mother, and the cause is rather to be found in the fact that infants under 1 are not generally consumers of cow's milk, while after that age cow's milk forms their stable diet. Again it is known that intestinal tuberculosis is more frequent in the child than in the adult. Boneme³⁸ states that the percentage of primary intestinal tuber-

culosis in childhood, as compared with tuberculosis in general is 23.84, in adults 16.03 and in the aged 6.25 per cent. When we remember that the child inspires the same air as the adult, it would be necessary in order to explain these figures on any but the ingestion theory to postulate that the child is not so liable to Pulmonary troubles as is the adult. This we know is not the case, and the true explanation is to be found by analogy with Calmette's experiments on the bovines. In young animals the bacilli absorbed from the intestine are arrested in the mesenteric glands, while in older animals, these glands being less resistant, the bacilli pass upwards to the lung. Thus in the child the ingestion of tubercle bacilli will cause *tabes mesenterica*, or, if the infection be very great, tuberculous meningitis, but in the adult pulmonary tuberculosis will result. The statistical study of the question will be more fully considered in the section dealing with "Age as a predisposing cause of Pulmonary Tuberculosis".

(d) Having appreciated the danger from the ingestion of tuberculous food, it but remains to briefly indicate the vast number of tuberculous cattle, whose carcasses or products in the form of milk, butter and cheese, find their way to the markets. The danger from this source has been appreciated by the Royal Commissions of 1895 and 1901. The Commission of 1898 reported that "any person who takes tuberculous matter into the body incurs risk of acquiring tuberculous disease."

TUBERCULOUS MEAT.

remarkable fact that whenever tuberculous disease at all

In Leipzig ⁴⁰ in 1895, of all cattle slaughtered over one year, it was found that 33.2% were tuberculous, while of cows 43.5% were so affected. It is difficult to obtain figures for this country, but of 500 carcasses of cows examined by King ⁴¹ 47.7% were tuberculous. Further the occasional seizures of meat by authorities give some idea of the nefarious trade which is carried on with this product. Mearns Fraser ⁴² of Portsmouth has recently revealed an appalling state of affairs so far as the London market is concerned. At Petersfield in one year 54 cows were sold for sums from 10/- to 30/- each, and at Chichester 7 cows were sold from 5/- to 65/- each. The carcass sold for 5/- was sent to Portsmouth, where it was seized and condemned for tuberculosis. One may only add that Sims Woodhead ⁴³ has proved that cooking does not sterilise the inside of a joint nor the interior of a roll of meat weighing 3 or 4 lbs.

TUBERCULOUS MILK

If the source of any milk were cows suffering

It is to milk, however, that everything points as the great cause of tuberculosis. Nor is the reason far to seek, for the most frequent disease among cows is tuberculosis, and milk is a product which in some form reaches every member of the population.

As we have seen vital statistics point to milk as being the great disseminator of tuberculosis, and on this point Collingridge ⁴⁴ wrote in 1902. "It is a

remarkable fact that while tuberculous disease at all ages has greatly diminished in this country during the last half century, that among children in the first years of life has practically remained stationary. The diminution in tuberculous diseases at all ages is due to the great improvement in the hygienic condition of the people generally, while the high rate among young children is probably owing to the ingestion of infected cow's milk. Observations show that a very large percentage of milk cows are affected to some extent with tuberculosis."

The occasional sampling of milk has proved this view to be well founded. Klein ⁴⁵ in 1904, found that of 39 samples of milk taken at railway stations, and representing the supply of 22 counties, 3 of these contains tubercle bacilli. In 1905 of 22 samples, 2 were tuberculous, and in 1906, 2 infected samples were found out of a total of 25.

THE INFECTION OF MILK

If the source of infected milk were cows suffering from tuberculosis of the udder, and from these alone, the danger would be relatively small. Unfortunately the source is more general, as the main pollution comes from the faeces of tuberculous cattle. Schroeder and Cotton ⁴⁶ have shown that a tuberculous cow passes 37 million bacilli daily in the faeces, and these are dropped on roads, f

fields and barns. Further the cattle are splashed and coated with the faeces, which are swished around by the tails of the animals, with the result that no ordinary precautions will prevent their entering the milking pail, which is proved in most dairies by the appearance of the straining cloth. One tuberculous cow in a byre is capable of polluting the entire milk supply of the dairy. The following are Schroeder and Cotton's striking conclusions:-

1. "Tubercle bacilli are disseminated with the faeces of tuberculous cattle. This is shown to be the case by microscopic examinations, by inoculation tests with guinea pigs, and by ingestion experiments with hogs.
2. Faeces are the most dangerous factor in the dissemination of tubercle bacilli by cattle affected with tuberculosis. In this respect faeces must be regarded as having a place with cattle similar to that commonly accorded to sputum with tuberculous persons.
3. It is not alone the faeces of visably affected cattle which disseminate tubercle bacilli in a way that is dangerous to man and animals, but also the faeces of cattle so slightly affected that the diagnosis of tuberculosis with them depends entirely on the application of the tuberculin test.
4. Tubercle bacilli that are swallowed by cattle are to a great extent passed entirely through the digestive tract and out with the faeces without loss of

infectiousness. As cattle do not expectorate, the infectious matter that is coughed up from their lungs is swallowed, passed through their bodies, and scattered with their faeces.

5. Bacilli may reach the environment of tuberculous cattle from their mouths, but this is evidently of rare occurrence compared with the dissemination through faeces especially when the cattle are not visibly tuberculous."

5. ABSORPTION OF TUBERCLE BACILLI BY THE TONSIL

So far this method of infection has attracted little notice, but Goodale ⁴⁷ gives an interesting observation on the tonsils which he removed from seven children, suffering from tuberculous glands in the neck. These tonsils microscopically showed tubercles and giant cells and when inoculated into guinea pigs, gave rise to tuberculosis. The cultures of four strains of the bacilli were studied by Theobald Smith, who found they were of the bovine variety. It is probable that the tonsil has greater powers of absorption than is generally supposed.

1. HEREDITY.

From the time of Hippocrates heredity has been supposed to play a considerable part in the predisposition to phthisis and the physical characteristics of the product of tuberculous parents are well known clinically.

It is only, however, during recent years that the scientific study of statistics has thrown considerable light on this problem. The results are as yet by no means complete, and a vast amount of material has yet to be sorted, before anything like an accurate finding can be obtained.

There are here appended two tables, showing according to different authorities the number of tuberculous parents corresponding to 100 tuberculous and 100 non-tuberculous children. As Hilton Fagge has pointed out, one fact must be remembered in appreciating the absolute value of these tables of heredity, which is, that the children of tuberculous parents are more liable to accidental infection, as well as to hereditary predisposition.

AUTHORITY	NATURE OF OFFSPRING and HISTORY	NUMBER of TUBERCU- LOUS PARENTS to 100 TUBERCULOUS INDIVI- DUALS.
Pearson	Tuberculous, incom- plete.	35
Pearson	Tuberculous complete	47
Dock & Chad- bourne (48)	Tuberculous presum- ably incomplete	27

Authority	Nature of Offspring and History.	Number of Tuberculous Parents to 100 Tuberculous Individuals
Kuthri (49)	Tuberculous, presumably incomplete	25
Fischer (50)	Tuberculous, presumably incomplete	36
Schwarzkopf (51)	Tuberculous, presumably incomplete	36
Buckhardt (52)	Tuberculous, presumably incomplete	37
Pope & Brown (53)	Tuberculous complete	90 (2nd series)
Pope & Brown	Tuberculous complete	97 (1st series)
Solly (54)	Tuberculous	28

TABLE II

Authority	Number of Tuberculous Parents per 100 Non-tuberculous Offspring.
Kuthri (49)	20.4
Schwartzkopf (51)	20.1
Buckhardt (52)	18.4
Fischer (50)	14.7
Dock & Chadbourne (48)	12.0
Pope & Brown	47.9

If we average these tables it will be found that of the parents of a hundred tuberculous children 47.7. will be tuberculous while of the parents of 100 non-

tuberculous children only 22.2 will be tuberculous.

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The following table constructed by Karl Pearson from corrected figures shows that non-tuberculous children have non-tuberculous parents, compared to parents of tuberculous children, in a ratio of 11.4 to 1.

TABLE III.

Parents			
Offspring	Tuberculous	Non-tuberculous	Totals
Tuberculous	157	509	666
Non-tuberculous	157	5,837	5,994
Totals	314	6,346	6,660

In the German Army 29% of phthisical patients gave a family history of tubercle. (Schjerning)⁵⁶

AGE.

During the last fifty years there has been a postponement in the age of the maximum mortality from phthisis, which affects both sexes, but males more than females, as seen from the following table taken from the Decennial Supplement to the Register-General's Report.

TABLE IV.

Phthisis, 1851-1900. Ages of maximum mortality.

Age-groups in Italics have the maximum Rates, the others being approximate.

Periods	Males	Females
1851-60	<u>20-25</u> , 25-35, 35-45	<u>25-35</u>
1861-70	25-35, <u>35-45</u>	<u>25-35</u>
1871-80	<u>35-45</u>	<u>25-35</u>
1881-90	35-45, 45-55	<u>25-35</u> , 35-45
1891-1900	35-45, <u>45-55</u>	25-35, <u>35-45</u>

In appreciating these figures several factors must be taken into account. For one thing the general improvement in the health of the community, following the public health legislation of the last fifty years, would do much to prolong the age of death from this as from other diseases. Again, the early mortality returns are extremely unreliable as in many cases the cause of death was reported on by lay persons where no doctor had been in attendance. At the same time during the last fifty years there has been a fall in the mortality from phthisis of 49%. This, however, will be more fully considered in "The State and Phthisis".

In view of the theory that the infection of the child means the disease in the adult, it is of interest to consider the annual mortality from *Tabes Mesenterica* and Tuberculous Meningitis per million living. In the

decennial period 1861-70, this was 576, while in the last decennial period, it was 433, that is a decrease of 25%.

Further, the following table (Tatham) shows that there is no change in the age period at which the majority of infants succumb to Tabes Mesenterica, the mortality of which amounts to 2.8 per 1000 births.

TABLE V.

England 1851-1903

Tabes Mesenterica (Tuberculous Peritonitis)

Deaths rates per million living, at each age.

(a) Persons of both sexes.

Ages	All Ages	0-	5-	10-	15-	20-	25-	35-	45-	55-	65-	75-
1851-60	260	1625	157	91	51	31	21	15	19	20	20	8
1861-70	295	1856	139	81	60	38	27	21	23	27	20	6
1871-80	319	2036	129	79	60	37	26	22	21	23	24	9
1881-90	277	1808	121	75	56	39	29	25	22	24	23	11
1891-1900	217	1458	103	68	58	46	38	34	30	27	21	9
1901-1903	172	1107	102	70	53	46	42	38	36	32	21	10

From these figures we are justified in stating that the amelioration of general conditions which has affected the age of mortality from phthisis in adults has not influenced the age period of tuberculous mortality in the child.

INFANCY AS THE PERIOD OF TUBERCULOUS INFECTION

The number of deaths from tubercle in infants under 5 is by no means the last point to be considered in

TABLE VI
 this connection, for of those children who do not die
 in this age period, it is certain that a vast number pass
 into other age periods with the tuberculous infection
 in their bodies, which may later develop into surgical
 or pulmonary tuberculosis.

In the Infant:

The infant is extremely susceptible to tuberculous
 infection. Wasserman found tuberculosis in a suckling
 who lived eight days in the house of a phthisical patient,
 and Reich records the case of a tuberculous midwife who
 was in the habit of starting respiration by applying
 her mouth to those of new born infants, with the result
 that 10 of the latter died of tubercular meningitis.

⁵⁸
 Botz found that 27.8% of those who died in the
 first year of life were tuberculous. Of 800 infants in
 his clinique Heubner ⁵⁹ had no cases of tuberculosis
 under 1 year, but 26 cases between the third and fourth
 years.

In School Children:

The percentage of declared tuberculosis in school
 children is small, as the figures of the following
⁶⁰
 authorities will indicate.

TABLE VI

Squire and Gowley (London)	found	0.47	Tuberculous
Leubuscher (Germany)	"	0.15	"
Newsholme (London)	"	0.3	"
Hay (Aberdeen)	"	0.1	"
Edin.Cannongate Schools	"	0.4	"
Greenwood (Blackburn)	"	6.7	"

If, however, we take the records of Sick Childrens
 61
 Hospitals a better idea is obtained of the amount of
 tuberculosis in childhood.

TABLE VII.

1906- Belfast Hospital for Sick Children	(No. of intern patients, 827 (No. tuberculous, 26.10 per (cent.
1906- Ulster Hospital for Sick Children	(No. of intern patients 247 (No. of tuberculous, 30.36 (per cent.
1905- Great Ormond Street, London.	(No. of intern patients 2,876 (No. of tuberculous 27 per (cent.
1906- Royal Edinburgh Hospital	(No. of intern patients 1,968 (No. tuberculous 20 per cent.
1905- Manchester Chil- dren's Hospital	(No. of intern patients 1,999 (No. tuberculous 21.3 per cent.
1905- East London Children's Hospital	(No. of intern patients 2,054 (No. tuberculous, 24.3 per cent.
1906- Glasgow Children's Hospital	(No. of intern patients 1,777 (No. tuberculous, 27,95 per cent
1907- Brompton Hospital	62 Out patient depart. No. of tuberculous 13 per cent.

These figures give the amount of declared tuber-
 culosis, compared to other diseases, and if we take

the post mortem records of children dying from all diseases, it will be seen that the numbers of infected children are far higher.

According to this theory, which the writer holds, a certain percentage of TABLE VIII tuberculosis is due to the

Percentage of Tuberculosis in Children dying of all Diseases in Children's Hospitals.

Bucharest Children's Hospital	60	found tuberculosis in	90%
Muller (Munich)	"	"	43%
Comby (Paris)	63	"	38.5%
Hamburger and Sluka (Vienna)	64	"	40%
Baginsky (Berlin)	"	"	18%

The large amount of alimentary tuberculosis in children will be gathered from the fact that Still in

266 Post mortems on children from 10 months to 9 years found 5 cases of tuberculous ulceration of the stomach - a relatively rare condition.

We thus find that the large percentage of tuberculosis of alimentary origin in children, taken in conjunction with the fact that cow's-milk forms their main source of nutrition, and that this is infected with the bacillus of bovine tuberculosis which is virulent to man, leaves no doubt as to the statement, already endorsed by the Royal Commission, that bovine tuberculosis is the chief source of danger to the child. Nor is this all, for every infected child does not die, there are some who pass on to later life with the bacillus latent in the

mesenteric glands, from where, as shown by Calmette's experiments on the bovines, it may pass on to the lung. According to this theory, which the writer holds, a certain percentage of pulmonary tuberculosis is due to the ingestion in childhood of the tubercle bacillus in cow's milk.

3. SEX.

It is a common belief that women are more affected with phthisis than men, but if we take the mortality from phthisis for all ages, it will be seen that such is not the case, as the following table taken from the returns of the last four decennial periods indicates.

TABLE IX.

The Mortality from Phthisis per million living.

Males	1550
Females	1245
Both Sexes	1392.5

At the same time a study of the age groups during the last decennium shows that more females succumb than males during the period from 15-20 years. It is at this time that the real mortality from phthisis for both sexes begins, from which it increases steadily to attain its maximum in women from 35-45, and in men ten years later 45-55.

Again, the wearing of corsets actually ensures full thoracic breathing, which if the theory of the stagnant apex were true, should insure the liability of women to this disease.

TABLE X.

Deaths from phthisis per million living at age

15 - 20.

Males	995
Females	1,290

It is probable that women have been less affected by the general improvement in the health of the community than have men, on account of the more sedentary indoor life they lead, and because of the strain of pregnancy and lactation.

Influence of Corsets:

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Ruata has put forward a theory, which has obtained credence in the lay press, that the greater mortality from phthisis among females from 5-25 than among men in the same age period, is due to the wearing of corsets, which produces faulty respiration, and predisposes to aerogenous infection.

While the figures are correct, the fact that the highest mortality among women is at the age period 35-45, makes it extremely improbable that the wearing of corsets has anything to do with the matter, and it is most likely than the increased male mortality after the age of 25, is due to alcoholism, the absence of which, prior to age 25, would tend to keep the male mortality low.

Again, the wearing of corsets actually ensures full thoracic breathing, which, if the theory of the stagnant apex were true, should lessen the liability of women to this disease.

4. RACE

The racial factor in the predisposition to phthisis counts as nothing compared to the hygienic conditions under which different races live, the latter alone determining their mortality from phthisis. Thus while phthisis is rare among the native races in Africa, it has been shown by Jones⁶⁶ that the mortality from consumption among the negroes of the United States exceeds that of any other people, and is due to the unsanitary conditions under which they live. Walker⁶⁷ has studied the question among the Oglala Sioux Indians, and finds that the disease has increased greatly among them, since they left their nomadic life, and have become tainted with the vices of civilisation. The Hebrews, on the other hand, enjoy a relative immunity from phthisis, which is to be explained by the cleanly, thrifty habits of even the poorest of this race. The following table from Osler⁶⁸ shows the mortality of different races in New York City.

TABLE XI.

Average Annual Mortality from Phthisis per
100,000 of Population.

Irish	645.73
Coloured	531.35
German	328.80
American Whites	205.14
Russian-Polish Jews	76.72

5. ENVIRONMENT.

(a) Houses.

A large body of evidence goes to show that to a certain extent phthisis is a house disease. Niven⁶⁹ investigated the deaths from phthisis in Oldham over a period of 11 years and found that, while the mathematical chance was that 68 houses should contain 2 cases, in reality 274 houses contained 2 cases.

⁷⁰Engelman tells of a flat in a house in which, after being tenanted for 1 year by a consumptive, there occurred 12 cases in 12 years, although prior to this it had been free of tubercle for 8 years. Kempf⁷¹ has reported the instance of a convent, in which, within four months after the introduction of phthisis, nine Sisters became consumptive.

The following striking case, where a whole family was decimated by the disease, has been recorded by

McDonald .

"A crofter at Tarbert, Morar, had acted as a beater or ghillie, in the summer season, and had lived in the same house for twenty-one years. Until the spring of 1906 the whole family, consisting of himself, his wife, five daughters and seven sons, the oldest being 21 years and the youngest 2, had always been healthy. In April, 1906, the eldest girl, aged 21, who had been in service, came home suffering from a suppurating finger. Symptoms of phthisis manifested themselves and she died of tuberculous meningitis on May 26th. In the following November another daughter, aged 14, was found to be suffering from well-marked phthisis in both lungs. Death took place on January 8th, 1907. During the time she was ill the father began to suffer from a cough, and the mother complained of pain in the abdomen and the left ankle. Two daughters, aged respectively 20 and 10, also complained of cough, and the baby aged 2 was anaemic and emaciated, also had a cough. Examination showed that the father was suffering from tuberculous disease of the ankle and there seemed to be reason to suspect the existence of similar disease in the abdomen; while the two daughters and the baby showed distinct signs of pulmonary phthisis. The baby died on January 3rd, and the daughter aged 10, about January 10th after an illness of about three weeks. The mother was removed to the Western Infirmary, Glasgow. The father who up to this point was the only one of the male side of the house to be attacked, did not improve. In March, however, it

was found that a boy aged 15, was suffering from phthisis. Meanwhile the father got worse and died soon afterwards."

There are two points of extreme interest in this case: Firstly, that the family had lived healthily in the house for 21 years, and that there was no predisposition to phthisis on either side; secondly, that the disease attacked the female side first, and that member of the family who was most likely to be concerned with the preparation of the food for the others.

In general the danger from a house depends on the habits of the occupants, and a careless consumptive will undoubtedly render his house a danger centre. On the other hand the writer knows a phthisical patient, where the disease is running an extremely chronic course, who has lived in a house for 20 years, without infecting any other member of a family of five. In this case the patient is a man and probably the danger is greatest where it exists in a woman, who has to deal with the food of the household.

b. Occupations.

Nursing: Does the nursing of phthisical patients render those so engaged more liable to infection, is a question on which great difference of opinion exists.

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Cornet has considered the mortality from phthisis for 25 years in 38 Catholic Nursing Institutions, with an average population of 400 in each, and in these the death rate from phthisis was 62.8% of all deaths, as against 15% for the general community. As the age group

of the deaths has not been dealt with, a reduction must be allowed on this score.

On the other hand, the statistics of the Brompton Hospital show that doctors, nurses, and attendants, are rarely attacked.

As we have seen it is far easier to induce pulmonary tuberculosis in animals by ingestion, than by inhalation, and as all those engaged in the treatment of phthisis must stand an equal chance of aerogenous infection, it is probable that the factor of infected food is very great in the causation of the disease in those brought into intimate contact with it.

Those occupations which cause the inhalation of irritating and toxic particles into the lung favour the onset of phthisis by lowering the pulmonary resistance to infection, whether that infection be aerogenous or haematogenous. Such occupations are those of brush-makers, tobacco cutters, hair-dressers, glass workers, stone cutters and coal miners.

With regard to hair dresses and barmen, classes in which a good deal of phthisis exists, the writer has come across several cases where the patients selected these occupations on account of their phthisis, as affording them what they term a "sheltered life".

Phthisis is frequent among mill workers, where the close, twice-breathed air, induces a general want of resistance to infection.

c. Poverty.

The effect of poverty, the evil of the cities, as a predisposing cause to phthisis, will be seen from the following table, taken from the mortality returns of the last decennial period.

TABLE XII.

Phthisis, Urban and Rural Mortality, Corrected

Rates per Million Living.

	Urban County Group.	Rural County Group.
Males	1,753	1,347
Females	1,250	1,240
Both Sexes	1,493	1,292

The mortality returns for Italy show this in a greater degree. In 206 of the largest towns the mortality from phthisis was 25 per 100,000, while in the villages it was only 16.5 per 100,000.

When we think what poverty means, a population, over-crowded and under-fed, of uncleanly habits, dwelling in the dark tenements of the slums, breathing foul and polluted air, earning their bread by long hours in gas lit workshops, drugged by bad liquor, and always living below par, it is no marvel that they, the indigent product of city life, should fall the first and easiest victims to the White Death.

d. Alcoholism.

Alcoholism, as a habit inducing bodily weakness and depression, and leading to poverty, must be reckoned as a predisposing cause of phthisis. At present there are no returns indicating how great a part it plays in this.

e. Vice and Crime.

These two factors, which are invariably accompanied by debility and disease, must rank extremely high as predisposing causes to phthisis. Baer⁷⁴ has shown the mortality from phthisis in prisons to be four times greater than it is outside. Taking the mortality from phthisis among the general community to be 15% of the total mortality, in gaols and prisons it has been found to be as high as 40, 50 or even 60 per cent of the total death rate.

f. Insanity.

It has long been known that the mortality from phthisis is greater among the insane than among the sane, and how great that mortality is will be seen from the following table prepared from the forty-eighth Annual Report on the General Board of Commissioners in Lunacy for Scotland. During the last decennial period the annual mortality from phthisis among the general population of the country was 1.3 per 1000 living, and with this the mortality of the insane is compared.

TABLE XIII.

The Asylum Insane in Scotland in 1905 General Population

	Population	Deaths from Phthisis	Deaths from Phthisis per 1000 living.	Deaths from Phthisis per 1000 living.
Males	6085.5	53	8.6	1.3
Females	6325.0	84	13.2	1.3
Totals	12410.5	137	11.0	1.3.

Making allowance for the fact that physical debility is the invariable accompaniment of insanity, these figures are startling in the extreme, and warrant an enquiry into the provision for phthisical patients in asylums in general. An interesting point is the greater mortality from phthisis, among women, out of all proportion to the larger number of female insane to male.

6. DISEASE.

a. Syphilis.

In considering the part played by other diseases in the predisposition to phthisis, we hold that syphilis should be ranked first.

Prior to 1853, it had been held that pulmonary tuberculosis might be caused by syphilis, for Gamberini was the first to insist that the so-called cases of pul-

monary phthisis cured by mercury were in reality syphilitic and that the diagnosis was in error, while Bazin⁷⁶ in 1866 stated that syphilis can only act as a debilitating cause in predisposing to phthisis, and is incapable of forming anything but "syphilitic tumours."

The passing away of the ancient conception of disease as an evil entity capable of assuming various guises, was followed by a reaction which found expression in the Hunterian dictum that two pathological actions could not occur in the same body at the same time. Not only so, but some of the older clinicians believed in an antagonism between the two diseases, thus Lanceraux⁷⁷ "the co-existence of syphilis and tuberculosis is so rare one may believe that between the products of tubercle and syphilis there is an antagonism, analogous to that which exists between tubercle and cancer. The general law of the body being true that the development of one pathological lesion will arrest the evolution of all processes of a different origin, it is only by the general debility of the economy that syphilis is capable of starting phthisis."

This too was the opinion of Mauriac⁷⁸ who had noted, with all respect to the hypothetical law, the sequence of phthisis on syphilis.

SYPHILIS AS A PREDISPOSING CAUSE TO PHTHISIS.

A clearer view was stated by Graves⁷⁹ in 1863, who maintained that syphilis greatly favoured the outbreak

of pulmonary tuberculosis, possibly by its debilitating influence, possibly by the abuse of mercury.

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Ricord remarks: "Syphilis is a blow to the economy capable of starting organic defects, and of awaking to activity every diathesis", while again we have the grim phrase of Landouzy : "Syphilis often makes the bed of phthisis."

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Fournier's ipse dixit on the question is as follows: "For my part, I have already seen numbers of young subjects in whom syphilis has powerfully exercised its depressing influence, become tuberculous in the first months or years of infection. I add, that developing in these conditions, phthisis sometimes evolves quickly, makes rapid progress, and kills without delay. Also, from my personal experience, and what has been said on this subject by the most competent observers, I do not hesitate to inscribe syphilis in the chapter on the aetiology of pulmonary tubercle."

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SYPHILIS IN THE PARENTS A CAUSE OF TUBERCLE IN THE CHILD.

In considering the part played by syphilis in the predisposition to phthisis, we may discuss this recent doctrine carefully elaborated by Emile Sargent . He considers that after an attack of syphilis the equilibrium of the body is restored, but that a special diathesis has developed, which is manifest in an acquired immunity against syphilitic infection, counter-balanced by a special receptivity to tuberculous infection. This

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he calls the "syphilitic soil" a state of body coming from old and worn out syphilis, and transmittable to descendants."

Thus he holds that the offspring of syphilitics may be affected by one of two conditions; by hereditary syphilis where the actual spirochaete has infected the child, or that, without having any manifestation of hereditary syphilis, they may inherit the "syphilitic soil". "Syphilis has created a special soil, which is transmittable to descendants in the same measure as syphilis itself."

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This distinction is analogous to that of Gaston, who described two forms of syphilitic heredity, complete and incomplete.

Sergent maintains that this syphilitic soil offers a special predisposition to phthisis. In support of this he cites five cases of pulmonary tuberculosis in children of syphilitics. These five cases were the only ones in which he was able to make a satisfactory investigation with regard to the possibility of syphilis in the parents and these all gave a positive result, the syphilis in each case being in the father.

This view is well worthy of further statistical test, and so far as predisposition in general is concerned there is no doubt but that the weakened product of syphilitic parents fall easy victims to a disease which stands highest in the death roll of every country in the world.

SYPHILIS INTENSIFIES A PREDISPOSITION TO PHTHISIS.

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Stieffel divided syphilitics into two classes - those who are predisposed to tubercle either by heredity or environment, and those who are not so predisposed. He found that the chance of tuberculous infection was far greater among the latter. ⁸⁶ Jacquet believes that the predisposition to phthisis in a young adult, who in the ordinary course of things might escape infection, is so intensified by the debility of syphilis, that the pulmonary disease is provoked and precipitated. ⁸⁷ Sargent writes "My personal observations have led me to consider that the aetiological relations between syphilis and tubercle are much closer than is generally admitted, and I am convinced that syphilis creates a site of election (terrain d'election) for tuberculosis."

b. Malaria.

During last century there was a belief abroad that malaria and tuberculosis were antagonistic and rarely seen together in one individual. This view was held by ⁸⁸ Boudin, who maintained that tubercle was rare in those countries where malaria was common, and that the converse was also true. Such an observation was probably due to the less perfect methods of diagnosis of that time, for the very reverse of this statement is true. In the south of Spain malaria and phthisis are rife, and frequently co-exist in the same individual. The profound anaemia following malaria explains its action in predisposing to phthisis.

c. Diabetes.

Phthisis is a grave and frequent concomitant of diabetes mellitus, not only appearing as a terminal infection in advanced cases, but also in milder forms where the patient appears to be holding his own until the onset of the pulmonary disease. The term "complication" as applied to this condition, is here avoided, for phthisis is not a complication of diabetes, as sometimes loosely described; a complication being the appearance of secondary lesions in the body due to the same agency as the primary disease.

The frequency of phthisis in diabetes is differently stated by various writers. Thus Sauvage⁸⁹, Brad-⁹⁰ley⁹¹, Bouchardat⁹¹, and Contour⁹¹ have regarded it as the inevitable consequence of diabetes, while Bouchard⁹² found that 8% and Griensinger⁹³ that 43% of diabetics died phthisical.

Contour⁹¹ held that the appearance of phthisis in diabetes was the last result of severe diabetes, and was a true complication of that disease. Such a view⁹² is no longer tenable, nor is Bouchard's⁹² belief that "Diabetic phthisis exists almost exclusively in those patients who at the same time have albuminuria", based on the fact that he only saw one case of phthisis in diabetes where albumen was absent. This is controverted⁹⁴ by Dieulafoy⁹⁴ of whose twelve cases only four had albumen, and who suggests that in many the albuminuria appears with the tuberculosis. Other writers have considered the increase in sugar in the body to render it a

more favourable nidus for the tubercle bacillus, but the lowered resistance from a wasting constitutional disease is quite sufficient to explain the great susceptibility of these patients to tuberculous infection. Da Costa⁹⁵ showed that in 16 cases of Diabetes mellitus the Opsomic Index to Staphylococcus aureus was subnormal, varying from 0.36 to 0.72, the average being 0.62. No relationship was found between the Opsomic Index and the amounts of sugar in the urine, or the leucocytes, haemoglobin, and red cells in the blood.

d. Bronchitis.

Bronchitis is frequently stated to be a predisposing cause of phthisis, but it is an open question in how many of these cases is the bronchitis a tubercular process, and in how many is actually a predisposing cause, by lowering the pulmonary resistance to latent or newly acquired infection.

e. Measles and Whooping Cough.

These two diseases are the most frequent fore-runners of the acute tuberculous broncho-pneumonia in the child. The respiratory catarrh associated with them leaves damaged areas in the lungs, which form suitable niduses for fresh or old standing infection.

f. Heart Disease.

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Stow has shown that organic lesions of the right heart, which produce pulmonary anaemia, set up a condition which is favourable to the growth of the tubercle

bacillus, and further that such lesions are most frequently associated with pulmonary tuberculosis. On the other hand, organic lesions of the left heart, when associated with well marked secondary changes of dilatation and hypertrophy, produce long standing passive hyperaemia of the lungs, a condition unfavourable to the growth of the bacillus, and which often effects the cure of a pre-existing tuberculous condition. The explanation of this is that even slight disturbances of the cardiac action influence the pulmonary circulation, and lesions accompanied with well marked pulmonary stasis are rarely followed by phthisis, because the immunising agents of the blood are concentrated in the lungs.

TRAUMA

⁹⁷ Mendelssohn has reported nine cases where a contusion of the chest, unaccompanied by fracture of the ribs or laceration of the lung, was followed by phthisis. Such cases are explicable by the fact of the injury causing a lowered resistance and so favouring the outbreak of latent infection.

CHAPTER III.

THE PATHOLOGY OF PULMONARY TUBERCULOSIS.

We will consider the pathology of pulmonary tuberculosis in the following order.

- A. Means of pulmonary tuberculous infection.
- B. The Pathology and Chemistry of sputum.
- C. The Pathology of chronic phthisis.
- D. The Pathology of sub-acute pulmonary tuberculosis.
- E. The Pathology of acute pulmonary tuberculosis.

A. Means of Pulmonary Tuberculous Infection.

The tubercle bacillus gains access to the pulmonary tissues in one of three ways: - (1) by the blood vessels, (2) by the lymphatics, or (3) by the air passages.

(1) Haematogenous infection is either a metastatic process whereby the bacillus is carried to the lung from some distant focus of infection such as a tuberculous lymph gland, or it is the means whereby the ingested bacilli reach the lungs. According to the number of bacilli which enter the blood stream there results a miliary infection or a circumscribed local infection of the lung.

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Calmette and Guérin found that in the bovines the tubercle bacillus after ingestion reaches the lung through the blood stream, and that in thirty to forty five days after infection tubercles appear beneath the

pleura at the upper and anterior borders of the lungs. The granulations never develop primarily in the alveoli, but always commence in the lung capillaries, with secondary involvement of the alveoli and bronchioles.

The work of Rabinowitsch⁹⁹ also points to this being the most frequent source of spontaneous tuberculosis in the apes. Koch believed that in monkeys the lung was the first organ to become infected, and that in these animals the disease took on a type corresponding to human military tuberculosis. In 45 tuberculous apes, however, Rabinowitsch found that in 5 alone was the thorax only affected, that in 9 the abdomen only was diseased, and that in 31 both the thorax and abdomen revealed tuberculosis. From this he deduces that in monkeys the intestinal origin plays a greater part in tuberculous infection than does the inhalation origin.

(2) Lymphogenous infection occurs when the bacilli are carried to the lung by the lymphatics from a tuberculous lymph gland, or from some centre of tuberculosis in the bones of the thoracic cavity. Once tuberculosis has started in the lung, this is the most frequent means by which the disease extends, for the bacilli find their way into the interalveolar, interlobular, peribronchial, circumvascular and pleural lymph channels, with which the lungs abound.

(3) Aerogenous infection is said to occur when air is inspired containing tubercle bacilli in dried dust. Against this, as being a common method of infection, is the fact that it is extremely difficult to induce pul-

monary tuberculosis by this method in animals, and that the greater amount of all dust in the atmosphere is arrested in the upper respiratory passages, on account of the "sheer" and rotatory movement which the air acquires when passing through the nose. At the same time, it is by this means that tubercle bacilli may reach the lung from previously infected points in the respiratory system, such as a laryngeal ulcer, the bursting of a tuberculous bronchial gland into a bronchus, or by the bursting of a closed cavity in the lung.

B. The Pathology and Chemistry of Sputum.

Sputum is an alkaline fluid containing nummular masses of greenish material, the product of pathological changes in the respiratory system. Its specific gravity is from 1.008 - 1.026, averaging 1.013, and varies according to the number of leucocytes. The greenish colour is due to pigment, which may be bacterial in origin. In cases where large cavities favour bacterial decomposition, the reaction may be acid.

In phthisis the sputum contains epithelial cells from all parts of the respiratory tract, fibrin, elastic tissue and leucocytes, chiefly of the poly-morpho nuclear variety. Besides its chemical constituents sputum contains various enzymes derived from leucocytes and bacteria.

Wanner¹⁰⁰ found that the amount of proteid contained in the sputum of different diseases varies considerably. In bronchitis it is small, and in bronchiectasis it is present in the form of uncoagulable nitrogen

on account of the autolysis which goes on in the enlarged bronchi. In phthisis the proteid does not exceed 1%; in pneumonia it is 3%, and in gangrene of the lung it is still higher. If the proteid in sputum on boiling gives more than a slight turbidity, it indicates that inflammation is present, so that in a diagnosis between acute tuberculosis and infarction, a large amount of proteid would indicate the first condition.

Renk ¹⁰¹ has considered the amount of proteid in sputum with reference to the loss of body proteid in phthisis. He found that the sputum of three patients, which averaged 145 grms. per diem in two cases, and 82 grms. per diem in the third, contained 5.6% of solids, composed as follows - Mucin 2.3%, proteid .1 to .5%, fat .3 to .5%, and ash .8 to .9%. The daily loss of nitrogen was .75 grms. which is 6% of the amount of nitrogen lost in persons undergoing starvation.

Plesch ¹⁰² has found that of all calorics of heat lost in cases of advanced phthisis, 4.8% are lost in the sputum.

The following table shows the organic constituents in the sputum of typhoid bronchitis, and various forms of phthisis (Bokey from Ott ¹⁰³).

	Typhoid Bronchitis	Fibroid Phthisis	Early Phthisis	Phthisical Cavities	Advanced Phthisis	Advanced Phthisis
Fat	.224	.845	.462	2.469	3.468	9.725
Free fatty acids	Trace	.184	.521	.370	.307	.902
Soaps	"	.380	.430	.537	.516	3.973
Cholesterin	"	.4	1.617	.172	1.160	.141
Lecithin	"	traces	1.543	-	1.165	1.245
Nuclein	"	.102	-	-	.260	.489
Proteid	.898	2.040	-	4.430	3.455	5.115

Muller ¹⁰⁴ stated that purulent sputum digested fibrin, while non-purulent sputum did not, a fact which is now known to depend entirely on the number of leucocytes present. For the same reason, as all the exudates are digested by leucocytes, sputum contains proteoses, peptones, and amino-acids, according to the number of leucocytes present, so that the presence of these bodies is greatest in pneumonia.

The fats present in sputum depend on the number of pus corpuscles, and the amount of fatty degeneration in the pus. Jacobson ¹⁰⁵ obtained .08 to 1.6 grms. of fatty matter per diem in sputum, which was composed of soaps 14.76%, higher fatty acids 15.79, water soluble fatty acids 10%, Lecithin 13.58%, and Cholesterin 10.49%.

Wells ¹⁰⁶ gives the following differences in the sputum of chronic and of acute phthisis.

Constituents	Chronic Phthisis	Acute Phthisis
Water	94.55	93.38
Organic Matter	4.67	6.88
Inorganic Matter	.78	.74
100 parts of the salts contained		
Chlorine	35.78	33.40
SO ₃	.70	.80
P ₂ O ₅	13.05	14.15
K ₂ O	24.07	19.99
Na ₂ O	27.90	31.69
Calcium Phosphate	1.63	4.32
Iron Phosphate	.09	.14
Mag. Phosphate	1.20	-
Ca. and Mg. carbon and sulphate	1.74	.22
Salicylic acid	.9	.3

The Mucin in sputum with hydrochloric acid gives 33.6% of glucosamin, which indicates the amount of mucin. As this body is found in large quantities in chronic bronchitis and in very small quantities in phthisis, it might be of some value in the differential diagnosis.

C. The Pathology of Chronic Phthisis.

Historical Sketch.

The oldest medical writers have described the symptoms of pulmonary tuberculosis, and the idea that consumption had some causal relation to tumourous formations in the lung is also of ancient date. The expression "tubercle" was first used by Sylvius ¹⁰⁷ at the end of the Seventeenth century, but it was not until the end of the Eighteenth century that Baillie, ¹⁰⁸ an Englishman, gave the first exact description of the appearance of young tubercles in the pulmonary tissues, and he is therefore to be regarded as the discoverer of the granulation tubercle, and the first to point to these as leading to consumption.

It was Schonlein ¹⁰⁹ who in 1835 introduced the word "tuberculosis." Shortly before this Laennac ¹¹⁰ established the unity of all tuberculous processes in the body. He believed in a "primary tuberculous substance", which if transplanted to suitable tissue would cause tubercle formation, and was thus possessed with infectious properties. His primary tuberculous substance had the power of building up tuberculous tumours of a gelatinous substance, which later on became grey, then yellow. Laennac will always be regarded as the first great

authority on tuberculosis, and if we read in the light of modern knowledge, "bacillus" for "primary tuberculous substance", it will be seen what an accurate view he held of this disease from ~~clinical~~ ^{clinical} knowledge alone. At the same time, as the old terms had a far wider meaning than those of our modern nomenclature, it is better that they be avoided, as little analogy exists between the two at the present time.

Distribution of the lesions in the Lungs.

1. Catarrh and proliferation.

In 427 cases of chronic phthisis, Osler ¹¹¹ found that the right apex was involved in 172, the left in 130, and both in 111. This finding endorses the truth of Laennec's statement that the oldest disease is to be found at the apices, and a lung affected by old standing phthisis will show, from the cavity at the apex, every stage of pulmonary tuberculosis. The lesion does not usually commence at the extreme apex, but at a point an inch to an inch and a half below the summit near the posterior and external border. From the right upper lobe the disease extends to the middle lobe of the same side, and first attacks its apex, which is situated posteriorly opposite the 5th dorsal spine. Following the involvement of the middle lobe on the right side, or the lower lobe on the left side, the disease next attacks the opposite apex.

Chronic phthisis seldom starts primarily in the basal lobes, and according to Percy Kidd ¹¹² the proportion of primary basal to primary apical lesions is 1 to 500.

PATHOLOGICAL PROCESSES IN CHRONIC PHTHISIS.

The pathological processes at work in chronic phthisis are -

1. Catarrh and proliferation.
2. Consolidation.
3. Caseation
4. Fibrosis and calcification.
5. Excavitation.

1. Catarrh and proliferation.

When a tubercle bacillus settles in the lung, whether it be in a capillary blood vessel, a lymphatic, a bronchiole or alveolus, the first result of its multiplication is invariably a cellular inflammation and proliferation of the surrounding tissues. The epithelial and connective tissue elements of the capillaries, lymphatics, bronchioles and alveoli all take part in this proliferative action, with the result that they are occluded by cellular elements.

2. Consolidation. This is the second stage of the tubercular nodule, which is a patch of broncho-pneumonia, whose size depends on the area of the lung infected by bacilli. A microscopic section of such a tubercular nodule shows the thickened walls of the bronchioles and alveoli to be infiltrated by epithelial cellular elements and leucocytes, which also occlude the alveolar spaces and the bronchioles, while scattered through the nodule are tubercle bacilli.

One characteristic of these patches of tubercular

broncho-pneumonia is the presence of giant cells - large cells with many nuclei. Some have thought these cells to be derived from the blood, others that they were a result of the fusing of several epithelial cells, but Podwysotski and Pirone ¹¹³ have experimentally shown that they are a preliminary phase of epithelial hypertrophy, due to the nuclear divisions being temporarily in advance of the protoplasmic.

Partial obliteration of the capillaries always occurs in the tuberculous area, but when the proliferative process attacks the walls of arteries or veins, the latter are weakened or destroyed, and there results the initial haemorrhage of phthisis.

3. Caseation,

The next change in the tubercular nodule is caseation, which commences at the centre, as it is due to the action of the tubercle bacillus, and a section through a caseous nodule shows the bronchioles and alveoli to be plugged with cheesy material.

Chemistry of Caseation.

Caseation is a form of coagulative necrosis whereby dead tissues are reduced to a condition resembling cheese, a mixture of coagulated proteid and finely divided fat. Thus in caseation we have coagulation of proteid and decomposition of fat.

Caseation is a direct result of the living tubercle bacillus, for Kelber ¹¹⁴ has shown that the dead tubercle bacillus does not produce this condition, as the substance

inducing it is not easily diffused from the bodies of the bacillus. According to Auclair ¹¹⁵ it is due to a fatty substance in the bacilli which can be extracted with chloroform - "tuberculosamin."

Schmoll ¹¹⁶ found that caseous material was free of soluble proteids or proteosis, and that what proteid it contains is almost solely pure coagulated proteid, related to fibrin and not to the nucleo-proteids. It is due to a coagulation of proteid with a dissolving out of its nuclear components, and the small amount of phosphorous in caseous material indicates that these latter products dissolve out rapidly from the caseous area.

Caseous tissue abounds in fat. Bossart ¹¹⁷ found that 13.7 to 19.4 per cent of dry caseous material was soluble in alcohol and ether, while Wells ¹¹⁸ gives 22.7 to 23.9 per cent as the amount from scrapings of bovine tubercular glands soluble in these fluids.

Of this soluble fatty material, cholesterin is given by Bossart ¹¹⁷ as 25 to 33 per cent, and Lecithin by Leber ¹¹⁹ at 28 to 31 per cent.

Sata ¹²⁰ states that lecithin and cholesterin are derived from disintegrated cells, but in sections of caseous areas most of the fat is found at the periphery, so that it most probably comes from here, and not from the caseous centre. Such is the view of Fichera and Gierke ¹²¹, who hold that glucogen and fat are produced by local impairment of oxidisation, and so are obtained at the margins of caseous areas, but that glycogen being labile disappears on account of the cell necrosis, while the fat remains. Another point is that the tubercle

bacillus consists of 40% of fat, but how far this influences the amount of fat in these areas is undecided.

According to Spietkoff¹²² the centre of pure caseous material contains no traces of albumen or peptone, but that they exist in minute quantities at the periphery, where undestroyed tissue elements are found.

Caseous areas are able to persist in the body for a long time without absorption, on account of the toxins of the tubercle bacillus having destroyed the autolytic ferments, and having produced caseation in place of heterolysis. Thus Schmoll¹¹⁶ found very little autolytic action in caseous areas. The tubercle bacillus itself is poor in proteolytic enzymes. Another factor which preserves the integrity of caseous areas for a considerable period is the absence of leucocytes, which are not kept off by the indigestibility of caseous tissue but by the absence of chemiotaxic substances, as is proved by the fact that when caseous tissue becomes infected with other bacteria, the leucocytes enter, attracted by chemiotaxic properties, and rapid disintegration follows.

4. Fibrosis and calcification.

In chronic phthisis, pari passu with the caseative change that is occurring in the tuberculous nodule, there is a proliferation of the fixed tissue cells around its periphery with the formation of fibrous tissue. It is by this proliferation that recovery may take place in phthisis even when caseation has occurred, for the nodule becomes encapsulated by an impermeable zone of fibrous tissue, so that it is shut off from the pulmonary tissues,

and may in time undergo calcification. It is obvious that the use of the term recovery in this connection is only relative, for although the disease be checked, so long as any living bacilli remain in the closed areas recovery is imperfect.

Spread of the Lesions.

The course of phthisis is a race between the processes of caseation and of fibrosis. If a small tubercular nodule forms in the lung, and is immediately and completely surrounded by fibrous tissue, there is an end of the tuberculous infection from that source, and it is this process which has occurred in the 90% of normal individuals, who are stated to show post mortem an old tuberculous nodule. In the less favourable event of caseation and fibrosis running a more equal race, with the advantage now to the one, now to the other, there results the clinical condition of chronic phthisis, and in those cases where the fibrous process is unable to hold caseation in check, there follows Rapid Phthisis.

When the primary nodule is not shut off by fibrous tissue, the tubercle bacilli proceed to cause an extension of the disease in the neighbouring pulmonary tissue, either by the caseous matter being aspirated into the surrounding bronchioles or alveoli, or by the bacilli finding their way into the interalveolar, interlobular, peribronchial, circumvascular, or subpleural lymph channels. In any case there follows an extension of the process of catarrh and proliferation over a wider area of the lung, and it is probable that this is the first pathological process

in phthisis which can be detected by the stethoscope.

The pathological changes in these secondary nodules are identical with those which occurred in the primary nodule. Consolidation is followed by caseation, and they are either shut off by fibrous tissue or they coalesce to form larger caseous areas. The pulmonary tissue between these nodules may remain for long unaltered. If the nodules become fibrous, however, the surrounding tissues are usually indurated, giving rise to patches of slate coloured tissue enclosing caseous nodules or nodes. Should the caseous process be advancing rapidly the inflammatory exudation affects the surrounding tissues, which become airless, consolidated, and caseous, so that eventually an entire lobe of the lung will become consolidated.

The caseous material may be aspirated into distant portions of the same or opposite lung, where it causes further patches of secondary broncho-pneumonia, which, according to the resistance of the tissues, the virulence and distribution of the bacilli, appear as small nodes or large irregular infiltrated areas. In these areas as in the others we find the same processes of proliferation, consolidation, caseation, and the production of fibrous tissue.

8. Calcification.

The fibrous tissue surrounding tubercular nodes and nodules in the lung may undergo calcification, which is the most effectual method whereby the bacilli are shut off from the lung, for calcified tissue presents a greater

resistance to caseation than does fibrous tissue.

Wells ¹²³ gives the following as the composition of inorganic salts in various calcified areas of the body.

Pathological Calcification	Mg ₃ (PO ₄) ₂	Ca CO ₃	Ca ₃ (PO ₄) ₂
Bovine tuberculosis	.84	12.8	85.9
" " " " " "	.9	13.1	85.4
" " " " " "	1.2	11.7	86.4
" " " softened glands	1.5	7.6	90.6
Human tuberculosis	1.2	10.1	87.8
Calcified nodule in thyroid	.85	13.4	85.4
Human Thrombus	1.1	11.9	86.5

Normal Ossification

Human bone (Zalesky)	1.04	12.8	83.8
" " (Carnot)	1.57	10.1	87.4
" " " "	1.75	9.2	87.8
Ox bone (Zalesky)	1.02		86.1
" " (Carnot)	1.53	11.9	85.7

Lung stones. Occasionally, however, the process of calcification is a cystic process, which by ulcerating into the pulmonary tissue may increase the enclosed area of the calcified areas, which may thus be loosened from the pulmonary tissue and expectorated, forming one variety of "lung stone." Stern ¹²⁴ has shown that calcified glands may also suppurate into the bronchi and form lung stones. Burgi ¹²⁵ was able to detect tubercle bacilli in decalcified lung stones.

(c) Young cavities, which are found where the tuberculous process is advancing, and are usually small,

Ott 126 gives the chemical composition of these lung stones as follows: -

(b) Old cavities. These are I large, and II usually

Calcium phosphate	52.0	72.6
Mag. "	- -	1.0
Mag. carbonate	2.0	-
Calcium "	13.0	6.0
Fat and cholesterolin	24.0	7.0
Other organic substances	4.0	10.0

to occupy an entire lobe of the lung.

5. Excavitation.

If the caseous nodes or nodules in the lung do not undergo calcification, they proceed to soften with a rapidity which depends on the nature of the surrounding tissue. If this be fibrous the softening process is slow, but if it be merely infiltrated with cellular elements the process is more rapid.

In any case the result is the production of a closed cavity or Vomica, filled with pulpy detritus of broken down lung tissue and pus-like matter. The wall of a cavity is a pyogenic membrane, which by ulcerating into the surrounding tissues may increase the enclosed area. Further by this ulcerative process the cavity may communicate with other cavities, with the pleural sac, or with a bronchus. Should the cavity communicate with a bronchus and with the pleural sac, pneumothorax follows.

There are three forms of Vomicae.

(a) Young cavities, which are found where the tuberculous process is advancing, and are usually small,

the walls being ill defined and composed of caseous and infiltrated tissue.

(b) Old cavities. These are large, and usually found at the apex in chronic phthisis. The contents are purulent, resembling the sputum, and are occasionally extremely foetid. The walls of such cavities consist of a pyogenic membrane, which are constantly secreting pus, and are joined by trabeculae, representing the remains of bronchi and blood vessels. These cavities slowly extend by the ulcerative process in their walls, and may come to occupy an entire lobe of the lung.

They may join with other cavities, or ulcerate into the pleural sac or into a bronchus, when the extension of their contents will cause a further development of the disease.

These cavities contain other bacteria along with the tubercle bacillus, especially micrococci and even mould fungi such as Aspergillina. Such bacteria are mostly saprophytes, but it is probable that the decompositions they induce act injuriously on the surrounding tissue, and assist in the extension of the cavity.

Haemorrhage from cavities. The arteries and veins of the lung, on account of their fibrous consistency, are the last structures to be affected by the ulcerative tubercular inflammation. Thus they often pass through a cavity even while the circulation is still going on. In the most fortunate event they become closed by an obliterating endocarditis, but should the ulcerative process attack the artery before this has occurred, there follows a weakening of the wall, with production of aneurysm.

aneurisms, and later rupture. This explains how the haemorrhage of late phthisis is so grave and serious a complication.

(c) Healed cavities. These are small cavities, lined by a smooth mucous membrane like wall, and surrounded by dense fibrous or calcareous tissue. A case of chronic phthisis may show every lesion which has been described in the preceding pages, from an excavated upper lobe, to a caseous solid middle lobe, and a congested catarrhal ~~lobe.~~ *lower lobe.*

THE PLEURA

Tuberculous pleurisy is the concomitant of every case of chronic phthisis, and usually occurs over those parts of the lung first affected by the disease. If seen early the surfaces of the pleura are covered by a thick creamy deposit, which is later replaced by fibrous tissue, so that the lung is bound down to the parietal pleura. In the thickened membrane, tuberculous nodules are also found. Serous, purulent, or haemorrhagic effusion also occurs, and pneumo- and pyo-thorax are frequent accidents.

BRONCHIECTASIS

Bronchiectasis frequently occurs in chronic phthisis, and has a dual causation. The walls of the bronchi may be weakened by the ulcerative process, and they are stretched by the contraction of fibrous tissue in other parts of the lung. The chest wall offering a concave surface to the strain, the bronchi offering a convex surface, it is obvious that the latter will be the first

to yield. (Hamilton ^{127.}) *Subacute*

D. The Pathology of Subacute Pulmonary Tuberculosis

Under chronic pulmonary tuberculosis, Ziegler ¹²⁸ describes the following pathological entity as a virulent form of the disease, which the writer holds is in accord with the clinical condition of Rapid Phthisis, first described by Trousseau.

"In other cases the disease is virulent from the outset, inasmuch as the tuberculous patches speedily become caseous and softened, with little tendency to the formation of firm fibrous tissue. Rapid disintegration of the affected parts is the natural result, cavities are soon produced, and the metastases that are formed tend in like manner to undergo caseous degeneration. The outcome is that in a very short space of time the lung is studded with caseous patches and riddled with cavities. The process might thus be termed caseative or ulcerative caseous tuberculosis; clinically, it is sometimes spoken of as phthisis florida. From its pathogenesis it might be called nodular caseous tuberculous broncho-pneumonia. The softening and disintegration of the pulmonary tissue are sometimes so extreme that the process almost resembles rapid suppuration.

In this form of tuberculosis the separate patches are usually larger than they are in the indurative form, and they readily coalesce to form still larger patches. Not uncommonly the infiltration spreads about the lesions over entire lobules or groups of contiguous lobules, which thus become at first greyish-red, then undergo grey

hepatisation, and finally turn yellowish, opaque, and caseous. In this way the nodular variety passes into caseous lobular broncho-pneumonia. By the coalescence of many such infiltrated lobules an entire lobe may become caseous, and the result is occasionally described as caseous lobar pneumonia."

E. The Pathology of Acute Pulmonary Tuberculosis.

(a) Miliary Tuberculosis.

(b) Acute tuberculous lobar pneumonia.

(c) Acute tuberculous broncho-pneumonia.

(a) Miliary Tuberculosis of the lung.

This is always a haematogenous infection, due to the presence in the blood stream of large numbers of tubercle bacilli which settle in the lung. The bacilli are scattered all over both lungs and pleura, and result in the appearance of minute grey tubercles of various shapes and sizes. As the bronchioles and alveoli become filled with the products of cellular proliferation, the minute grey tubercles become rounded off into solid nodules. Their appearance is accompanied by congestion and hyperaemia of the lung, which is dark red in colour and abnormally firm. The bronchi contain blood stained mucus.

(b) Acute Tuberculous lobar pneumonia.

This is the most severe form of acute pulmonary tuberculosis, and is pathologically a lobar pneumonia, due to the aspiration of tubercle bacilli into all portions of one or more lobes of the lung.

The lung is heavy, the affected portion being airless, and on section resembles ordinary hepatisation.

Osler ¹²⁹ writes "The most remarkable picture is presented by cases of this kind in which the disease lasts for some months. A lobe or an entire lung may be enlarged, firm, airless throughout, and converted into a dry, yellowish white cheesy substance."

On section these lungs show a homogeneous greyish white appearance, due to the aggregation of tubercles. At the apex may be found a small cavity from which the process originated.

Fraenkel and Troje ¹³⁰ suggest the large amount of exudative inflammation is due to some poison secreted by the bacilli.

(c) Acute Tuberculous broncho-pneumonia.

This is an acute broncho-pneumonia, due to the aspiration of tubercle bacilli from a caseous focus or in the blood of a tuberculous haemoptysis, into the alveoli and bronchi of different parts of the lungs. The affected areas are at first greyish red, but are later white, due to the occlusion of the bronchioles and alveoli with cheesy substance. The size of the tubercles vary according to the area involved, and are found from 1 to 3 c.m. in diameter, up to nodes the size of a marble.

These lesions are entirely due to the tubercle bacillus which has been shown by Pudden ¹³¹ to be not only capable of producing tuberculous nodules, but also exudative phenomena.

Osler ¹³² gives the following post mortem record of the disease in a child, aged 4 months.

"On section the right upper lobe is occupied with

caseous masses from 5 - 12 m.m. in diameter, separated from each other by an intervening tissue of deep red colour. The bronchi are filled with cheesy substance. The middle and lower lobes are studded with tubercles, many of which are becoming caseous. Toward the diaphragmatic surface of the lower lobe there is a small cavity the size of a marble. The left lung is more crepitant and uniformly studded with tubercles of all sizes, some as large as peas. The bronchial glands are very large, and one contains a tuberculous abscess."

CHAPTER IV.

ACUTE PULMONARY TUBERCULOSIS

There are three varieties of acute Pulmonary Tuberculosis, these being (1) Acute Miliary Tuberculosis of the lungs, (2) Acute Tuberculous Lobar pneumonia, and (3) Acute Tuberculous Broncho-pneumonia; the two latter constituting the clinical group of "Phthisis Florida" or "Galloping Consumption."

Clinically we may distinguish the various forms of pulmonary tuberculosis according to the length of their course; thus the varieties of acute pulmonary tuberculosis may kill in weeks, Rapid Phthisis may kill in months, and Chronic Phthisis in years.

1. Acute Miliary Tuberculosis of the Lungs.

This, the most acute form of pulmonary tuberculosis, is the result of a general infection of the blood stream by tubercle bacilli, which become arrested in the capillaries of the lung. Their primary source in the body may be a tuberculous bronchial gland, or some other focus of tuberculous infection.

The disease is frequently ushered in with fever, anorexia, malaise, and in children, convulsions.

The symptoms soon point to the respiratory system being involved, there being cough with muco-purulent expectoration, which is always swallowed in children. Holt recommends that it be secured by irritating the pharynx with a piece of gauze to excite coughing, when the sputum

will be found on the gauze. In a few cases haemoptysis was present.

From the start the most marked symptom is dyspnoea. The respirations increase to 60 or even 70 per minute. Inspiration is short, followed by a pause, and expiration is prolonged and difficult.

The dyspnoea is also manifest in cyanosis, the face being pale grey, the lips and ears cyanosed, the fingers and nails blue and cold. The Hippocratic facies soon develops, the nose "being sharp as any pen," while the nostrils dilate with every inspiration. With regard to the cyanosis, Osler ¹³³ remarks "Apart from emphysema and the later stages of severe pneumonia, I know of no other pulmonary condition in which the cyanosis is so marked."

Physical Signs. The temperature rises to 102 - 103°F. and is irregular, while the pulse is feeble and rapid.

The pulmonary signs are at first those of a diffuse bronchitis, changing later as the tubercles increase in size, to those of broncho-pneumonia, from which the disease is indistinguishable by auscultation. Percussion may reveal areas of impaired resonance, while auscultation will elicit areas of broncho-vesicular or bronchial breathing, with increased vocal resonance and fine crepitant rales. Jurgensen ¹³⁴ has noted fine pleural friction from the presence of tuberculous nodules on the pleura.

Diagnosis. The disease is diagnosed by the history of previous tuberculous infection in the body, by the cyanosis out of all proportion to the physical signs, and by the blood reactions of tuberculosis.

Course. Miliary tuberculosis of the lungs is invariably fatal, after an illness lasting from a week to a few months.

2. Acute Tuberculous Lobar Pneumonia.

This form of acute pulmonary tuberculosis occurs in those who have previously enjoyed good health, or have been known to be the subjects of chronic phthisis.

The disease sets in with all the symptoms of lobar pneumonia. There is chill, pain in the side, and a sharp rise of temperature. The breath sounds are at first feeble and suppressed, but with consolidation there comes bronchial breathing, dulness, increase of vocal fremitus and resonance. Dyspnoea may be extreme.

The temperature is not so continuous as in true pneumonia, and remissions of one to two degrees occur.

The sputum is rusty, and contains the tubercle bacillus.

The condition is almost invariably diagnosed as ordinary lobar pneumonia, until the absence of crisis in the second week, the aggravation of symptoms, and the night sweats, give rise to suspicions, which lead to the examination of the sputum.

The difficulties of diagnosis will be appreciated from the following case reported by Osler¹³⁵. "A healthy robust looking young Irishman, a cab-driver, who had been kept waiting on a cold blustering night until three in the morning, was seized the next afternoon with a violent chill, and the following day was admitted to my wards at the University Hospital, Philadelphia. He

was made the subject of a clinical lecture on the fifth day, when there was absent no single feature in history, symptoms, or physical signs of acute lobar pneumonia of the right upper lobe. It was not until ten days later, when bacilli were found in his expectoration, that we were aware of the true nature of the case.

It is probable that more of these cases would be early recognised if the examination of sputum for tubercle bacilli were made a routine measure in every pulmonary condition.

(3) Acute Tuberculous Broncho-pneumonia.

Acute tuberculous broncho-pneumonia is the most frequent form of acute pulmonary tuberculosis, and may occur either as a primary or a secondary affection.

AETiology. As a primary lesion it is more commonly found in children who are debilitated by measles or whooping cough, and in such cases has been said to supervene on the ordinary broncho-pneumonia, which may follow these fevers. It is extremely probable, however, that as the art of diagnosis advances, such cases will be found tuberculous from the onset, and that in true broncho-pneumonia, as in true lobar pneumonia, the disease does not take on secondary tuberculous characters. In adults it is occasionally seen as a primary affection, when debility has so weakened the resistance that latent phthisis or initial infection takes on this acute form. As a secondary affection it is seen in the course of ordinary phthisis either early or late, when, through some accident at the primary seat of infection, such as haemoptysis

or the bursting of a cavity, the bacilli are suddenly carried to all parts of the lung; the tuberculous Aspiration Pneumonia of Braümler.

Symptoms. The symptoms of acute Tuberculous Broncho-pneumonia vary according to whether the disease breaks out in a child, an adult, or in a phthisical patient, but in general the onset is that of an acute fever. Malaise, pains in the limbs, and chills may precede a sudden rise of temperature, reaching 102°F. or more, which marks the onset of the disease. With the fever there is increased frequency of the pulse and respirations, but at first the ratio of the two is not disturbed. A short dry explosive cough, tending to be paroxysmal, soon appears, and the patient may put up sputum with difficulty. As the disease progresses the cough and sputum become freer, and may be followed by vomiting. The pulse and respirations will be modified according to the amount of toxæmia and the extent of pulmonary tissue involved. Pain in the side is not an early symptom, but may occur later from involvement of the pleura. In a case seen by the writer diarrhoea was a prominent symptom, but here the disease followed the operation of gastro-enterostomy. Night sweats may occur.

Physical Signs. The first signs on auscultation are those of bronchitis, the breath sounds being feeble, and accompanied by moist and sonorous râles. A few days later these are replaced by broncho-vesicular breathing, with harsh inspiration, accompanied by occasional râles and increased vocal resonance over areas of the chest. Still later, as the disease advances and greater areas of

the lung become involved, it is possible to elicit dullness over the affected lobes, and areas of bronchial breathing, with moist and musical râles, and increased vocal resonance, will become apparent. The sputum contains tubercle bacilli, and large quantities of elastic tissue.

Fever. The disease sets in with a sharp rise of three or four degrees, after which it remains continuously high and irregular, there being no well marked remissions, and any that occur being usually less than a degree Fahrenheit (Fig. 1) It is not influenced by rest in bed.

Diagnosis. The diagnosis, especially in children who swallow the sputum, is often a matter of great difficulty, particularly to differentiate this disease from non-tuberculous broncho-pneumonia. In the main, being a tuberculous process, the disease is less acute than true broncho-pneumonia, and the temperature, pulse and respirations are comparatively lower. The crucial point, however, is the sputum, and probably more cases would be diagnosed in time were this examined as a routine proceeding in every case of pulmonary disease. In children the greatest tact and patience is necessary to succeed in obtaining even a small quantity of the sputum, and it may be necessary to search for particles in the vomited material. In adults a knowledge of previous phthisis, or the signs of extensive damage at the apices, will give considerable help in diagnosis. When available the methods of serum diagnosis should be applied to the case.

In one doubtful instance, the writer estimated the Opsonic Index to tubercle, which was normal on three occasions, thus indicating ordinary broncho-pneumonia. Later on a pyogenic pleural effusion endorsed this opinion. It is an easier matter to distinguish acute tuberculous broncho-pneumonia from an exacerbation of chronic phthisis - in the latter case the disease is more limited, the temperature more remittant, and usually falls with rest in bed.

Course. The disease usually runs a short, rapid, and fatal course, and has well earned the name of "phthisis florida" or "galloping consumption." In a few more favourable cases after a few weeks the condition abates, and the patient enters a state of chronic phthisis. In the child Osler¹³⁶ recognises three groups of the disease. In the first the child dies in a few days, in the second in a few weeks, while in the third after a few weeks, the case becomes one of chronic phthisis.

Treatment. This is of necessity mostly palliative and supporting. The patient should be kept in bed in a warm, well ventilated room (65° - 68°F.), wearing flannel next the skin, and in a blanket bed. A febrile mixture (Formulae I) may be given, and if the fever is high, tincture of aconite should be added, Should the cough be difficult and depressing, a stimulating expectorant mixture (Formulae II) is indicated, or in young children vomiting induced by Ipecacuanha wine. It is better to reserve alcohol until the first indication of heart failure, and then to prescribe generous doses. A nourishing liquid diet is required - milk and lime water, broths, albumen water, switched eggs, and Koumiss, these being

frequently given in small quantities, The symptoms of extremis must be alleviated as they arise.

AGE INCIDENCE OF ACUTE PULMONARY TUBERCULOSIS

The vast majority of cases occur in young children, and frequently under the age of three. Leroux ¹³⁷ gives the following age distribution of 219 cases in children under three: - 23 from one day to three months, 35 from three to six months, 53 from six to twelve months, and 108 from one to three years.

CHAPTER V.

SUB-ACUTE PULMONARY TUBERCULOSIS, - RAPID PHTHISIS

The term Rapid Phthisis was used by Trousseau to denote a form of pulmonary tuberculosis, which resembles chronic phthisis in its clinical and pathological aspects, but differs from it in the rapidity of its pace. Chronic phthisis kills in years, Rapid Phthisis kills in months. The condition is most frequently seen in those debilitated by malaria or by the ravages of secondary syphilis.

The following case illustrates the course of the disease.

CASE. A.G.B., male child aet. 6, a patient of Dr. Pedro Seras, Huelva, was first seen on 12th August 1907, complaining of pain in the chest, cough, and vomiting.

Family History. The child's parents are both alive and well, as are also the grandparents, and no history of tubercle could be elicited. Patient is the third child, the two previous children being both dead, - the first at one month, after three days' "gastric fever", the second at ten months from "bronchitis". The family are in fairly comfortable circumstances.

Patient's History.- Born weak, the patient was reared at the breast for the first two years. During his first six months of life he was thin and frequently vomited, but when 1 year old appeared to be a healthy

child. At the age of 1 he had "swollen throat", and when 2 years old suffered from "gastric fever". (Dr. Macdonald of Huelva informs us that this popular synonym is frequently tantamount to remittent malaria.) The patient's mother is quite certain that during these two years he had absolutely nothing but breast feeding. After this he was given "what was going" - the food mainly consisting of cow's and goat's milk, cow's and goat's flesh, bread, soups, fish, a variety of vegetables and fruits, large quantities of sugar, and a considerable amount of olive oil throughout the entire cuisine. At the age of 3 one of the anterior sterno-mastoid glands on the left side of the neck enlarged, and, when patient was 4, this was opened at the Provincial Hospital, Huelva and pus let out. Prior to the present illness the child slept well, but always, so the mother noted, with open eyes.

Patient got definitely ill in January 1907, but prior to this was feverish for some time. In January he had pain in the chest with slight cough, and in April 1907 he underwent an operation for pyothorax of the left pleural cavity, when a considerable quantity of pus was removed.

At date of present examination, 12th August, the patient complains of pain, cough, and vomiting. The facies is distinctly tuberculous. The features are well formed, the frontal eminences being unusually prominent. Of pale complexion and thoughtful expression

the patient has fine dark silky hair, growing well forward on the temples, large brown eyes overhung by long eyelashes, with eyebrows which tend to meet in the middle line. The pupils are equal, moderately dilated, while the sclerotics are clear and tinted blue. The child is thin, of poor muscular development, fretful and highly nervous. Height, 104 cms.; weight, 16.4 kilos. The cough is short, dry, explosive in character, and is worse at night. The patient coughs up a little sputum in the morning, which has never been noted to contain blood. He has pain all over the chest, especially over the right apex. The appetite is poor, vomiting immediately after food is frequent, and there is pain in the stomach after meals. The bowels are irregular, - constipation and diarrhoea alternately. He has great sweats at night, has lost weight rapidly, and his sleep is broken by dreams and nightmares. Pulse, 90; respiration, 27.

INSPECTION. - Thorax. - The chest is badly formed, flat, cone-shaped, and of greater antero-posterior dimensions below than above. The shoulders are sloping and supraclavicular hollows are present, increased on inspiration. Over the upper anterior portion of the chest a blue venous network is well marked, and over the 6th left rib in the anterior axillary line there is the scar of a 3-inch incision by which the pyothorax was tapped. This has the red-blue colour of a tuberculous scar, as has also the cicatrix on the left side of the neck. All the muscles show myotatic irritability, and myoidema is well marked on the pectorals and on the

spino-scapular muscles. Expansion is diminished at both apices and the breathing is mostly abdominal.

Mensuration	In expiration	Amount of expansion.
At level of nipples	57 cms.	0.4 cms.
At level of eighth rib	56.5 "	0.5 "
Abdomen at umbilicus	56 "	0.75 "

PALPATION.- When the child cries a marked increase in vocal fremitus is palpable over the entirety of the left lung, and over the upper lobe of the right.

PERCUSSION.- The percussion note is dulled all over the left lung, and over the upper lobe of the right both in front and behind.

AUSCULTATION.- Left lung: Breathing is high pitched bronchial in character over the entirety of the lung, accompanied here and there by moist rales on expiration. Vocal resonance is increased to aegophony.

Right lung. High pitched bronchial breathing over the upper lobe in front and behind. When the child breathes deeply, pleuritic friction is heard, soft in character. Over the middle and lower lobes the breath sounds are weak and feeble in quality.

Sputum. This contains tubercle bacilli and elastic fibres.

Heart. The apex-beat is diffuse, but best felt in the fifth intercostal space in the mid-clavicular line. The base and left border of the heart are normal, but the right heart is considerably dilated, the cardiac dulness extending 3 cms. from the right margin of the sternum, and on expiration the outline of this portion of the heart is apparent against the thoracic wall. On auscultation there is a loud reduplicated second sound, and a soft, blowing, systolic, tricuspid murmur.

Abdomen The abdomen is prominent, enlarged, and moderately firm. The umbilical sulcus lies flush with the skin, and the superficial abdominal veins are dilated. In their arrangement these latter show the early "cave-porte" type of Gilbert and Villaret ¹³⁸ to which reference will later be made. There is no pain on palpation, and no fluid can be detected. The liver is enlarged, extending one finger-breadth below the right costal margin. The outlines of the stomach are normal, but the mesentery can be felt as a thickened mass lying across the abdomen. No glands are palpable, and the spleen is not enlarged.

Urine. Sp. gr., 1022; acid reaction, and no abnormal constituents.

Treatment was palliative in view of the obviously hopeless prognosis.

September 13. For the last fortnight the patient has coughed up a considerable quantity of nummular sputum, has been fevered, and has coughed more. The

left lung is unaltered. Over the upper and middle lobes of the right lung there is high-pitched bronchial breathing, with loud sonorous rhonchi on expiration, and extremely loud pleuritic friction. Over the lower lobe, inspiration is harsh, and at the base of the lower lobe behind there is fine pleuritic friction giving the "bruit cuivre". Pulse, 120 - temperature $97^{\circ}.5$ Fahr.

September 14. Over the lower right lobes there are fine crepitations on inspiration. Pulse, 132; temperature, $97^{\circ}.4$ Fahr.

September 23. The condition of the lung is the same except for the cessation of pleuritic friction over the upper lobes of the right lung, and the appearance of medium moist rales in the lower lobe of the same lung. Pulse, 140; respirations, 48. Further, a swelling has appeared at the lower border of the right pectoralis major muscle and in the lower right quadrant of the mamma. It was noticed three days ago, is the size of a hen's egg, is tender on pressure, and apparently is an infected gland.

October 16. Swelling was opened 30th September by a "practicante" (1) and wound is now a discharging tubercular sinus.

If now we consider the abdomen we find it presents

(1) A "practicante" is a practitioner corresponding to the old apothecary.

a symptom complex of tuberculous lesions, which complicate the pulmonary condition. Apart from the vomiting and stomachic pain after food (possibly also a sign of the disease in the lungs), the child has no symptoms referable to the abdomen, yet nevertheless there are physical signs from which definite conclusions can be drawn. The abdomen is enlarged, tympanitic, but not tender, constipation is followed by offensive diarrhoea, and on palpation a thickened mesentery can be made out across the upper part, although no actual glands can be felt. Considerable light is thrown on the situation by a consideration of the arrangement of the dilated superficial veins, which show the early "cave-porte" type of Gilbert and Villaret¹³⁸. According to these authors, if there be a compression of the inferior vena cava alone, as occurs at the beginning of some forms of tubercle peritonitis, in limited abdominal tumours, and in hernias of the abdominal wall, the collateral circulation is always sub-umbilical and limited to the level of the iliac regions. As the disease spreads and the circulatory obstruction is increased, the dilated veins mount higher and invade the super-umbilical region, as is seen in the case of those veins which dilate in the case of obstruction in the course of the portal vein. The result is the mixed "cave-porte" type of collateral circulation most frequently seen in tuberculous or cancerous peritonitis. From the veins then, in conjunction with the other signs, we may deduce the existence of a lesion in the mesentery and in the peri-

toneum of the lower part of the abdomen - tabes mesenterica and chronic tuberculous peritonitis. In view of this the temperature which was taken twice daily for a week, is of considerable interest. No antifebrile or antipyretic drug was given, yet the temperature was subnormal, although the pulse remained persistently rapid. Considering the lesion in the lung, this is only to be explained by the nature of the abdominal condition, and according to Osler ¹³⁹, subnormal temperatures are common in chronic tuberculous peritonitis, the curious thing being, however, that in this case an "abdominal" temperature should take precedence of the ordinary "pulmonary" temperature.

REMARKS.- It is clear that in this case we are dealing with a grave form of pulmonary tuberculosis, which threatens to destroy life in the space of a few weeks. Were one to attempt the definition of the extent of the disease by an anatomical classification, as that of Turban, it would be recorded under "Stadium III"., - that is, severe disease affecting two or more lobes of the lung; but when one remembers that the patient got ill in January and that eight months later both lungs are infected in every lobe, the limitations of an anatomical classification as applied to a clinical pathological process are surely apparent. For, while this patient would be registered under "Stadium III" so also would be an old-standing case with vomicae in both lungs; and further, such a nomenclature is incompetent to express

the pathological process at work in different portions of the lungs, as here elicited by the physical signs - congestion, catarrh, and consolidation; nor can it give the faintest suggestion of the dire rapidity of the disease. If now we turn to the pathological nomenclature of pulmonary tuberculosis, according to Osler, the case is still difficult to class. Osler ¹⁴⁰ groups pulmonary tuberculosis under: "(1) Tuberculo-pneumonic phthisis - acute phthisis; (2) chronic ulcerative phthisis; and (3) fibroid phthisis." The second and third are at once ruled out of court, for the disease is neither chronic ulcerative phthisis, nor is it this condition with an acute tuberculous pneumonia superimposed. One is left with Group 1 "acute phthisis, galloping consumption, or phthisis florida" and in this group two types are recognised -

(a) Pneumonic form, whose onset resembles that of lobar pneumonia, and where death may occur in three weeks, or the case pass into one of chronic phthisis. It is pathologically a lobar pneumonia, due to the pneumatogenous distribution of tubercle bacilli.

(b) Acute tuberculous broncho-pneumonia, resembling catarrhal pneumonia, and where also death may occur in a few days or weeks, or the case become chronic. Pathologically it is a catarrhal pneumonia of tuberculous origin, the pneumatogenous miliary tuberculosis of Ziegler ¹⁴¹.

From this brief but sufficient summary of the classification as present in use, it would seem that a clinical

group is yet wanting for cases such as the present. We have the disease coming on in the left lung, rapidly extending by the lymphatics, as indicated by the tuberculous pleurisy and exudation, and now spreading throughout the entirety of the right lung. When first seen the upper lobe alone was solid, but a month later the middle lobe had undergone consolidation, its pleura was the seat of tuberculous inflammation, and the lower right lobe was also involved. In its totality we find that this case in nine months has traversed those stages through which chronic phthisis may pass, it may be, for years, and excepting the difference in time, it appears to the writer that the two present a striking similarity, both clinically and pathologically.

One may recall a similar case seen in the Liverpool Hospital for Consumption. The patient, a lad aet 15, under the care of Dr. John Owen, Hon. Assistant Physician to the hospital, had been ill for one year. He had attended the hospital at the beginning of his illness, but not since, and when seen was greatly emaciated, the lungs showing complete consolidation of all lobes, bronchial breathing, with moist rales and rhonchi all over the chest. He returned a week later, and was still at his work - that of chemist's assistant. Two things were remarkable in this case; the mental attitude of absolute unconcerned cheerfulness, and the absence of dyspnoea, illustrating the small amount of pulmonary tissue compatible with life. The next day patient's brother came to the hospital for a death certificate. From his

description death was due to syncope.

For cases such as these - virulent forms of pulmonary tuberculosis, running a course similar to that of chronic phthisis clinically, except for the destruction of life in a shorter time, and pathologically, barring the absence of fibrous tissue and attempts at repair - for such cases, not acute but rapid, which at present wander through the gamut of every classification the writer suggests the advisability of a return to the simpler clinical nomenclature of Trousseau who¹⁴² described these cases as "Rapid Phthisis". The dictum of the great clinician was as follows:

"With the exception of the rapidity of its pace, this form of the disease, to which we apply the term rapid, presents the same symptoms during life, and the same anatomical lesions after death, as ordinary phthisis the progress of which is generally chronic. It is the same disease as ordinary phthisis, though it generally runs its course with much more rapidity. There are also cases to which the term latent phthisis is given, because the symptoms remain obscure, and are marked by complications which are apt to lead us astray in our diagnosis. Nevertheless, whether the form be rapid or latent, regular or irregular, it is, I repeat, always the same disease. But acute, or galloping phthisis as it is more generally called, is not the same disease as ordinary phthisis."

are destined to play a great part. The individual symptoms of early phthisis may be caused by a variety

CHAPTER VI.

THE SYMPTOMS OF CHRONIC PULMONARY TUBERCULOSIS - PHTHISIS

PART I:- The Symptoms of Early Phthisis.

PART II:- The Symptoms of Late Phthisis.

THE EARLY DIAGNOSIS OF PHTHISIS.

There is no more important or practical problem in the whole range of clinical medicine than the early diagnosis of phthisis. The results of the scientific treatment of consumption have clearly demonstrated the fact that if the disease is to be cured, it must be taken in time. Further our knowledge of what constitutes the early stages of phthisis has undergone considerable change. Only a generation ago, it was customary for medical writers to regard bacilli in the sputum, altered breathing at one apex, and rapid loss of weight, as constituting the early stage of the disease. We know today that such a condition is indicative of far advanced disease, while the signs and symptoms of early phthisis are much more delicate. At the same time, it must be recognised that a diagnosis of phthisis is often made on an insufficient basis, to the great discomfort of the patient and his friends, and it is in these cases that the new methods of diagnosis by blood reactions (Chapter XII) are destined to play a great part. The individual symptoms of early phthisis may be caused by a variety

of other conditions, and it is only by a careful consideration of their history of onset, their relations to each other and to the organ involved, that a true deduction as to the presence or absence of the disease may be made.

Once the physician is convinced that he has presumptive evidence of the existence of early phthisis, especially if confirmed by some of the methods of serum diagnosis, treatment should be commenced immediately. It is only too often that one hears from a patient with consolidation of one apex that "a year ago a doctor said my lungs were delicate". Other expressions such as "not very strong" and "threatened with consumption" are likewise utterly unscientific, and imply a want of confidence in their own skill on the part of those who make them. If a patient is phthisical, he is phthisical, and the Art of Medicine is firstly the Art of Diagnosis.

In the early diagnosis of Phthisis we will consider:

- A. Constitution,
- B. The Mental State,

and the following symptoms, one or more of which may predominate in the onset of the disease;

1. Cough.
2. Bronchitis.
3. Dyspepsia.
4. Emaciation.
5. Sweats

6. Fever
7. Haemoptysis
8. Pleurisy
9. Lability of the Pulse.

A. CONSTITUTION

The erethic constitution is frequently seen in early phthisis. This type is intellectually keen and alert, capable of rapid deductions, and of considerable physical and mental effort, but is soon tired and fatigued. Such a condition is said to be due to the want of nervous and vaso-motor stability.

B. THE MENTAL STATE IN EARLY PHTHISIS

The leading psychological traits of early phthisis appear to be hyperexcitability, optimism, egoism, and sentimentality. Such patients worry little with the affairs of life, and when informed of their illness appear to be unable to grasp the situation. One of the most striking things to those who commence the study of phthisis is the easy way in which patients receive information as to their state of health. Berillon¹⁴³ considers that in the prodromal stages the will is deficient, the mind is restless, the patients are easily led and have little power of application. All of which traits constitutes a condition which he terms "Hypersuggestibility". This he states is followed by "Egotism", so that the patient may make a will, the

terms of which appease his vanity.

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Lepinay holds that long before symptoms appear there is nervous and sexual hyperexcitability which has a diagnostic value. He believes he has seen the same in cows and dogs, who showed depraved appetite and morbid sexual instinct.

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Regnault has pointed to the mental state due to phthisis by citing the case of a Myxoedematous patient who becomes phthisical. The apathy of the first disease ceases, and gives place to liveliness and great improvement of intelligence.

THE SYMPTOMS OF EARLY PHTHISIS

1. Cough.

This is the most frequent of the early symptoms of phthisis, and appears as a short, dry, cough, which is most often present in the morning after breakfast. At the start it is not accompanied by expectoration.

While it is present in the vast majority of cases, there is no symptom which patients will deny more strenuously than this. The reason is not far to seek. Cough is associated in the lay mind with consumption, and as humanity has a natural disinclination to face facts, a patient who readily admits other symptoms which point to phthisis will deny cough with the same assurance as other patients will deny venereal infection. A great deal naturally depends on the class of patient with which we are dealing, but where cough is too strenuously denied at the outset of the interrogation,

the matter should be pressed, which frequently results in the patient admitting to "a little cough in the morning."

While it is probably true that a tuberculous process may be proceeding in the lung in the absence of cough, such a condition is undoubtedly rare, and if, in a case where other symptoms are those of debility and dyspepsia, we are absolutely satisfied that there has been no cough, the probability is that phthisis is not the cause of the patient's ill-health.

2. Bronchitis.

This is frequently met with in the earliest stages of phthisis. The patient is very liable to colds, which as he expresses it "go to the chest", and linger as a sub-acute bronchitis for a longer or shorter period. Whether this bronchitis is in itself a tuberculous process, or whether it be due to the lowered resistance of the lung is still a disputed point, but it certainly differs in several important particulars from ordinary bronchitis. It is for one thing less acute, the rhonchi are fewer in number, more sonorous, and are less evenly distributed than they are in the ordinary type of this disease. That is to say they may be confined to the apices of the lungs. The cough too is different. It lacks the paroxysmal quality of the true bronchial cough, and is shorter, more frequent, and irritating. The bronchitis passed, the breath sounds at one apex may be found to be hoarse. Months may pass, and then the patient has another 'cold' and bronchitis.

If a patient be kept under observation, it will be found that the breath sounds become ~~harsher~~ after each succeeding attack.

3. Dyspepsia.

There is a difference of opinion as to the part played by dyspepsia in the early stages of phthisis. Thus Lindsay ¹⁴⁶ writes: "The voluble dyspeptic, who complains loudly of many miseries, but retains a fair degree of nutrition (the type, I may say without irreverence, immortalised in the letters of Thomas Carlyle) seems to me in no greater danger of tuberculosis than the healthy man."

Undoubtedly, this is so, but "the voluble dyspeptic" to use this happy description, is not the dyspeptic met with in early phthisis, where, however, we frequently find the nervous dyspeptic.

The dyspepsia of early phthisis is characterised by irritation of the stomach, only occasionally accompanied by vomiting, but usually marked by a feeling of discomfort and hyperacidity after meals. It is most usually present in the morning, and after breakfast these patients have acid eructations, and the phenomena commonly known as "hawking". Saundby ¹⁴⁷ found that in these cases the stomach contents showed an excess of hydrochloric acid.

4. Emaciation.

The wasting in phthisis, as has been shown by experimental work to which reference will later be

made, is dependent on fever. Thus a patient with early phthisis but no fever will not show emaciation. As no attention is paid to fever by patients in the early stages, loss of weight has thus a dual significance. The loss of weight in early phthisis is usually well-marked, and if not explained by malignant disease, pernicious anaemia, syphilis, bone disease, or suppuration it is strong presumptive evidence of phthisis. Generally ~~however~~, loss of weight in a young man points to phthisis, in an old man to cancer.

5. Sweats.

Like wasting, sweats are dependent on fever, and although not usually given as an early symptom of phthisis the writer believes that they are present earlier and more frequently than haemoptysis. If they occur in the afternoon they are apt to be overlooked, and if in conjunction with chill and fever, they are often confused in subtropical climates with malarial fever. In the early stages they are much less severe than in the later, and all the patient may notice is a slight perspiration soon after turning in at night.

6. Fever.

In every stage of phthisis the progress of the lesions in the lung are associated with fever. In the early stages this is intermittent, and may only consist of an afternoon or evening rise of one degree. Exercise will also bring it on at any time, as the

increased circulation carries the toxins to the heat controlling centres. As was stated with regard to sweats this intermittent fever may be mistaken for malaria, and according to Osler¹⁴⁷ scores of cases of early phthisis are treated for ague in Philadelphia and Baltimore. In early phthisis the rise of temperature is not nearly so high as in malaria. There is one disease which must always be excluded in young males, when an intermittent temperature is associated with emaciation, before phthisis is diagnosed, and that is syphilis.

7. Haemoptysis.

The frequency of haemoptysis as an early symptom of phthisis has been differently stated by various authorities. According to Laennac¹⁴⁸ "In the majority of phthisical patients the first symptom indicating cause for anxiety and supervision of the patient is a haemoptysis."

Walshe¹⁴⁹ puts it as the first symptom in 36 per cent of cases, but Turban¹⁵⁰ only found it to be the initial symptom in 47 among 408 cases. It is probable that in the great majority of cases, haemoptysis is preceded by quite recognisable symptoms.

The haemoptysis of early phthisis differs from the haemoptysis of later phthisis in every point. It is small in amount, and is never dangerous to life. The amount varies from a teaspoonful to a teacupful, put up during 24 hours. The haemorrhage comes on suddenly without any warning. The patient notices a sickly feeling in the larynx, a salt warm taste in the mouth, and spits out blood, and this bleeding is apt to recur,

the main point being that the patient spits blood, or blood-stained sputa, for at least twenty-four hours. After such a bleeding, the sputa may or may not contain tubercle bacilli, and the examination of the chest may be positive or negative.

In one case the writer found tubercle bacilli in the leucocytes of the expectorated blood, where auscultation had revealed no signs of phthisis.

The haemorrhage of early phthisis is due to the bursting of capillaries in the zone of congestion, proliferation, and ulceration. Being a capillary haemorrhage it has not the dangers of the arterial haemorrhage of later disease. Blood from the lungs is to be distinguished from blood from the nose or the stomach. Blood of pulmonary origin is red or dark red in colour, and frothy from admixture with air. It is alkaline. In haematemesis the blood is dark, like coffee grounds, having been acted on by the gastric juices, and further in this condition the patient has actual nausea, not merely a sickly feeling. The reaction is acid. Blood from the nose is dark red in colour, and is not mixed with air. It is usually in young children.

The benefit of early haemoptysis is that it calls the patients attention to his disease, so that he seeks medical advice.

The evils of early haemoptysis are that a certain amount of the blood containing tubercle bacilli is aspirated into other lobules of the lung, where it may

cause an extension of the disease; and so a slow and very chronic case of phthisis may be changed into one of acute phthisis.

Patients must be closely questioned with regard to haemoptysis. The shock caused to many will lead them to admit it readily enough, but there are others who are only too willing to put it down to other causes, such as blood from the throat and nose. At the same time a patient who has had haemoptysis may in good faith ascribe it to haematemesis, because an actual vomiting, such as sea-sickness, may have induced the pulmonary haemorrhage.

The various other diseases which may produce haemoptysis will be dealt with later in this chapter, but it may be stated that if no other cause can be found for the bleeding, and if there is evidence that it has come from the lung, it should be regarded as strong evidence of tuberculosis, and the patient should be informed of this. To tell a patient that he has "strained his lung" is to tell him a falsehood.

8. Pleurisy.

Pain at the apex from pleurisy is a frequent symptom of early phthisis, and is the result of tuberculous disease of the pleura. There is a growing belief among the profession that a majority of so-called simple pleurisies are tuberculous. Thus Bowditch¹⁵¹ found that of 90 cases of pleurisy with effusion, 30 later developed pulmonary tuberculosis.

Osler¹⁵² states "I confess that the more care-

fully I have studied the question, the larger does the proportion appear to be of primary pleurisies of tuberculous origin."

9. Lability of the Pulse.

This condition is probably due to weakness, but is an early symptom of phthisis. It is best elicited as follows. The pulse rate having been taken after putting the patient at his ease, ask a few questions as to cough or haemoptysis, after which again take the pulse rate, when, if lability be present, it will be found to have increased 20 beats or more per minute.

Of the earliest symptoms, cough, fever, dyspepsia, and loss of weight are the most important, and in conjunction constitute a strong probability of pulmonary tuberculosis.

PART II: THE SYMPTOMS OF LATER PHTHISIS.

In this we will first consider:

A. The Mental state in later phthisis, and the symptoms in the following order.

1. Cough.
2. Emaciation.
3. Dyspepsia.
4. Sweats.
5. Fever.
6. Haemoptysis.

A. The Mental state in later phthisis.

In this condition the mental state is one of extreme

optimism, which has well earned the name of "spes phthisica", for apparently it increases as the disease advances. The writer has seen cases on the verge of the grave, where both lungs were infected in every lobe, in which the patients were making most extensive plans for the future. This extraordinary feeling of well being may lead the patients into habits most prejudicial to his condition, such as sexual excesses, gambling and drinking.

1. Cough.

The cough of later phthisis is very different from that of the early disease. It is looser, more constant, and parosysmal, and is accompanied by muco-purulent expectoration. It is usually most marked in the morning, yet may be so persistent after meals as to induce vomiting. If the disease is advancing the coughing may be so distressing at night as to prevent sleep. On the other hand, during the quiescent periods of phthisis, the cough is diminished or absent.

2. Emaciation.

This is one of the most marked and obvious symptoms of later phthisis, the patient being, thin, wan, and haggard, literally worn to a shadow. The skin is loose, dry and wrinkled from the loss of sub-cutaneous fat, and the bones throughout the body stand out prominently. The wasting of phthisis is due to the metabolism being altered by the fever. Vannini and Collini ¹⁵³ have shown that when fever is present, there is usually a steady

loss of nitrogen from the body. The urine contains more nitrogen as NH_3 , and less as urea than is normally the case, while the nitrogen as uric acid remains unchanged.

3. Dyspepsia.

The dyspepsia of later phthisis is an atonic dyspepsia, with deficiency of hydrochloric acid. When the temperature is normal there may be little or no gastric disturbance, but when fever ensues, particularly ~~the~~ hectic fever, the gastric disturbance which follows is very great, and one sees in such patients the symptoms of gastritis, in all stages. Most serious of all is chronic gastritis, so often seen in the last stages of phthisis, for it interferes with the alimentation of the whole body, and makes the nourishment of these patients a matter of great difficulty. The stomach condition is further aggravated by the cough, which after meals is a frequent cause of vomiting. Bourget 154 considers that it appears "as if at this moment there was an increase of congestion around the tuberculous nodules". In fact it is probable that the syndrome of phthisical vomiting is as follows - the food on an irritated stomach induces a reflex congestion of the pulmonary vessels, which heightens the sensibility of the damaged areas so that coughing is induced, which in its turn induces a reflex vomiting. Should such be the case, it makes the treatment of the gastric condition one of the primary indications in the treatment of the disease.

4. Sweats.

Drenching perspirations are frequent in later phthisis. They usually occur when the temperature is falling in the early hours of the morning, and are a source of great weakness to the patient.

5. Fever.

Fever is not always present in chronic phthisis, for if the lesions be latent there is no temperature, but it is the invariable accompaniment of active lesions. As we have seen in the early stages of the disease the fever is usually intermittent, and this is also commonly seen in later chronic phthisis, but when the lesions are very active, and the disease is undergoing an exacerbation, the fever tends to be remittent. This is explainable by the fact that all stages of pathological changes are occurring in the lungs, so that the autointoxication is more constant.

Fig. 2. shows the intermittent temperature of chronic phthisis.

The maximum rise of temperature usually occurs between 5 and 7 p.m., and the lowest point reached is between 3 and 5 a.m., "when the vital tide has reached its lowest ebb."

Cause of late haemoptysis.

6. Haemoptysis.

The cause of haemoptysis in later phthisis is the ulceration of the walls of a blood vessel in a cavity, or in a caseous area, so that the vessel becomes aneurismal, and eventually bursts.

Exciting Causes.

Haemoptysis is usually brought on by physical exertion or by a fit of coughing, after which the patient has a sickly feeling in the larynx, and proceeds to cough up blood, the amount of which varies from a few spoonfuls to several pints. If the bleeding takes place into a large closed cavity, the patient may die of haemorrhage without coughing up blood.

Dangers Immediate and Remote.

The immediate danger of haemoptysis is that the patient will die from shock, syncope, or suffocation. The latter is a frequent cause of death, and requires prompt and energetic treatment. If the bleeding be profuse, the patient may fall dead from syncope. Among the remote effects of haemoptysis, there is the weakness due to anaemia, while the inspired blood has carried the bacilli to different parts of the lung, where they light up fresh fever of infection.

Frequency of haemoptysis in phthisis.

155
 Fox found haemoptysis in 55.7 per cent, 157
 : 156
 Turban 57.6 per cent, Walshe in 80 per cent,
 158
 while Osler finds it occurs in 60 - 80 per cent of
 159
 all cases of tuberculosis. Stricker finds that 80 per
 cent of all cases of haemoptysis are associated with
 tuberculosis.

Age in relation to Haemoptysis.

Haemoptysis is rare under twenty, and in phthisis appearing at the decline of life. It is also rare in the acute forms of pulmonary tuberculosis. Some writers have described a particular type of "Haemorrhagic phthisis" where there are profuse bleedings at long intervals, in which cases the prognosis is said to be relatively good. While such cases undoubtedly do occur, they form so minute a portion of all cases of phthisis, there is hardly a justification for placing them in a separate class.

Haemoptysis or Haematemesis?

These two conditions may be confused in the following circumstances; the patient may believe the blood to have come from the stomach, when the act of vomiting has induced haemoptysis, or the blood from the lungs may be swallowed and later vomited, or vomited blood may be drawn into the lungs.

Blood from the stomach is dark and tarry in appearance, from the action of the gastric juices, is acid in reaction, and is mixed with the stomach contents. It is not preceded by cough, the haemorrhage ceases at once, and there is a history of gastric symptoms without fever. Haematemesis is got in young anaemic women with gastric ulcer.

In haemoptysis the blood is often bright red, frothy from admixture with air, is alkaline in reaction

and mixed with mucous or saliva. There is usually a history of cough.

Non tuberculous causes of Haemoptysis.

1. From upper air passages.
2. Hysteria
3. Malingering
4. Cardiac disease
5. Purpura
6. Haemophilia
7. Leucocythaemia
8. Malignant Small Pox.

Very rare causes:

1. Actinomycosis of the Lung.
2. Asthma
3. Aortic aneurism.
4. Bronchiectasis
5. Carcinoma of the lung
6. Emphysema
7. Hydatids of the lung
8. Plastic bronchitis
9. Distomum pulmonate of Japan
10. Vicarious menstruation.

1. Blood from the upper air passages is comparatively rare, but should this explanation be given search should be made in the nose and pharynx, for congested ulcerated areas. If none are found, it is most unlikely that the blood is from this source. Advanced laryngeal ulceration will give rise to haemorrhage, but

in this condition the voice and history will indicate the source of the mischief. It is not to be forgotten, however, that phthisis may commence secondarily to tuberculosis of the larynx.

2. Hysterical girls will suck their gums to gain sympathy by showing haemoptysis.

3. Malingerers will simulate haemoptysis in many ways. The writer saw a case where a malingering steward terrified the captain and officers of a ship by showing his tongue to be covered with bright red blood. After a long examination, he admitted the blood had come from a fowl killed that morning.

4. Haemoptysis occurs in cases of mitral stenosis *or* ~~and~~ regurgitation. It is due to the increased backward pressure on the lungs, and is got when compensation is failing. In these cases the symptoms of mitral disease are too apparent to be overlooked, and the long history of shortness of breath always points to the heart. The amount of blood is small, and the patient may feel relieved. *Turgid veins will be seen on the pharynx.*

When the bleeding is due to purpura, haemophilia, leucocythaemia, or to malignant small pox, the primary disease is nearly always apparent.

In the Chapter on Differential Diagnosis, reference will be made to some of the rarer causes of haemoptysis, which may simulate phthisis (Chapter IX).

In the history of a case of phthisis the writer uses the following form of chart:

CHART

Name:

Residence.

Age , Occupation ,

Date.

1. Patient complains of
2. Family history
3. Patient's previous illnesses
4. When was patient last well
5. Cough
6. Expectoration
7. Haemoptysis Date
8. Pain Situation
9. Appetite
10. Digestion
11. Vomiting
12. Bowels
12. Weight, any loss of
14. Palpitation
15. Able to work
16. Menstruation
17. Sleep
18. Sweats
19. Pulse
20. Respiration
21. Temperature

CHAPTER VII

- 22. Weight
- 23. Bacilli in Sputum
- 24. Stadium (Turban)
- 25. Stadium Pathological

Diagnosis

Complications.



CHAPTER VII.

THE SIGNS OF PHTHISIS.

PART I. A. The Surface Anatomy of the Lungs.

B. Method of Examining the Chest.

PART II. The Signs of Early Phthisis.

PART III. The Signs of Later Phthisis.

PART I.

A. The Surface Anatomy of the Lungs.

In the examination of the chest it is essential to have an accurate idea of those areas of the thorax which correspond not only to the lung, but also to the pleural sacs, for it is these which limit the expansion of the lungs, and in diseased conditions of the latter it is of primary importance to detect a deficiency of expansion. The relations of the pleurae to the lungs in expiration are shown in Figs. III and IV.

THE PLEURAL SACS

With the arms by the side the pleural sacs extend into the supraclavicular areas, rising about 2 inches above the clavicle, and lying under cover of the clavicular head of the sterno-cleido-mastoid muscle.

From this point the anterior border of the pleural sacs runs downwards and inwards to meet slightly to the left of the middle line behind the sternum at the level of the angle of Ludwig. From this point they pass vertically downwards parallel to each other as far as the level of the fourth chondro-sternal articulation.

The right sac continues downwards in this straight

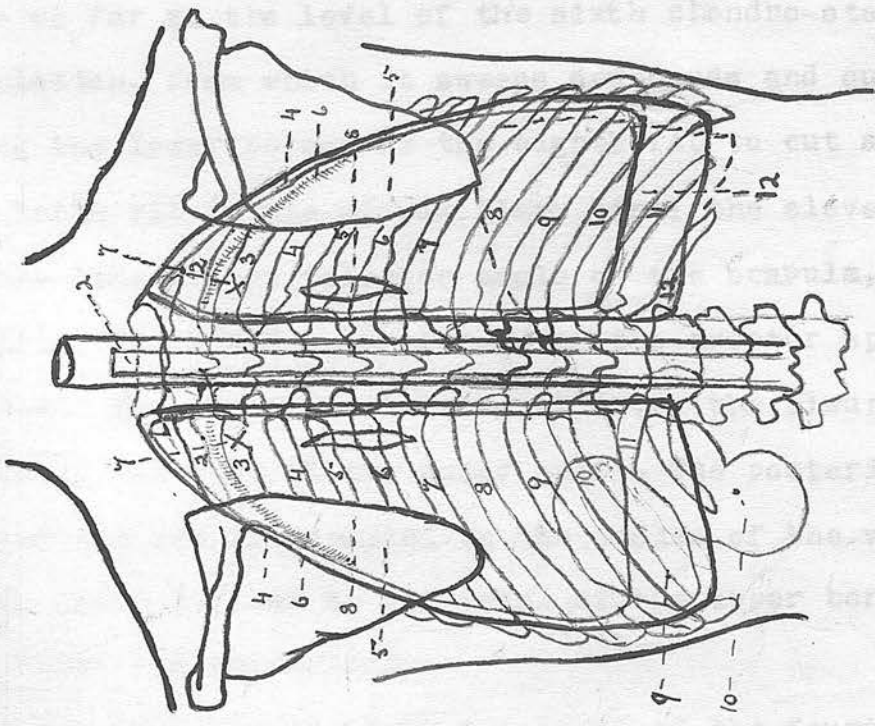


Fig. IV.

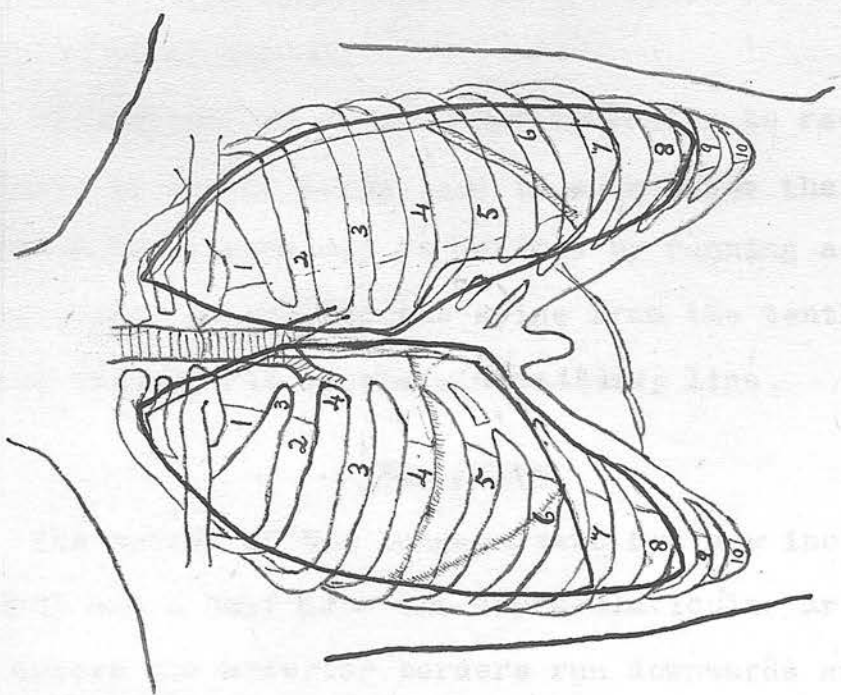


Fig. III.

line as far as the level of the sixth chondro-sternal articulation, from which it sweeps downwards and outwards along the lower border of the eighth rib to cut across the tenth rib in the mid-axillary line; the eleventh rib in the line of the inferior angle of the scapula, and the twelfth rib at the outer border of the erector spinae muscle. The twelfth rib being oblique, the pleura is actually below it in its inner half. The posterior border of the sac is parallel to the bodies of the vertebrae, along which it runs to the level of the upper border of the first rib posteriorly.

The left pleura, from the level of the fourth left chondro-sternal articulation is deflected obliquely outwards by the pericardiac sac, and passes downwards and outwards behind the fifth, sixth and seventh costal cartilages, after which it follows the lower border of the eighth rib to cut the tenth rib in the mid axillary line, the eleventh rib in the line of the inferior angle of the scapula, and the twelfth rib at the outer border of the erector spinae muscle.

In practice it is a difficult matter to recognise the twelfth dorsal spine, and in such cases the posterior limit of the pleura may be defined by running a line transversely inwards to the spine from the tenth, that is the lowest, rib in the mid axillary line.

THE LUNGS.

The apices of the lungs extend for one inch or an inch and a half into the supra-clavicular area. From the apices the anterior borders run downwards and in-

wards behind the sterno-clavicular joints towards the angle of Ludwig.

The anterior border of the right lung passes vertically downwards in the middle line to the level of the sixth chondro-sternal articulation, from which it sweeps downwards and outwards along the lower border of the sixth rib to reach the eighth rib in the mid axillary line, and the tenth rib in the line of the inferior angle of the scapula, from which point it runs transversely inwards towards the tenth dorsal spine.

From the angle of Ludwig the anterior border of the left lung runs down behind the left border of the sternum as far as the level of the fourth costal cartilage, where it is deflected outwards in a concave manner along the border of the fourth costal cartilage, then curving down towards the sixth costal cartilage, from where it sweeps downwards and outwards along the lower border of the seventh rib, to reach the eighth rib in the mid axillary line, the tenth rib in the line of the inferior angle of the scapula, and then runs transversely inwards like the right lung, but on a slightly lower level.

Next the spine the right lung is at the upper border of the eleventh rib, while the left lung is at the lower border of the same rib.

The most important points may be remembered as follows:

	<u>Lung</u>	<u>Pleura</u>
Mid axillary line	8th rib	10th rib
Scapular line	10th rib	11th rib.
Next spine.	11th rib	12th rib

FISSURES OF THE LUNGS

The main fissure of each lung starts at the level of the second dorsal spine posteriorly, so that this point corresponds to the apex of the lower lobe on each side. The fissure runs downwards and outwards, with its convex surface upwards, to cross the fifth rib in the mid-axillary line, and to terminate at the inferior border of the lung at the level of the sixth chondro-costal junction. When the hand is placed on the opposite shoulder the internal border of the scapula corresponds to the line of the fissure, to which it is a useful guide. When we auscultate behind, we are listening to the breathing mostly over the lower lobe, and in front mostly over the upper lobe.

On the right side the lower part of the upper lobe is cut off to form a middle lobe by a smaller fissure which leaves the main fissure in the mid axillary line at the level of the fifth rib, and runs transversely inwards along the lower border of the fourth rib. The middle right lobe is thus in relation to the anterior surface of the chest.

The roots of the lungs lie opposite the spines of the fourth, fifth and sixth dorsal vertebrae.

B. Method of examining the chest.

Position of the Patient.

The patient should face a good light in a quiet room. If his strength permits it, he should stand rather than sit, as the latter adds to the difficulties of examination. The patient should be stripped to the waist, those

portions of the chest not engaging our attention being covered with a blanket. The first difficulty is to induce the patient to breathe easily and naturally, and a good deal of patience is at times necessary, as patients will breathe artificially with vocal accompaniments, which are particularly trying.

We now proceed to

1. Inspection and Mensuration.
2. Percussion.
3. Palpation.
4. Auscultation.

From Inspection and Mensuration we learn -

- A. The circumference of the Chest.
- B. Condition of the Integuments,
- C. General Form of the Chest.
- D. Movements of the Chest.
 - a. Respirations
 - b. Type of Respirations.
 - c. Local movements.
 - d. Deficient and delayed expansion.

A. By taking the measurement of the chest round the nipples we gain a rough idea of the capacity of the chest, which is diminished in phthisis. The chest measurement of a man 5 ft. 6 inches should be about 34-35 inches, and deep inspiration should give an increase of $1\frac{1}{2}$ to 2 inches. The "vital capacity" of the chest, the amount of air that can be forcibly expired, is registered by the Spirometer. With the Cyrtometer, we measure the relative

size and shape of the two sides of the chest.

The following table (Saville 160) shows the normal size and capacity of the chest.

ADULTS (AT 30)			CHILDREN		
Height	Average chest Measurement	Chest capacity (Spirometer)	Age	Average height	Average chest measurement
Ft. Inch.	Inches	Cubic inches		Ft. inch.	Inches
5 1	34	175	10	4 5 $\frac{1}{2}$	25 $\frac{1}{4}$
5 2	35.1	177	11	4 7	26 $\frac{1}{4}$
5 3	35.7	189	12	4 8 $\frac{3}{4}$	27 $\frac{1}{2}$
5 4	36.2	195 $\frac{1}{2}$	13	4 10 $\frac{1}{2}$	28 $\frac{1}{2}$
5 5	36.8	203 $\frac{1}{2}$	14	5 0 $\frac{3}{4}$	29 $\frac{1}{2}$
5 6	37.5	214	15	5 3	30 $\frac{3}{4}$
5 7	38.1	225 $\frac{1}{2}$	16	5 5	32 $\frac{1}{2}$
5 8	38.5	229	17	5 7	34 $\frac{1}{4}$
5 9	39.1	238 $\frac{1}{2}$	18	5 8	35 $\frac{1}{2}$
5 10	39.6	246			
5 11	40.2	250 $\frac{1}{2}$			
6 0	40.8	260 $\frac{1}{2}$			
over 6 ft.	41.0	276			

B. In the condition of the integuments we look for oedema, dilated veins, myxoedema, and emaciation,

C. General Form of the Chest.

We look for three main types of chest; the alar tubercular chest, the pigeon rickety chest, and the barrel shaped emphysematous chest.

D. In movements of the chest the respiration rate is to be noted, normally 15 - 20 per minute or one fourth

of the pulse, and whether the type of respiration be thoracic, abdominal, or thoracico-abdominal. As to local movements, we note drawing in of intercostal spaces, flattening, immobility, or bulging at the apices. Further the apices are to be examined for delayed expansion, or for action of the extraordinary muscles of respiration.

2. Percussion.

This is the most difficult yet most valuable method of chest examination. The first finger of the left hand must be laid firmly on the chest, and is struck in a sharp incisive manner with one, two, or three fingers of the right hand, according to the pitch of the note desired. Two points are essential to good percussion - the blow must come from the wrist, and be delivered through the pulp of the fingers, If the very tips be used, a "bony" note is got from the bones of the fingers.

Skoda ¹⁶¹ was the first to show what percussion means; "The different sounds which percussion produces over the regions of the liver, spleen, heart, lungs, and stomach do not depend upon any peculiarities of these organs, but upon variations in the quantity, distribution, and tension of the air present in these regions in which they lie, and upon the force of the percussion stroke." He also showed that the percussion note varies in resonance and in pitch, "The sound is a tone, clear or muffled, even to complete privation; this is the first and great distinction. And next, the tone is of a pitch higher or lower. Upon these two hang the whole theory and practice of percussion." ¹⁶² We can alter the pitch of the note,

but we cannot alter its tone.

The note given by the percussion of healthy lung tissue is called resonant.

Hyper-resonance is usually got in emphysema, and is slight or marked. If it be very marked it is called "Tympanitis", mostly found in pneumothorax.

Deficient resonance is due to thickening of the pleura, or consolidation of lung tissue, or to effusions in the pleural sacs, and is -

- a. slight dulness.
- b. marked dulness.
- c. absolute dulness.

Special notes.

1. Boxy note. This is a mixture of dulness and resonance, and is got over a cavity.

2. Cracked-pot sound. This is got over large cavities communicating with a bronchus, and is got by percussing while the patient's mouth is open. It is a resonant note which varies in pitch, and has a metallic quality, like that got by striking a cracked pot. (Bruit de pot fêlé)

3. The Bell sound is got in pneumothorax by auscultating, while one coin is struck against another over a distant part of the same side.

Percussion is thus of value in determining pathological changes in the lung, but a new application of this method has been introduced by Dr R. W. Philip¹⁶³ for determining the functional activity of the lung, which the writer has already found to be of great service in the early diagnosis of phthisis.

PHILIP'S METHOD OF "TIDAL PERCUSSION"

The following is the originator's description of the method. "It consists essentially in the practice of percussion alternately during expiration and full inspiration. For early diagnosis, this is to be carried out, particularly at the apices. It is no less applicable, however, to the bases and the anterior edges. "Take any healthy subject, and, on careful application of the test, you will find that the difference between the degree and limits of supraclavicular resonance in expiration, and in full inspiration, is considerable. The accompanying drawing (Fig. 1) reproduced from one of the patients, illustrates the point. The lower line indicates the limit of lung resonance during expiration, and the upper line the limit during full inspiration. These limits are subject to considerable modification in various physiological and pathological states. Thus the sedentary individual, with relatively lazy lungs, offers, on percussion, relatively slight tidal increase of resonance. In his case, the deficiency is probably equal at both apices. In early tuberculous infiltration, - it may be before actual percussion dulness is determinable, - a limitation of the tidal difference at one or other apex may be noticed. As consolidation advances, even in slight degree, limitation of the tidal change becomes more marked. In cases of doubtful diagnosis, the evidence afforded by tidal percussion is of much value. Fig. 2 shows the amount of tidal change to be sufficient on the left side, while on the right side it is much below the normal. In Fig. 3 the tidal change on the right side is fair, while on the

left side it is nil (the total extent of supra-clavicular resonance being actually reduced.)

What is true as to the value of tidal percussion at the apex is equally true - and still more evident - in relation to the bases (anteriorly, posteriorly, and laterally.)

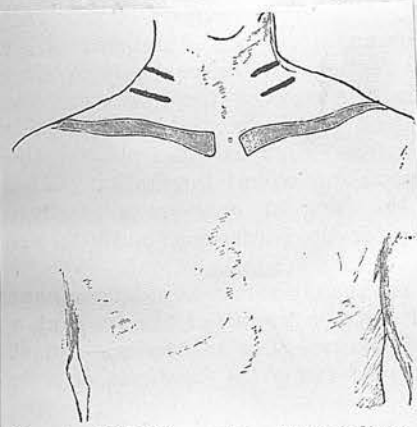


FIG. 1.—Tidal Percussion. The two lines—upper and lower—in the supraclavicular area indicate the limits of lung resonance during full inspiration and full expiration respectively.

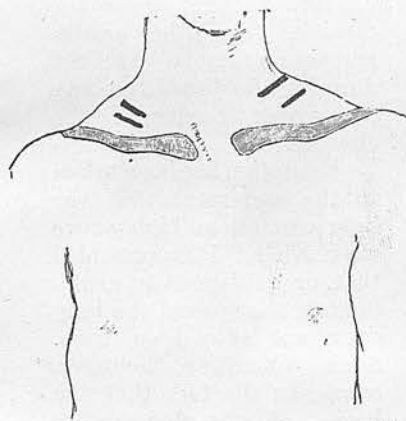


FIG. 2.—Tidal Percussion. Amount of tidal change is sufficient on the left side; it is much below the normal on the right side.

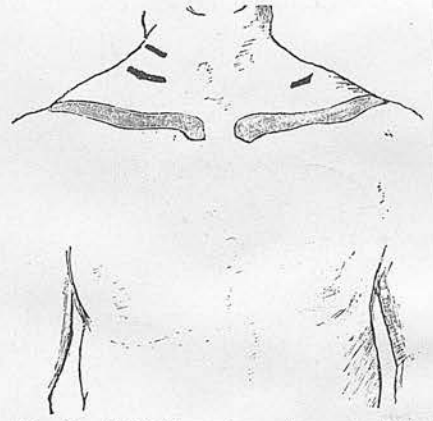


FIG. 3.—Tidal Percussion. Amount of tidal change is fair on the right side; it is *nil* on the left side.

The procedure is as follows: - "The index finger of the left hand is used as pleximeter, and is placed horizontally across the apex of the lung from back to front in a plane at right angles to the vertical diameter of the lung. Percussion is practised with the middle finger of the right hand in a direction at right angles to the pleximeter. The pleximeter finger should be closely apposed, and percussion should be firm and sharp, but not too strong."

4. Auscultation.

By auscultation we determine the presence or absence

















of pathological lesions of the lung by (1) the character of the breath sounds, (2) their accompaniments, and (3) by alterations in vocal resonance.

It is most unfortunate that there is no standard in the nomenclature of auscultation, which is particularly evident when one is dealing with foreign writings. As the writer will use in the following pages the terms which are current in the Edinburgh School, as they are stated by Professor Wythe, and as a paraphrase would only detract from the incisiveness of his teaching, the latter is quoted here verbatim. 164

"AUSCULTATION.

"Listen for (1) Type of Breathing, (2) Accompaniments, (3) Vocal Resonance.

1. TYPES OF BREATHING

(1) Vesicular or Rushing						
	<u>Puerile. Normal. Feeble. None. Interrupted, Harsh</u>					<u>with expir. prolonged.</u>
	<u>in Adult</u>					
	<u>Transition Type (Broncho-vesicular or "indeterminate" breathing)</u>					
(2) Bronchial or Blowing						
	<u>High pitched</u>	<u>Medium pitched</u>	<u>Medium pitched</u>	<u>Low-pitched</u>	<u>Low-pitched</u>	<u>or Cavernous.</u>
	<u>or Tubular</u>					
(3) Amphoric.						
	<u>High-pitched</u>	<u>Medium-pitched</u>	<u>Low-pitched</u>	<u>Low-pitched</u>		

"NOTES. - 1. Vesicular Breath-sounds. - In the auscultation of normal Vesicular Breathing, the Inspiratory sound, represented in the diagrams by a single line, is a fine, continuous, rushing sound, soft in the adult and loud in the child, and audible from beginning to end of

the act. The Expiratory sound, on the other hand, is thin in quality and of short duration, being audible only during the earlier part of the Expiratory act. It is generally believed that in normal Vesicular Breathing the Expiratory sound passes, as represented in the diagram, directly into the Expiratory without a break. Having, however, paid special attention to this point, Dr W. believes that there is often, in perfectly normal Vesicular breathing, a distinct break between the two sounds. When the breathing is quiet and easy, the Expiratory sound is often totally inaudible, even in children; but in such cases it can usually be brought out by causing the patient to breathe deeply.

"The term "prolonged expiration" is used to signify not a prolongation of the act of Expiration, but only a prolongation of the Expiratory sound, resulting from the encroachment of the audible upon the inaudible part of the act. There is indeed one form of breathing, common to advanced Emphysema and Asthma, in which the act itself is really prolonged, being often much longer than the Inspiration. In such "Asthmatic" breathing, the type of respiration, primarily Vesicular, is as a rule totally masked by the loud wheezing accompaniments of both Inspiration and Expiration.

"2. Bronchial Breathing. - The auscultatory sound of Bronchial Breathing, indicated in the diagrams by a double line, can be imitated, as pointed out by Skoda, by holding the tongue in the position for the pronunciation of the guttural ch sound (as in the German word Ach or the Scotch word Loch), and causing the air to pass in-

wards and outwards over it. The blowing sound thus produced can be made to represent the various pitches indicated in the diagram. There is always in Bronchial Breathing a distinct break between the sounds of Inspiration and Expiration, and the two sounds closely resemble each other. The higher-pitched varieties of Bronchial Breathing should be associated in the mind with conditions of consolidation of Lung Substance, such as that of Pneumonia and the Low-pitched or Cavernous variety with Excavation, as in Phthisical cavity. Bronchial Breathing is never produced by Bronchitis.

"3. The Amphoric type of breath-sound can be well imitated by whistling with the mouth. Inspiratory and Expiratory sounds can thus be produced by causing the air to pass inwards and outwards, and the pitch can be varied according to the variety of Amphoric Breathing that is being imitated. Amphoric Breathing is best developed in Pneumothorax, but is also sometimes met with in very large Phthisical cavities.

"4. In the Healthy Chest the respiratory sounds are purely Vesicular (without harshness of quality or prolongation of expiration) over the whole surface of the lungs, except (1) opposite the Roots of the Lungs, at level of third dorsal vertebra behind, and lower part of manubrium sterni in front, where the proximity of the large Bronchi generally renders the breathing Broncho-vesicular, by the addition of a Blowing or Bronchial element, most distinct during expiration; (2) over the Apex of the Right Lung, especially above the clavicle and Spine of Scapula, where, in health, from causes as yet

imperfectly ascertained, the Vesicular Breath sound has very generally a more or less prolonged, and often harsh or even somewhat blowing, expiration.

"The only example of purely Bronchial Breathing that can be heard on auscultating the healthy subject, is the "Tracheal" Breathing, to be obtained by placing the stethoscope over the Larynx or Trachea. This is low in pitch, and if heard over the apex of the Lung would be termed "Cavernous."

[Unfortunately some of the great original authorities on Auscultation applied the term "Bronchial" to the type of breathing heard over the roots of the lungs, but, as this is partly of Bronchial and partly of Vesicular origin, the term "Broncho-vesicular" is much more appropriate.]

II. ACCOMPANIMENTS.

- | | | | |
|--|---|-----------------------|---|
| (1) Friction
(in Pleurisy) | // | // | // |
| | <u>Fine</u> | <u>Medium</u> | <u>Coarse</u> |
| (2) Dry sounds or
Rhonchi (in
Bronchitis) | /// | /// | /// |
| | <u>High-pitched</u>
(<u>Cooing, Wheezing,</u>
<u>etc., called</u>
<u>"Sibilant"</u>) | <u>Medium-pitched</u> | <u>Low-pitched</u>
(<u>Sonorous</u>) |
| (3) Moist Rales, or
Crepitations. (1 and
2 in Pneumonia, 2 in
Bronchitis, 2 and 3
in Phthisis) | ••••• | ••••• | ••••• |
| | 1. <u>Fine</u> | 2. <u>Medium.</u> | 3. <u>Coarse</u>
(<u>Bubbling</u>) |

May be Consonating in three degrees - tough, metallic, tinkling. (Tinkling rales are specially important in connection with Pneumothorax.)

III. VOCAL RESONANCE.

- (1) Simple Increase. (a) Slight, comparative
 (b) Marked (Bronchophony)
 (c) Very marked (Pectoriloquy)

(The chief conditions which cause Increase of Vocal Resonance are Consolidation and Excavation of the Lung substance).

- (2) Simple Decrease. (a) Slight, comparative.
 (b) Marked decrease.
 (c) Total absence.

(Decrease of Vocal Resonance is most frequently due to Thickening of the Pleura or to Pleuritic Effusion).

- (3) Qualitative Alterations. (a) Aegophony (Nasal timbre)
 (b) With metallic echo (Amphoric Resonance, or Nachklang).

(Aegophony occurs in Pleurisy with Effusion when the layer of fluid is thin. Metallic echo is one of the signs of Pneumothorax.)"

While auscultation is of extreme value in diagnosis, it is very important that the changes in breathing should correspond to changes in the expansion of the lung, as elicited by percussion. Thus Leube (Specielle Diagnose der Innern Krankheiten, p. 149) writes: "Still more important for the certainty of the diagnosis is a variation of the percussion sound corresponding to stethoscopic variations and to changes from the normal breath sounds. In such cases the slightest changes in the respiratory murmur are sufficient for diagnosis: cog wheel breathing, protracted expiration, weak, aggravated, or rough breath sounds. So also if the breath sounds have a bronchial character, are rough, or enfeebled, and if there exists

the slightest dulness on percussion at the apices, they have a great diagnostic importance." In those prone to phthisis the features are regular and well formed. The

PART II.

The Physical Signs of Early Phthisis.

Here we will consider -

1. Patient's build.
2. Patient's facies.
3. Signs in the chest.
 - a. Shape of Chest
 - b. Dilated Veins
 - c. Delayed expansion
 - d. Tidal breathing.
 - e. Pulmonary Signs
 - f. Radioscopic Signs.

1. A particular conformation of build has long been supposed to be associated with pulmonary tuberculosis. Thus Hippocrates ¹⁶⁵ wrote: "The form of body peculiar to subjects of phthisical complaints was the smooth, the whitish, that resembling the lentil, the reddish, the blue-eyed, the leuco-phlegmatic, and that with the scapulae having the appearance of wings."

We can recognise the following types of patient:

- (a) The nervous irritable patient, who appears to be ill and wasting. Such patients are usually tall.
- (b) The lymphatic patient, who is slow, dull, and languid, yet complains little.
- (c) The wiry patient, who is thin and active, but not emaciated. These are usually of small stature.

2. Facies.

A distinct facies is often seen in those prone to phthisis. The features are regular and well formed. The hair is long and silky, growing well forward at the temples. Long eyelashes overhang large bright eyes. The mouth and chin are well marked, and the malar ridges appear prominent. The complexion is soft and clear.

3. Signs in the Chest.

(a) Shape. The chest of phthisis resembles the chest of childhood, the antero-posterior being longer than the transverse diameter. It is the long, narrow, flat chest, although the flatness is more apparent than real owing to the scapulae slipping forwards,

(b) Dilated Veins. On inspection of the integuments dilated veins are frequently seen over the upper area of chest. There are two varieties, the red and the blue. The red which is probably due to vasomotor disturbance may be seen early in the disease, but the blue are associated with more marked lesions of the lung, being due to some interference with the venous circulation.

(c) In looking for delayed expansion we place the hands with the fingers extended flat on the apices, the thumbs just touching in the middle line over the sternum. The patient takes a deep breath, and we watch the tips of the thumbs to see if one moves outwards more slowly or to a less degree than the other.

(d) "Tidal Breathing."

Philip 163 has shown that a diminution of "Tidal Breathing" at the apices is one of the earliest physical signs of phthisis, and is apparent long before any dul-

ness is to be obtained below the clavicle, may be ac-

(e) Pulmonary Signs of Early Phthisis.

In the chapter on Pathology, we have seen that the pathological process at work in a tuberculous lung is divisible into four stages. 1st Proliferation and catarrh; 2nd Consolidation; 3rd Caseation; and 4th Excavitation. The pulmonary signs of phthisis will be described in reference to these stages. Some have objected to this as being an artificial combination. On the other hand we hold it to be the most natural, for although one was proficient in the whole vocabulary of Laennec yet was unaware of the pathological significance of the terms used, such knowledge would be of little clinical value.

In early phthisis we will therefore consider the pulmonary signs of

Stage I. The proliferation and catarrh of a few apical nodules.

Stage II. The consolidation of tissue about these nodules.

Stage I. Inspection will reveal neither flattening nor delayed expansion. Percussion will fail to produce any alteration in resonance, but will show a diminution in "Tidal Breathing." On Palpation there will be no increase of vocal fremitus. These signs are negative because the pathological condition is a catarrh and proliferation of the cellular elements of the alveoli and bronchioles of one apex, which will give on auscultation the signs of localised bronchitis. The breath sounds are harsh, with prolonged expiration, due to the congestion

of the tissues, while this hoarse breathing may be accompanied by one or two sibilations, which are brought out by coughing.

It must be remembered that in children the breathing is normally hoarse over both apices, and that in adults it is hoarser over the right than over the left apex, on account of the high position of the right bronchus, so that to denote a pathological change hoarse breathing must be marked, and distributed over a wider area than is normally the case. At this stage vocal resonance is not increased.

Cog-wheel breathing is also got in early phthisis. It is hoarse, interrupted, and wavy in character. Some hold it is due to the nervous action of the respiratory muscles, or to the beating of the heart, but the writer believes he has frequently heard it in the early stages of disease, The sputum may contain tubercle bacilli.

Stage II. The proliferation and catarrh have now resulted in the consolidation of the bronchioles and alveoli in the neighbourhood of the tuberculous nodules. This is not sufficient to give flattening on inspection but will cause delayed expansion, and a varying amount of dulness on percussion. On auscultation the breathing has become broncho-vesicular, as the area of consolidation is not large enough to produce pure bronchial breathing. Vocal Resonance and Vocal Fremitus are increased. In this stage the hoarse inspiration of broncho-vesicular breathing may have an interrupted or cog-wheel element. The sputum contains tubercle bacilli.

(f) Radioscopic Signs.

This method of diagnosis has been chiefly used in France, where it has yielded excellent results. It is claimed that by the recognition of a limitation of the diaphragmatic movements, particularly of the right side, phthisis may be diagnosed earlier than by any other method. 166

Thus, Béclère writes 167, "The Röntgen rays are particularly valuable in cases where tuberculosis is only suspected in the very early stage. Examination by the radioscope and radiograph supersedes all other methods, and shows a diminution in the clearness of the lung at one of the apices, often accompanied by a diminished descent of the corresponding half of the diaphragm; and for a certain time this last symptom may be the only one observed. More often this new method confirms the record made on auscultation, and shows that attending the slight and dubious modifications of the respiratory murmur there is positive condensation of the pulmonary tissue. However, in other cases auscultation and percussion forestall Röntgen rays in proving change in the lung." As to methods: "These different methods, radioscopic examination, simple radiography, cinemato-radiography, and stereoscopic radiography, are of mutual assistance to one another in diagnosing thoracic affections, but to the medical practitioner they are not all of equal importance. Of the above methods, radioscopic examination is the simplest, easiest, quickest, and least expensive. But that is not all; it surpasses all other methods in the amount and importance of the information it gives in a short

time. Hence it comes first, and more often than not by its use in diagnosis it is possible to dispense with the others."

METHOD OF X.RAY EXAMINATION OF THE LUNGS

The patient stands eighteen inches from the tube, and a barium platino-cyanide screen is placed against his back.

The first thing to be noted is the "piston-movement" of the diaphragm, which rises and falls during expiration and inspiration, to an extent of from half an inch to three inches, which varies with the depth of breathing. If there be a tuberculous deposit even at the apex of one lung, the movement of the diaphragm is greatly lessened on that side.

The ribs and clavicles are seen horizontally on either side, and the shadow of the scapula on each side of the spinal column. The shadow of the heart is seen to the left, and frequently there is a vertical shadow to the right of the cardiac area. This Cunningham believes to be due to the puckering of the outer edge of the pericardium during cardiac systole.

The healthy lungs have a transparent appearance which extends up to the apices, which are fully lit up on inspiration. If there be a diminution of light at one apex, it is absolutely diagnostic of active or arrested disease.

Further if there be pressure on the bronchi, it is possible to determine the cause of this, whether enlarged glands or aneurism, as the pulsations of the latter will be seen.

PART III.

The Physical Signs of Later Phthisis.

These are -

1. Patient's appearance
2. Signs in the chest.
 - a. Integuments
 - b. Flattening and delayed expansion
 - c. Pulmonary Signs.
 - d. The Heart and circulation.

1. There is no mistaking the appearance of a patient with later phthisis. Worn and languid, the face is suffused by a hectic flush. The eyes are bright, the sclerotics being a bluey white, forming a contrast to the dark and deepened sockets.

2. Signs in the chest.

(a) The integuments are thinned, the fat being mostly lost in the supra- and infra-clavicular regions. In extreme cases, the ribs are countable through the skin. Large dilated blue veins are seen over the upper portion of the chest, and occasionally patches of pityriasis ~~verrucosa~~⁵~~verrucosa~~, the parasite of which has probably found a nidus in those areas where the perspiration lies longest. A sign of accompanying debility is my^oedema, when a tap on the skin causes a local slow contraction of the subjacent muscle.

(b) Flattening is most marked at the apices, and large supra-clavicular hollows may be present.

The supra-clavicular region may also show great flattening, and over these flattened areas expansion is delayed and diminished.

(c) Pulmonary Signs.

STAGE III. The consolidated area has now undergone caseation, and is of greater extent than it was in the earlier stages. Percussion reveals distinct and marked dulness, and vocal fremitus is increased. The breath sounds have now become more bronchial, or tubular, and are accompanied by sub-crepitations in the form of clicks, cracklings, or musical râles. These are intensified by making the patient cough, and are produced by the movements of air in the caseating material in the alveoli and bronchioles. They may also be caused by the presence of small quantities of fluid blood, from a haemoptysis, in the alveoli.

STAGE IV. Excavitation.

This is caused by the softening of the caseous areas with the surrounding production of fibrous tissue, which results in the formation of a cavity. Inspection shows hollowing above the clavicle, with flattening below.

Percussion would be expected to give a tympanitic note over a cavity, but this is masked by the dense wall of fibrous tissue with which it is surrounded, so that the percussion note contains the elements of resonance and dulness, which gives the "Wooden or Boxy note." Another note given by percussion over a cavity is the "cracked

pot" note, which is elicited by forcible percussion while the patient breathes audibly with the mouth open. The metallic sound, resembling that got by striking a cracked pot, is said to be due to the sudden expulsion of air from the cavity.

Auscultation over a cavity reveals breath sounds which vary from low pitched bronchial breathing to loud amphoric breathing, which is got in large cavities. These breath sounds are accompanied by loud coarse bubbling rales, from the splashing of the pus in the cavity, but if the pus be small in amount, the rales have a consonating character, and are metallic or tinkling.

Vocal resonance is increased to Pectoriloquy, so that the voice produces the same painful impression to the ear, as is apparent when someone speaks too loudly into the telephone.

COURSE OF PULMONARY LESIONS

We have here seen that in pulmonary lesions the health sounds usually become hoarser as the disease advanced. This applies particularly to the upper lobes. It is well known, however, that such is by no means always the case with regard to the lower lobes, and frequently the first sign of their involvement is a diminution of the breath sounds. It may be that they are more usually involved to a larger extent than was the upper lobe, where the lesion commenced, with the result that their functional activity is diminished. Diminution of breath sounds is also due to thickened pleura, following on pleurisy.

(d) The Heart and Circulation.

Barie 168 has shown that the changes in the heart depend to a large extent on whether the pathological condition in the lungs is mostly caseous or fibrous. If the disease is caseative with cavity formation, there are few changes in the heart, which, however, may show the usual degenerations. When the process of fibrosis is extensive in the lungs, the heart suffers from the obstruction to the pulmonary circulation, which induces hypertrophy of the right heart.

CHAPTER VIII.

BLOOD REACTIONS IN THE DIAGNOSIS OF TUBERCULOSIS

It is becoming more and more apparent to those who are to-day carrying out the treatment of tuberculosis, pulmonary and surgical, on hygienic lines, that the success of this method is largely dependent on the early recognition of the disease. During the past few years this fact has been so realised that many clinicians have written on the diagnosis of phthisis by means of physical signs and symptoms before tubercle bacilli have appeared in the sputum, in what has been termed the "praetubercular" stage. When one remembers that the most frequent early manifestations of phthisis - acid dyspepsia, gastric irritability, chlorosis, amenorrhoea, palpitation, faintness and general indisposition may be caused by a variety of other conditions, while slight changes in the breath sounds are due to many diseases apart from phthisis - chronic pulmonary and bronchial catarrhs of various bacteria and irritants, cardiac disturbances, malaria, and last, but it may be by no means least, syphillis - it is obvious that there is at least a danger of the niceties of diagnosis being carried too far. If phthisis is to be diagnosed by general indisposition, indigestion, and a slight alteration in breathing over a small area of the lung, before the appearance of tubercle bacilli, it is clear that many patients will be most un-

justly condemned to Sanatoria life, to enforced residence abroad, and to pecuniary loss, and that the statistics of Sanatoria "cures" will be subject to corresponding error. There is a shrewd suspicion abroad in the profession that such cases have been by no means uncommon, and therefore the application of recent advances in blood reactions is to be welcomed, as assisting in the solution of what is often an extremely difficult clinical problem. Early treatment cannot be overrated, but there are certain conditions in which the absolute diagnosis of tubercular infection is often a matter of great difficulty

(a) Where the patient presents the symptoms so often indicative of early phthisis without showing bacilli,

(b) in acute tubercular lobar pneumonia simulating ordinary pneumonia, (c) in the phthisis florida of children following unresolved broncho-pneumonia, and (d) when the diagnosis of surgical tuberculosis is doubtful. In such cases six means have been suggested whereby the question of tuberculosis can be definitely answered

1. Opsonic Index
2. Reaction of Opsonic Index to Tuberculin T.R.
3. Heated Serum Test
4. Von Pirquet's Sign.
5. Ophthalmic Reaction of Calmette.
6. Old Tuberculin Reaction.

1. OPSONIC INDEX

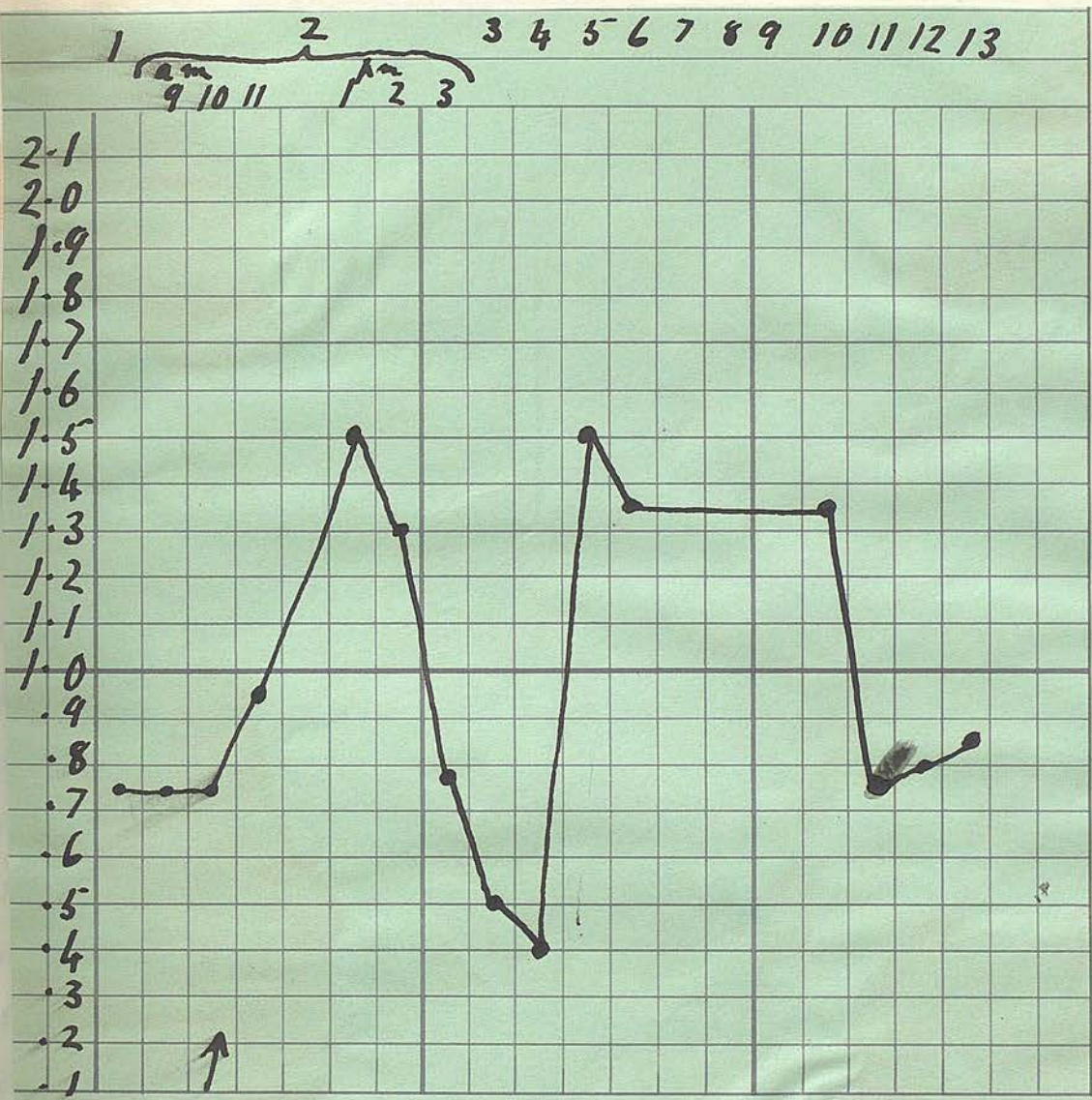
The Opsonic Index represents the amount of those bodies in a patient's serum antagonistic to any given bacterium, compared with the amount present in normal

serum. It is by means of these bodies, named by Wright "Opsonins" (from the Greek "to prepare a feast") that the phagocytes are enabled to digest the bacteria, whose toxic properties are neutralised by the opsonins, so that the amount of opsonins present in the serum is an index of the patient's resistance. As has been stated the amount of opsonins in a healthy person is taken as normal, and we have now to consider how infection by the bacillus of tubercle will affect the amount of opsonins present antagonistic to that bacillus. It is well known that if a healthy animal be injected with small or attenuated doses of an organism, or with the products of an organism, it will become immune to the injection of large quantities of that organism or its toxins. Now it has been shown by Wright and Douglas, Bullock and others, that by injecting the dead bodies of bacteria or a solution of dead bodies and endotoxines, such as Koch's Tuberculin, T.R., it is possible to raise the amount of opsonins in the blood. This phenomena may be explained as follows. When the bacterial debris is subcutaneously injected, a very small quantity is immediately absorbed by the capillaries into the general circulation, where, by absorbing a minute amount of opsonins, it sets in action the mechanism of repair. By the law of regeneration there follows an excessive production of opsonins so that a few hours later there is an immediate rise in the opsonic index - the prenegative rise ¹⁶⁹. Later on the whole injection of tuberculin is gradually absorbed by lymphatics and dilated capillaries, with the result that

the opsonins are neutralised and a negative phase is recorded by the index. This negative phase persists a longer or shorter time according to the amount of tuberculin injected, but this latter once absorbed the body starts afresh the manufacture of opsonins, and the index records the positive phase. The positive phase lasts a certain time after which the index falls to its previous level, but if a very large dose of tuberculin be given the negative phase will persist for three weeks with the entire absence of a positive phase. If again during a positive phase more bacterial debris be injected the balance of opsonins will be disturbed and the index fluctuate. The above facts have been proved experimentally, and appear to be quite analogous to the opsonic phenomena in tubercular infections. (Chart. 1.)

In health the opsonic index varies from .9 to 1.2. and although different observers have found different averages, an index remaining within these limits may be regarded as normal. The index may vary slightly from day to day in health, but the daily variation never exceeds three degrees. The following are the average indices in health noted by various observers:

	Number of Estimations	Average Index	Range of Index	Total varia- tion.
170 Urwick	20	1.006		
171 Bullock	86	0.96	.8 - 1.2	.4
172 Lawson & Steward	25	1.0		
173 Shaw	35	1.075	.97 - 1.32	.35



T.R. $\frac{1}{100}$ myr

Chart. 1.

The writer found the following indices in nine healthy persons - 0.99 1.1, 0.97, 0.95, 1.2, 0.98, 0.92, 0.91, 0.97.

9. 0.99 0.91 - 1.2. .29

In ill-health apart from phthisis, the opsonic index may fall to .1 or .3¹⁷⁴. When the opsonic index is low, the resistance to infection is weakened and if it remain persistently low there is a predisposition to phthisis.

In the insane the opsonic index is low. Shaw¹⁷³ found that the daily opsonic index of thirty insane persons averaged .88 for a period of five days. Of these thirty cases, five had previously suffered from tubercle, but in these the index also averaged .88. These observations are of interest as the incidence of phthisis among the asylum insane is according to Clouston¹⁷⁵ three or four times greater than it is among the general population at the same ages.

When a tubercle bacillus settles down in the lung and proliferates, the result is that, on account of the great vascularity of this organ, small quantities of bacterial products and débris will find their way into the general circulation, where they will act in the same manner as a small dose of tuberculin, and raise the opsonic index. This is auto-inoculation, and so long as it remains small the opsonic index will be high, constituting the natural resistance of the body against

infection, which doubtless in many cases is sufficient to overcome the invader. It has been proved experimentally that one millionth of a c.c. of Tuberculin will raise the opsonic index ¹⁶⁹, so that the presence of even a minute number of tubercle bacilli will be indicated by a high index. The value of this fact in diagnosis is illustrated by the following case:

A.B. aet. 17, medical student, was seen by the writer with Dr John Owen of Liverpool. Six months previously the patient had influenza, and now complained of general weariness, malaise, and loss of appetite. There were no signs in the lungs. Now and again he would cough up a little sputum in the morning which showed no tubercle bacilli even after digestion with pepsin and centrifugalisation. The opsonic index was 1.4. A diagnosis of early phthisis was made, and the patient removed to the seaside where he commenced the Open Air Treatment. He was again seen a month later, when the opsonic index was 1.1. The sputum was negative to microscopic examination, but a small quantity being injected into a guinea pig, the animal in six weeks showed two caseous glands.

This case indicates that a high opsonic index in conjunction with ill health is diagnostic of phthisis, and this is probably the earliest sign of the disease.

If now the disease is rapidly advancing there will be local congestion at the periphery of the tubercular foci and larger quantities of bacterial debris will

Opsonic Index Active Phthisis.

1 2 3 4 5 6 7 8 9 10 11 12 13

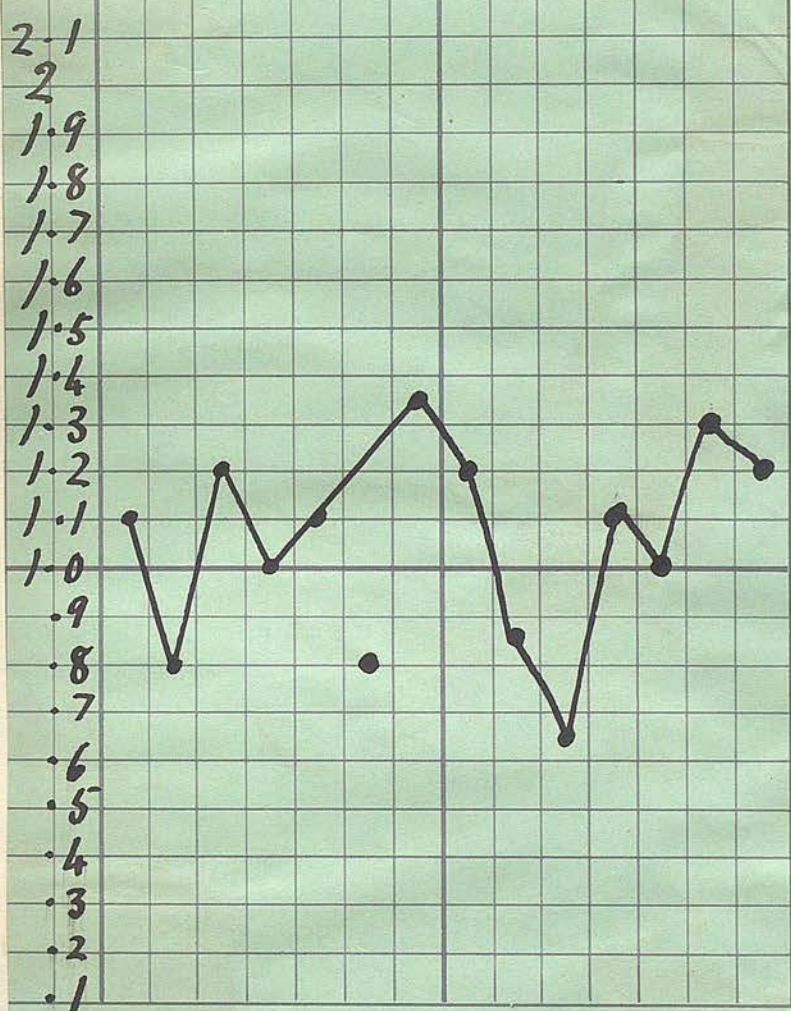
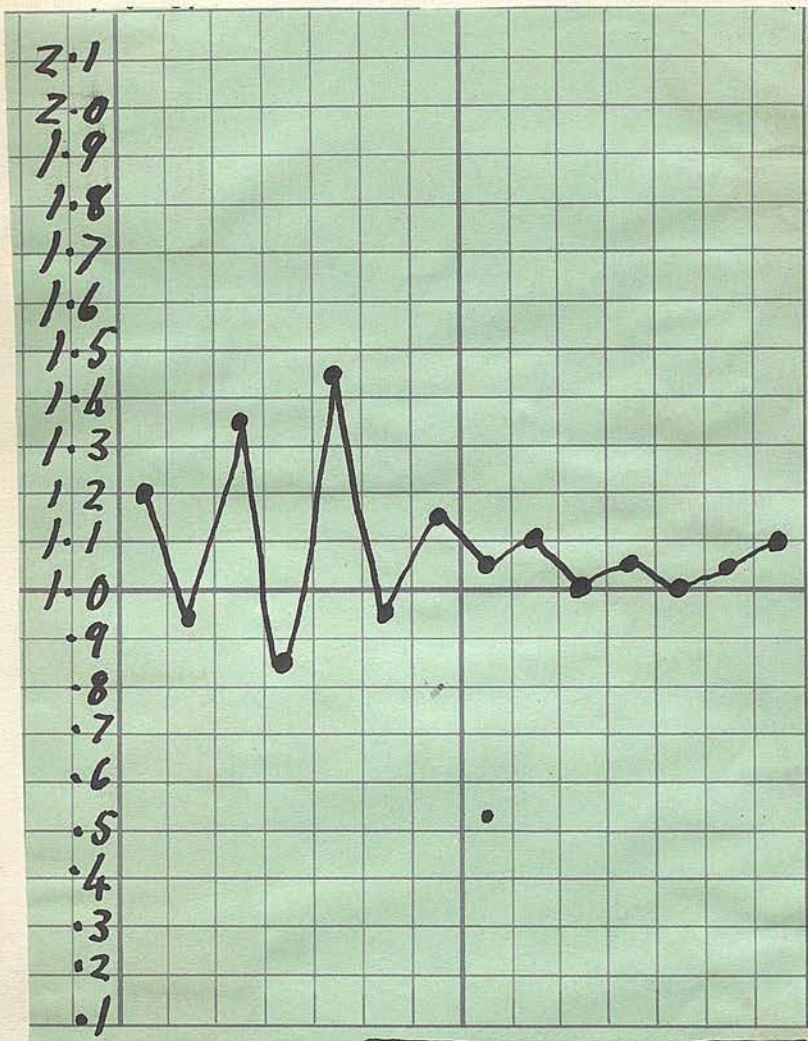
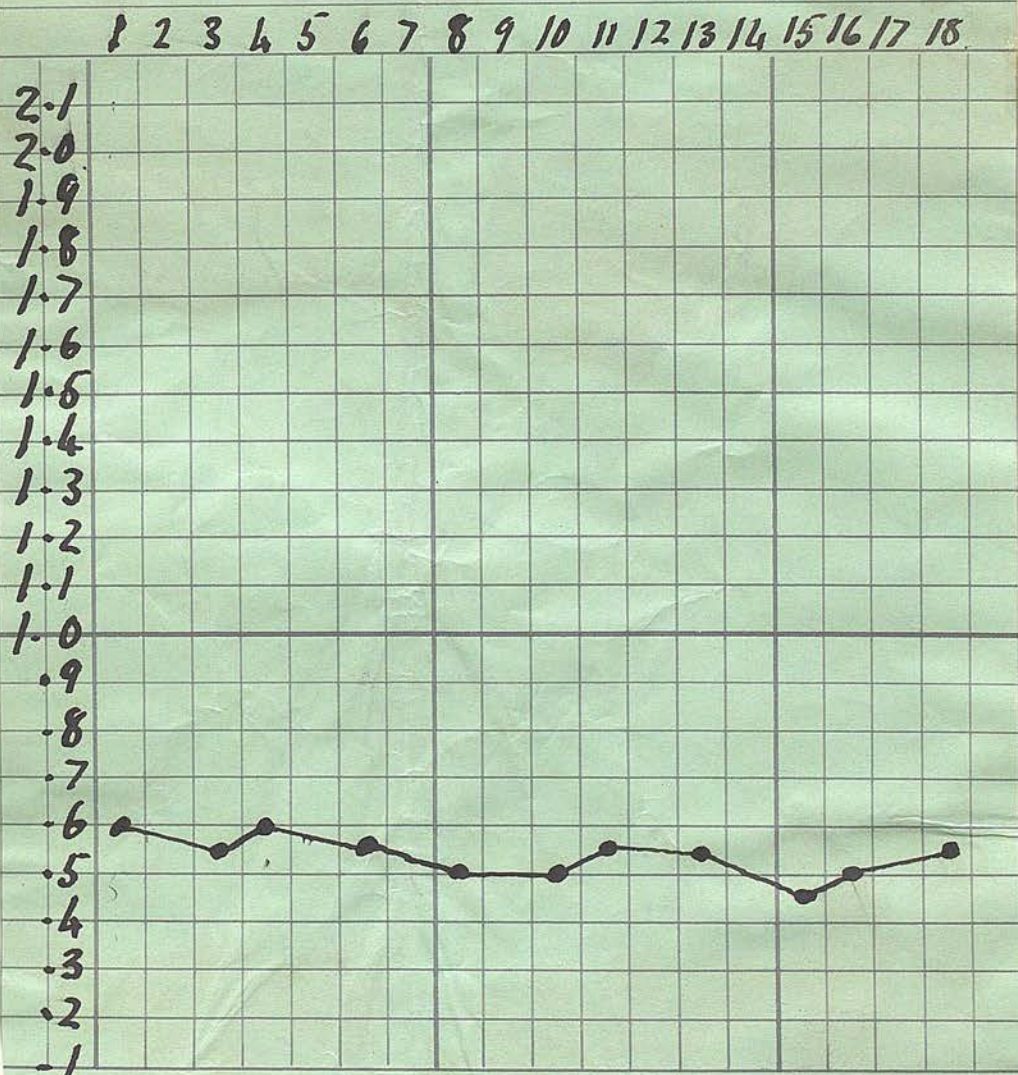


Chart II.



Rest in bed.

Chart III.



Opsonic Index
 Chart IV Surgical Tuberculosis.

find their way into the general circulation, so that the opsonins present will be neutralised and the index fall, after which it will rise. A fluctuating index thus indicates very active disease. (Chart II).

If however a patient be taking excessive exercise a smaller lesion will produce the same effect from excessive auto-inoculation due to the increased blood and lymph flow through the lung. This is distinguished from the previous case by putting the patient to bed, when the opsonic index will become steady. (Chart III).

When phthisis of the lung is healing, we have a precipitation of fibrous tissue around the tubercles, accompanied at times by calcification, whereby the spread of infection in the lung is arrested, the foci of disease localised, and shut off from the general circulation, so that auto-inoculation having ceased the opsonic index remains steady. The actual figure at the opsonic index of healed phthisis stands is a matter of lesser importance, but in thirty cases of sanatorium cures Lawson and Steward¹⁷² found the opsonic index to vary from .5 - 2.1. In surgical tuberculosis, where in the absence of accidents the lesion is localised, the opsonic index is always low. (Chart IV).

It is thus obvious that in all varieties of tubercular infection the opsonic index has its place in diagnosis and prognosis. The deductions to be drawn from these indices may be tabulated as follows:

It is assumed that several estimations have been made, one being quite insufficient.

A. Low Index, below .9 indicates:

1. In the absence of pulmonary signs:
 - (a) General ill-health
 - (b) Predisposition to tubercle.
2. In the presence of pulmonary signs:
 - (a) Healing tubercle
 - (b) Healed tubercle.
3. In the presence of suspected surgical tubercle:
 - (a) This is confirmed.

B. Normal Index, .9 - 1.2 indicates:

1. In the absence of pulmonary or other signs:
 - (a) Freedom from tubercle.
2. In the presence of pulmonary signs:
 - (a) Healed tubercle.
 - (b) Healing tubercle.

C. High Index above 1.2 indicates:

1. With general ill-health
 - (a) Early phthisis.
2. With pulmonary signs
 - (a) Healed tubercle
 - (b) Healing tubercle.

D. A fluctuating Index, swinging four points or more per diem, indicates in all conditions active disease of the lung, and signifies:

1. If the patient is going about
 - Excessive exercise.
2. If it persists after rest in bed:
 - Rapidly advancing phthisis.

2. THE REACTION OF THE OPSONIC INDEX TO TUBERCULIN, T.R.

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 Wright noted that in tubercular Patients the negative phase following an injection of tuberculin is more prolonged than in healthy persons, and suggested that a negative phase might be produced by a smaller dose of tuberculin than that required to produce the same effect in health. Lawson and Steward¹⁷² injected four healthy persons with tuberculin, (dose not stated) which resulted in the immediate production of a positive phase, the negative phase being absent, from which they concluded "that the appearance of a negative phase after inoculation with a small quantity of tuberculin, T.R. should enable one to distinguish between the blood of a subject of tubercular invasion and that of a person in sound health." On the theory which the writer has sketched above that tuberculin is neutralised in the body by the opsonins present this conclusion would hold true if the opsonins present were diminished in amount. That is if there be a small amount of opsonins in the blood a small quantity of tuberculin will completely neutralise it and so produce a negative phase, while if the opsonins be abundant the same quantity of tuberculin will not be sufficient to produce a negative phase. Shaw¹⁷³ has demonstrated this in practice. Two control cases, whose indices prior to injection were 1.01 and 1.11 respectively, received an injection of $\frac{1}{750}$ mgr. T.R. which was also given to eighteen insane patients with an

average index of .89. The two controls showed an immediate rise to 1.25 and 1.33 respectively without a negative phase. Of the eighteen patients only four showed a negative phase (of these four, one had evidence of old infection) while the remaining fourteen missed the negative phase. Although the low index of these fourteen patients was not sufficiently low to produce a negative phase with $\frac{1}{750}$ mgr T.R. its effect was yet manifested in a striking manner..."The chief difference noted between the two control cases and the 14 insane patients in whom no negative phase following inoculation with $\frac{1}{750}$ of a milligram of tuberculin T.R. was the slower reaction of the latter after injection. Both control cases reached their maximum on the day following injection, while the insane took an average of four days to reach their positive maximum, but there was little difference in the level ultimately reached, though the average index before injection was lower in the insane ¹⁷³.

As a result of his experiments Shaw considers that $\frac{1}{750}$ mgr T.R. is too large a dose to be applied in this manner for diagnostic purposes. He concludes as follows: "The injection of a small dose of tuberculin T.R. in healthy persons produces no negative phase to the tubercle bacillus and therefore may be used as a method of diagnosis. A smaller dose of tuberculin will, however, produce a negative phase in a predisposed person than in one less liable to tuberculous infection ¹⁷³."

These experiments are of interest as throwing light on the nature of the reaction of the blood fluids to tuberculin, but owing to the influence of dose, individual idiosyncrasy and the height of the opsonic index on the reaction, it is doubtful if this method is as accurate or simple for diagnostic purposes as a daily estimation of the index alone.

3. HEATED SERUM TEST

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Wright and Reid found that if normal serum be heated for 10 minutes at 60°C. it loses nearly all the opsonins present, whereas the serum of a patient with active tubercle or under treatment with tuberculin retains a considerable quantity of opsonins. This test is of value if only one serum reaction is to be done. The theoretical question involved as to the duality of opsonins will be considered later.

4. VON PIRKET'S SIGN.

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Von Pirket found that when a small quantity of Tuberculin is introduced into a slight abrasion on the skin of a tubercular subject, redness and oedema appear after forty-eight hours, and that this is frequently followed by a papule like that of vaccination. At the end of eight days the papule drops off and the redness disappears. Von Pirket considered this sign to be exceptional in healthy people. Vallée¹⁷⁹ of Alfort stated that this diagnostic use of Tuberculin which he

of trying the effect of Tuberculin on mucous membranes
 named "cuti-reaction" gave the same results in bovines.

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Fernand Arloing tested this method in 19 animals (cows, sheep, dogs, rabbits and rats), of which ten were tubercular, but failed to find a specific cutaneous reaction to Tuberculin. He observed a fleeting redness and swelling at the edges of the scarification at the end of twenty-four hours, but this was as well marked in the healthy animals as in the tuberculous. Thinking that the glycerine contained in the Tuberculin might be responsible for the local reaction, he made a new series of observations in which he applied 50% glycerine to the skin abrasion. The result was the production of a local reaction, less marked, however, than that obtained with Tuberculin.

From this it would appear that Von Pirket's sign, by its inconstancy, the method of eliciting, and by the time required, is not suited for diagnostic purposes.

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Bandler and Kreibich have tested the reaction on 26 cases of tuberculous skin disease, and of 37 central cases of syphilis and gonorrhoea. Of the 26 cases of tuberculous skin disease, 22 gave a violent reaction, while 4 cases of miliary tuberculosis of mucous membranes did not react at all. Of the 37 controls 22 gave a reaction at once, which was well marked in 12 cases, which included two cases of syphilis in individuals whose apices showed suspicious signs.

5. CALMETTE'S OPHTHALMO-REACTION

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Von Pirket's Sign suggested to Calmette the idea

of trying the effect of Tuberculin on mucous membranes and particularly on the conjunctiva, which readily absorbs many microbic, vegetable and animal toxins, such as diphtheria toxin, abrin, and snake venom. The experiments were done in conjunction with his pupils preton and petit in the Hospital at Lille. Calmette had already commenced his experiments on this subject, when Wolff-Eisner¹⁸³ at the Berlin Medical Society, speaking in a discussion on Von Pirquet's Sign, suggested replacing this by instilling a 1. in 10 solution of Tuberculin into the eye of suspected phthisical patients but he had not yet proved the value of this method.

As ordinary Tuberculin is in a 20% solution of glycerine, which itself irritates the conjunctiva, dry tuberculin was obtained by precipitation with alcohol and dissolved in distilled water 1 - 100. A drop was placed in one eye of each patient. The following is Calmette's description of the reaction in tuberculous patients.

"Five hours, sometimes three, after the drop has been placed in the eye tubercular patients show a very marked congestion of the palpebral conjunctiva, which takes on a livid red tint, and is the site of more or less oedema. The caruncle is red, swollen, and covered by a thin fibrous exudate."

"The injection of the blood vessels increases slowly and is accompanied by lacrymation. At the end of six hours the fibrous secretion becomes more abundant and the inferior conjunctival sac is full of filaments."

"The reaction reaches its maximum in six or ten hours at latest. The patients have no pain, only a little discomfort with a slight sensation of heat and some trouble of vision, according to the abundance of the exudate. There is no chemosis, and the rectal temperature is unaltered. The intensity of the reaction is easily appreciated by comparison with the sound eye."

"In children at the end of 18 hours, in adults at the end of 24 or 36 hours, the congestion is less marked and then disappears."

"In healthy subjects and in those not suffering from tuberculosis the drop of Tuberculin is perfectly inoffensive, and in 1½ to 3 hours there is nothing but slight redness, which disappears quickly, and is neither accompanied by fibrous secretion nor by lacrymation."

The reaction was got in every case of pulmonary or surgical tuberculosis. Comby¹⁸⁴ in trying the reaction on 24 children got a very violent result in two cases - the whole eye injected, epiphora, oedematous swelling of the lids, fibro-purulent discharge, and in general the appearance of purulent ophthalmia. This phenomona took eight days to disappear.

To avoid this he used a solution of 1 - 200 tuberculin and this in a series of 108 cases only gave moderate reactions, which were quite typical and had the further advantage of not inconveniencing the patient.

With this dilution when the reaction is positive the eye becomes injected at the internal angle in from five to ten hours, with swelling of the caruncle. The

redness spreads to the lower lid, to the ocular conjunctiva, and is generalised in twenty-four hours. In many cases, however, it remains localised at the internal angle, where it should always be looked for. Along with the redness there may be epiphora, and a slight sero or fibro-purulent secretion, which is not serious and disappears in two or three days.

Comby distinguishes three degrees of the reaction, mild, moderate and violent.

1. Mild Reaction. This is so mild that it may pass unnoticed unless carefully looked for in the internal angle, by asking the patient to look outwards and by carefully comparing the conjunctiva of the two eyes for redness and swelling.

2. Medium Reaction, which can be seen at a distance, the redness having passed from the internal angle over the whole conjunctiva, so that the eye has the appearance of acute conjunctivitis.

3. Violent Reaction. Here the eye is vividly injected, the lids swollen and oedematous, with epiphora and a purulent secretion. The patient cannot open the eye, which resembles purulent ophthalmia. This violent reaction never occurred with a solution of 1 - 200 Tuberculin.

The reaction is contra-indicated in all acute or chronic diseases of the eye, blepharitis, conjunctivitis, keratitis, etc. If one eye is diseased the reaction is still contra-indicated, owing to the risk of sympathetic

infection of the sound eye with the disease in the other and the further difficulty of judging the degree of reaction, there being no normal conjunctiva for comparison.

The value of this reaction has been demonstrated by Calmette¹⁸⁵, Letulle¹⁸⁶, Grasset¹⁸⁷, and others in a large number of cases of pulmonary and surgical tubercle, in which the reaction was constantly present, and further it has been obtained in cases where tubercle had not been suspected, but which later at the autopsy were proved to be tubercular. A striking case is recorded by Letulle¹⁸⁸ of a morpho-maniac with chronic nephritis in whom a positive Ophthalmo-reaction was obtained: "This last patient entered the Hospital dying, his skin covered by ulcers and subcutaneous abscesses caused by the morphia needle. Four days after obtaining the positive reaction, we found at the autopsy Pott's disease of the lumbar region complicated by two abscesses, which, after all, singularly justified the prolonged abuse of morphia."

Among untoward results from the use of the ophthalmo-reaction, Brunetière¹⁸⁹ has reported a case where a one in five hundred solution was instilled into an eye previously free from tuberculous lesions, but in which at the end of six weeks a phlyctenular keratitis developed. Lapersonne¹⁹⁰ has recorded a case in which corneal and iredo-cyclic complications occur between two and three months after the reaction. It is certain that ocular lesions after the reaction are extremely rare and slow in developing, and it is a question if there is any

casual relationship between them or if it is merely coincidence. Comby makes it a rule that both eyes must be sound, or there is a risk of the sound eye becoming involved after the reaction. True and Maillet¹⁹¹ have done the reaction in eyes affected with a variety of lesions, without noticing any aggravation of symptoms.

A vast number of observations have proved the presence of the ophthalmo-reaction in the great majority of cases of tuberculosis, even where the disease had not been suspected, although some advanced cases have failed to show it. The fact, however, that it has not yet been adopted by the French Army, shows that the questions of its dangers are not decided.

6. OLD TUBERCULIN IN DIAGNOSIS

Old Tuberculin which is chiefly used for diagnostic purposes on animals in this country, is a filtrate of a bouillon culture of tubercle bacilli, which contains the exo-toxins. When introduced it soon fell into bad repute on account of the disastrous effects which followed excessive doses, but it is now becoming extensively used in small doses for diagnosis on the Continent.

Method. A solution of 0.2 mgr. of old Tuberculin should be hypodermically injected, and the temperature taken every four hours. If there is no rise of temperature within two days, 1 mgr should be given, and two days later, if there is still no result, 5 mgr. Should this fail to produce a reaction the case is not tuberculous.

Roepke states that the majority of cases will give a temperature reaction with 0.2 mgr, and that if this fails, it may be followed by injections of 1 mgr, and later by 5 mgr. In a series of seven hundred cases, he has shown that these doses are quite as valuable as the larger doses previously given.

On the other hand, Lowenstein and Kauffmann recommend that for purposes of diagnosis the dose of the tuberculin should not be increased when no reaction follows the first dose, but that in order to obtain a reaction the same amount of 0.2 mg. should be injected if necessary four times over within from twelve to sixteen days, and only when these injections have caused no reaction should larger doses be employed. The method recommended by them is especially suitable for cases of recent tuberculosis with doubtful physical signs - that is, just for those cases in which a correct diagnosis is of the greatest importance. The method is based upon the fact that the first injection, even if no obvious reaction occurs, yet temporarily increases the sensitiveness of the organism towards a second injection, and the second again towards a third. This increase of susceptibility is best seen when the amount of tuberculin used is small, and it is so well marked that the reaction after the fourth injection of 0.2 mg. may be little less in intensity than that after a single dose of 10 mg. After the first four injections, if no reaction has been obtained, the dose is increased to 2 mg., then to 5 mg., and finally to 10 mg. The injections are made in the morning; the patient is kept at rest in bed, and the temperature is taken every three hours. The reaction, according to Koch's rule, is considered positive if the temperature rises at least 5° higher than the mean temperature and there are at the same

time marked subjective symptoms. A three days' interval at least is left between each injection. A tendency to haemorrhages and heart affections, when they are not too advanced and occur in young people do not contraindicate the use of tuberculin, but the injection should not be made in cases where kidney disease or pregnancy is present. In 62 cases of undoubted tuberculosis with bacilli in the sputum, 51 reacted to one of the four initial injections. In 10 of the remaining cases, 8 of which reacted at the fifth injection, that of 2 mg the explanation being that in cases of old tuberculosis with a strong tendency to recovery the resistance of the organism to tuberculin is greatly increased. The second question, as to the possibility of a sound person reacting to the injections, needs no great consideration. If reaction under the old method of quickly-increasing doses is held to be decisive as to the presence of tuberculosis, much more must this be the case when injections of small quantities only are employed. As to the reaction of persons with healed tuberculosis, while it is certain that resistance in such cases may be increased, it is not possible to fix upon a dose of tuberculin such that absence of reaction to it shall definitely occasion a diagnosis of healed tuberculosis; the authors incline to the view of Bandaliers, who would choose the dose of 10 mg. Of 300 cases treated by the method of repeated injections of 12 mg, 69, or 23% reacted at the first injection; 73, or 24.3 per cent, at the second, 107 or 35.7 per cent., at the third, and

51, or 17 per cent, at the fourth. The greatest number of reactions thus followed the third injection; 242 of the cases were in the first stage of phthisis, and of these 99, or 40 per cent, reacted at the third injection. The general subjective symptoms were decidedly mild as compared with those caused by other methods. The reaction usually set in in from five to twelve hours, and reached its highest point in from sixteen to twenty hours. The characteristic local reaction was the same with the smaller as with the larger doses; after the injections râles could often be heard for the first time, or were increased, and in 46 cases tubercle bacilli could be demonstrated in the sputum, although before the injection they had not been present. When the smaller injections are used a protracted reaction is seen less often than with the older method. As a result of their observations the authors conclude that in the greatest number of cases it is not necessary to increase the dose, but that it is enough to inject the same dose four times within from ten to twelve days; that 10 mg, as a limit is arbitrarily chosen, and that by the repeated injection of the same dose the qualitative character of the phenomenon becomes apparent.

1. SYPHILIS

The varieties of syphilitic lesions fall to be considered in the differential diagnosis between this disease and phthisis: - syphilitic pleurisy and pneumonia

CHAPTER IX

THE DIFFERENTIAL DIAGNOSIS OF PULMONARY TUBERCULOSIS

The following conditions may simulate pulmonary tuberculosis:

A. Rare Conditions

1. Pulmonary Syphilis
2. Malignant disease of the lung.
3. Hydatids of the Lung.
4. Actinomycosis of the Lung

B. Less rare Conditions.

1. Malaria
2. Septicaemia
3. Broncho-pneumonia
4. Bronchitis
5. Pleurisy
6. Pulmonary Collapse
7. Bronchiectasis
8. Embolism
9. Asthma
10. Anaemia.

A. Rare Conditions.

1. SYPHILIS

Two varieties of syphilitic lesions fall to be considered in the differential diagnosis between this disease and phthisis: - syphilitic pleurisy and syphilis

of the lung, both of which are extremely rare.

a. Syphilitic Pleurisy

The extreme frequency of pleurisy in phthisis, and its rarity in syphilis has resulted in little attention being paid to its possible existence in the latter disease, especially at a time when all dry pleurisies are held by some to be tuberculous. Rare indeed as syphilitic pleurisy undoubtedly is, it should yet be borne in mind that every pleurisy in a syphilitic subject is not of necessity tuberculous.

Two types of syphilitic pleurisy have been described 1: a localised pleurisy secondary to syphilitic inflammation in the neighbourhood of the pleura, 2: a specific pleurisy in the early stages of syphilis.

1. LOCALISED SYPHILITIC PLEURISY

This first type was described by Moxon in 1870, who found post mortem in a case of syphilis that the lower half of the left pleura was covered with recent lymph, while the lung underneath showed grey fibroid changes. This localised syphilitic pleurisy may be dry or exudative.

The dry form is described by Lancereux, "A dry pleurisy, forming membranous adhesions in the form of thick bands is the necessary acolyte, so to speak, of syphilitic lesions, diffuse or circumscribed in the parenchyma of the lung. This pleurisy, whose special features recall the adherence of the syphilitic liver to

the diaphragm is manifestly peculiar to syphilis." We may regard this form as analogous to the localised pleurisy one finds in pulmonary tuberculosis around a patch of consolidated lung, and has been shown by Nikouline to accompany syphilitic lesions of the lung or syphilitic periostitis of the ribs.

Jacquin in 1884 recorded a striking case of the exudative variety. A man entered hospital with all the signs of caseating pneumonia, complicated by a pleurisy with effusion, which was diagnosed as tuberculous pleurisy. Fourteen days later, death occurred, when syphilis of the liver and gummata in the lungs were found. Some of the latter encroached on the pleura, which was thickened and contained two pints of bloody fluid, in which tubercle bacilli were absent.

Similar cases of exudative pleurisy associated with syphilis have been described by the elder Dieulafoy and by Gaucher, who reported a case where a sanguinous exudate with pulmonary syphilis cleared up on specific treatment.

2. SPECIFIC EARLY PLEURISY

The question of the existence of a specific early pleurisy in syphilis is by no means settled. Its existence had been held by Chantemesse and Vidal, Talamon, Preloriot, Rochon, Carra and Montseret. It is denied, however, by Landouzy who believes it is an early tuberculous pleurisy in those

persons weakened by syphilis, and that injections of the pleural fluid into the rat, reveal the true nature of the infection.

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Oettinger and Malloizel have tried to prove the specific nature of pleurisy in the secondary period of syphilis by examination of the exudate. They failed to find the Spirochaetae Pallida, but found lymphocytes in connective tissue, macrophages, and towards the end of the process cosmophile cells. These results are not conclusive.

b. Pulmonary Syphilis.

The recognition of pulmonary syphilis dates from 1777, when Bambilli recorded his classic case. An electuary was ordered for a phthisical patient in a hopeless state. By the mistake of the apothecary a syphilitic patient received the electuary to rub into his skin, while the phthisical patient got a mercurial ointment to take internally. Not aware of the mistake the "consumptive" swallowed the ointment, taking a lump the size of a nutmeg two or three times a day, with the result that "he was radically cured of his malady to the great astonishment of the doctor", who later learnt from the apothecary how it all came about. Later in 1853 Gamberini insisted that so-called cases of phthisis cured by mercury were in reality cases of pulmonary syphilis.

It is little wonder when we remember the disadvantages under which the older clinicians laboured, that syphilis of the lung as distinct from phthisis

should only have been realised by the error of an apothecary. Yet to-day, with all our modern methods, the diagnosis of pulmonary syphilis presents a problem, solved on so few occasions, that one suspects it is present more frequently than is supposed. In the history of medicine, where diagnosis has been difficult, disease has been absent. For long it was believed that certain parts of the body, such as the lungs and stomach, were not liable to be attacked by syphilis, but it has been gradually proved by careful pathological and clinical investigations that no tissue in the body enjoys immunity from the ravages of syphilis.

Fournier's dictum is at once suggestive and impressive:

"Given in an infant, an adolescent, or even in an adult, a pulmonary disease presenting the general and local symptoms of phthisis, there is always room to look for syphilis as the possible cause, and in these conditions the most elementary prudence should cause the physician to enquire into the patient's antecedents, and to seek for not only active or acquired syphilis, but for syphilis which is cured or is believed to be cured, and for hereditary syphilis.

If we examine the greater number of recorded cases of pulmonary syphilis, and ask how syphilis was suspected and discovered as the cause of a supposedly tuberculous lesion, the invariable answer is that it was by chance - by a fortuitous hazard, or by the quite unexpected appearance of some clear sign of syphilis, such as

exostosis, gummata, etc., Without this hazard, - I repeat and insist intentionally on this word - without this hazard the pulmonary disease would have continued to be mistaken for tuberculosis, and the result of such an error is clear.

It will not be allowed that the diagnosis of a disease so grave as pulmonary syphilis should be left to the mercy of a chance, of a hazard. It is a medical duty to anticipate this ~~cause~~^{chance}, and to discover syphilis before the appearance and without the addition of an accidental manifestation. This is quite simple if in every case of pulmonary disease we make a complete and sufficient enquiry into the antecedents of the patient for syphilis;— even for far off hereditary syphilis."

We will here consider:

- (a) Acquired syphilis simulating Rapid Phthisis.
- (b) Acquired syphilis simulating chronic phthisis.
- (c) Hereditary syphilis simulating phthisis.
- (d) Existence of Syphilis and Tubercle in the same lung?

(a) That acquired syphilis may simulate Rapid Phthisis is proved by Jacquin ²⁰⁷ who has recorded the classic observation of Girandeau on a case where a typical picture of galloping consumption, with rapid softening of the lung, nummular sputum, profuse sweats, hectic fever, and rapid emaciation, was diagnosed to be syphilitic by the absence of tubercle bacilli, enlarged inguinal and occipital glands, and ulcerations

of the vagina, with the result that the patient was cured in a few weeks by specific treatment.

(b) It is most usually, however, the chronic form of phthisis which is simulated by syphilis. The physical signs are due to the formation of localised gummata in the lungs, or of a more general sclerosis, or of an admixture of both. The softening of gummata leads to the formation of cavities, so that pathologically the syphilitic process simulates the stages of the tuberculous process. The sclerotic changes of syphilis will cause bronchiectasis, and are thus the homologue of fibroid phthisis.

The clinical signs of pulmonary syphilis will be gathered from the following cases taken from the literature.

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Cade and Savy verified at a post-mortem a diagnosis of syphilitic bronchiectasis, which they hold to be the most frequent lesion of tertiary syphilis, and often associated with laryngeal syphilis. The disease is fatal by the obstruction of the pulmonary circulation, which leads to cardiac failure. Even specific treatment is of little value, as fibrous tissue once deposited never resolves. Panas²⁰⁹ reports the case of a large cavity in the lung, the symptoms being cough, haemoptysis, fever and emaciation. The appearance of a syphilitic gumma in the eye led to a recognition of the disease, and to its cure by mercurial injections.

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Sergent (E) records the case of a woman of 32, whose parents were alive and well, as was also her husband, none of these having any sign of syphilis. She had history of cough for five years. There was congestion of the right apex, with dulness of the left, and tubular breathing. No tubercle bacilli were found, but Sergent diagnosed pulmonary tuberculosis, with cavitation and tendency to sclerosis. Three months later he found that the patient had contracted syphilis when six years of age, from a nurse; the initial lesion being on the sternum which was operated on by a surgeon for tuberculous osteitis. In view of this, he gave mercury, with the result that the patient was perfectly well in three months.

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Gaucher described a case where both apices were affected and arthropathies of both elbows existed. The patient had no traces of acquired syphilis, but had aborted on four occasions at the end of seven and eight months. At the back of one arm three squamous copper coloured papules were found arranged in a semi-circle, having the appearance of a syphilide. She was cured by specific treatment.

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Barthelemy had the case of a man, aet 38, who had been treated by several physicians for phthisis. The bacilli were absent, but auscultation showed areas of induration and softening, surrounded by congested tissue. The weight had fallen from 140 lbs, to 114 lbs, while the temperature was 38.5°C. in the morning, 39.3°C. at night. In giving creosote, Barthelemy noted

signs of syphilis on the skin and bones. On specific treatment the signs of the disease disappeared in three months, while at the end of a year the patient weighed 140 lbs.

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Lancereux reported the case of a woman who had suffered from apical disease for two months, with great dyspnoea and a clear expectoration, which contained yellowish lumps. Two years before she had suffered from sore throat for two months, and had lost two children who died of convulsions shortly after birth. She had partial destruction of the palate, and a long depressed cicatrix opposite the right sterno-clavicular articulation. The symptoms entirely disappeared after a months' course of specific treatment.

(c) Pulmonary lesions have been found in hereditary, as well as in acquired syphilis, and here also simulate phthisis, as is seen from the following cases.

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Gaucher and Dubousquet record the case of a girl of 8, whom they treated for some time as phthisical. The face and build were tuberculous, the patient was emaciated feeble, and wasted. There was constant cough, the temperature being 39, the pulse 140. The right apex in front and behind was dull, and cavernous breathing with rhonchi were heard in the supra-spinous fossa. Gross rales and crepitations were heard all over the chest. Next, a tumour appeared in the middle of the sternum, grew rapidly and soon attained the size of a small orange. On being opened it discharged a thick gelatinous fluid.

This raised the question of its being a gummata.

The teeth were all notched along the free border, and crossed by transverse lines. No traces of syphilis were found in the mother, while the father was not examined. The child was completely cured by specific treatment at the end of two months.

It was later found that the father had contracted syphilis three years before the birth of the child, as Dubousquet had treated him for specific periostitis of the tibia.

215

Latty reported a case of pulmonary syphilis in a child of 8, manifestly syphilitic, where the excavation of the pulmonary gummata was accompanied by gangrene foetid sputum, and by atrophy of the left side of the chest.

216

Terrien tells of a child admitted to hospital with pharyngitis, enlarged glands in the neck and subcrepitant rales all over the chest. Tuberculosis was diagnosed from the pharyngeal lesion, but the presence of Hutchinson's teeth and interstitial keratitis pointed to syphilis, especially as both parents had been treated for syphilis. The patient was cured by specific treatment.

217

Zuber records the case of a girl of 13, suffering from cough and dyspnoea, and showing lesions of the lung and pleura supposed to be tuberculous, also ulcerated gummata of the legs. She died a few days after admission from dyspnoea, just when the late diagnosis

had indicated specific treatment. Post-mortem, widespread syphilitic lesions were found in the lungs, pleura, spleen and kidneys. In this case, in the absence of hereditary stigmata and of knowledge of syphilitic infection, the diagnosis was based solely on the absence of tubercle bacilli and on the presence of cutaneous gummata.

(d) The question of the existence of syphilis and tuberculosis in the same lung is mostly theoretical, for in life it is impossible to know with any certainty if they even exist together, and absolutely impossible to say if their presence is separate and distinct, or, as was contended by Hiller²¹⁸ in 1882, if the phthisis has grown on a previously syphilitic lung. The classical case of the separate existence of these lesions reported by Conguenheim²¹⁹ cannot be accepted as definite, as it was noted prior to Koch's discovery of the tubercle bacillus. Potain²²⁰ and Sokolowski²²¹, however, have reported cases where the post-mortem showed phthisis to have super-²²²vened on an already syphilitic lung. William Porter in 1887 admitted the possibility of such a combination, but regarded it as clinically improbable. Nevertheless, as cases have been reported where undoubted phthisis underwent considerable improvement on specific treatment, it is still an open question whether phthisis occurring in an old syphilitic may not be aggravated by tertiary lesions in the lung, and whether this supposition should not be put to a therapeutic test. Certain it is that the supposed intolerance of phthisical patients to mercury

is a relic of the old treatment by salivation, and is not encountered in treatment by Hutchinson's small doses, which he recommends should be given when "local and unusual lung changes show themselves in those who are known to have suffered from syphilis."²²³

DIFFERENTIAL DIAGNOSIS.

This subject has been treated at some length on account of its importance, for it is probable that improved methods of diagnosis will make syphilis of the lung less rare. The diagnosis of the condition is a matter of extreme importance, for while pulmonary syphilis if left untreated will prove fatal, even the most helpless cases will respond to treatment. This emphasises the importance of a careful enquiry into the antecedents, and a search for signs of syphilis in the face, palate, teeth, eyes and ears, and for copper-coloured scars and syphilides on the body.

Syphilis usually attacks the middle lobe of the right lung, as was shown by Grandidier²²⁴ as early as 1875 from thirty cases collected from the literature of that day. He remarks "Where signs of infiltration or excavation are found in the middle lobe of the right lung the diagnosis of syphilis should be made without restriction even in the absence of all other manifestations." As we have seen from the preceding cases this rule is not absolute, and only constitutes presumptive evidence.

The differential diagnosis depends upon so few points that they are best tabulated side by side.

Pulmonary Syphilis	Pulmonary Tuberculosis
1. Tubercle bacilli are absent	Present
2. Usually affects right middle lobe	Right Apex
3. Dyspnoea very marked	Not marked
4. Fever not high.	Fever high
5. Little constitution disturbance, patient does not <u>look</u> so ill as lesions would indicate	Great
6. Signs of old syphilis present	Absent
7. Emaciation moderate.	Great
8. Yields to mercury.	No effect

2. MALIGNANT DISEASE OF THE LUNGS.

This condition is extremely rare, particularly the primary variety. It simulates phthisis by cough, fever, emaciation and haemoptysis. The various points are brought out in the following case of Sarcoma of the Lung, which the writer saw under the care of Dr. John Hay.

NEOPLASM INVADING THE LUNG

A. B. Marine Engineer, aet 29, complained of cough and shortness of breath. He was admitted to the Liverpool Hospital for Consumption, under the care of Dr. John Hay, and was there seen by the writer.

Family History was good and he denied syphilis or excess in alcohol. He had been troubled with the cough for four months, and recently had put up a good deal of sputum, which had been bloodstained for the past two weeks. He had lost a stone in weight, but had never suffered from night sweats, and enjoyed a good appetite. He complained of pain in the left side, and of slight pain at the base of the left lung. The thorax was well developed, but the movements of the left side were considerably diminished. There was no increase of vocal fremitus, but dulness was marked over the lower lobe of the left lung. Breath-sounds and vocal resonance were diminished but numerous moist rales were audible on inspiration and expiration. The cough was hoarse, husky, and not explosive. The sputum was dark, viscid, frothy and blood-stained, while tubercle bacilli were absent.

Larynx. The left vocal cord was paralysed, but laryngeal tugging was absent.

Praecordia. Cardiac dulness was normal, but there was also dulness to the right of the sternum below the inner third of the right clavicle. The heart sounds were normal, with the exception of a soft blowing systolic murmur over the aortic area, but there was no pulsation.

The temperature was normal, and there was no interference with swallowing. On admission the pupils and the radial pulses were equal, but ten days later Dr Hay noticed that the left radial pulse was smaller, and that there was slight oedema over the lower lobe of the left lung.

Patient became rapidly worse, suffering greatly from gnawing pain in the left side, and from paroxysmal dyspnoea, relieved by amyl nitrite. One morning fourteen days after admission, he became very breathless, cyanosed, and appeared in extremis. He sat up in bed for five minutes, labouring for breath, then fell back dead.

The post-mortem was performed by the writer six hours after death, and the following condition found:

The pericardial sac was distended with about a pint and a half of fluid arterial blood. There was a firm mass in the superior mediastinum in the region of the aorta. This extended into the posterior mediastinum, and completely surrounded the transverse portion of the aorta, growing up around its branches in the superior mediastinum. It completely encircled the innominate artery, the right subclavian and right carotid arteries, also the left carotid and subclavian arteries. The innominate veins and superior vena cava were lying on the anterior surface of the malignant mass, but not involved in the growth. The trachea was not infiltrated, and at its bifurcation the growth left the post-mediastinum and, encircling the left bronchus, narrowed its lumen and

passed into the left lung. A most interesting condition existed in the pericardial sac. On its parietal layer a warty excrescence about the size of a shilling was found in the upper part of the posterior wall to the left, at which point the tumour was distinctly felt behind the pericardium, which had become infiltrated. The valves were not diseased. The wall of the left auricle was infiltrated by a spongy mass, spreading from the point of entrance of the left pulmonary veins, which in this case entered by a common orifice along the lateral wall to within half an inch of the auricular appendage, and for one inch along the posterior wall.

The auricular septum was not affected, nor had the growth passed the auriculo-ventricular groove. In the diseased area of the left auricle a perforation was found situated immediately posterior to the point of entrance to the left pulmonary vein. This perforation admitted the tip of the little finger. The first portion of the aorta was dilated and lengthened. The left lung showed thickening of the pleura and old adhesions everywhere, except at the base. It was dark red, collapsed, and deeply injected. The lower portion of the upper lobe was invaded by the tumour, which branched in all directions and was of a creamy colour. The centre of the mass had undergone fatty necrosis, and was semi-fluid. The root of the lung was composed mainly of tumour, which had infiltrated the lymphatic glands in this region. The oesophagus had escaped, though some of the lymph glands in its neighbourhood were involved."

This case presented a symptom complex of extreme interest. Against tubercle were the symptoms, the absence of bacilli and of increased resonance in the solid area, which is explained by the constriction of the left bronchus. The systolic aortic murmur was caused by the dilation of the aorta between the aortic valve and the part constricted by the tumour. Of the distinction between aneurism and tumour, Dr Hay ²²⁵ writes.

"The systolic bruit over the aortic area, the dullness behind the manubrium, the diminution in the volume of the left pulse, and the paralysis of the left vocal chord, constituted a "symptom complex" strongly in favour of aneurism. On the other hand, the short history, the age of the patient, and the denial of syphilitic infection, the absence of pulsation and of diastolic shock, together with the presence of haemoptysis, suggested mediastinal tumour. With mediastinal tumour, however, one would have expected involvement of the veins rather than interference with the arteries."

DIFFERENTIAL DIAGNOSIS

This may be tabulated as follows:

Malignant Disease of the Lung	Phthisis
1. In secondary deposits there is history of previous disease	No history
2. Pressure signs are usually present	Not usually
3. Pleuritic pain is a late feature	Early
4. Bases generally first affected	Apices

Malignant Disease of the Lung	Phthisis
5. Prune juice sputum	Nummular sputum
6. Tubercle bacilli absent	Present
7. Progress is very rapid	May be rapid
8. Dyspnoea very great	Not marked

3. HYDATIDS OF THE LUNG

The first to give a full consideration to hydatids of the lung was Davaine ²²⁶ in 1860, who pointed out that most commonly there is but one hydatid present, generally in the inferior lobe of the right lung, that second in frequency is one cyst in each lung, but that it is extremely rare for two or more to exist in the same lung. This refers to primary hydatids of the lung, for as Dolbeau ²²⁷ showed in 1856 hydatids of the liver may attenuate the diaphragm and so enter the thoracic cavity.

Primary hydatid of the lung is extremely rare, as will be appreciated from the fact that Trousseau in his vast experience only encountered two cases. Of the 2721 ²²⁸ cases in the statistics of Davaine, Cobbold, Finsen, Niesser, Mosler and Thomas, the parasites involved the lungs and pleura in 10 per cent.

These cases present extreme difficulties of diagnosis, and most commonly pass for phthisis, gangrene, pyo- or hydro-thorax during life. The symptoms are pain,

cough, and shortness of breath. If the hydatid be large there is a localised globular bulging of the thoracic wall, which Trousseau considered to be pathognomonic of this condition.

The physical signs are dulness on percussion, with absence of breath sounds, while the surrounding area shows rales and rhonchi indicative of congestion or general bronchitis. The chief difficulty is to distinguish this condition from a localised pyo- or hydrothorax, but the area of dulness will be more irregular than in this condition.

4. ACTINOMYCOSIS OF THE LUNG

This is said to occur in 13% of cases of actinomy-
 cosis, and is to be distinguished from phthisis by a
 consideration of the following points. The disease is
 most frequent in farm servants, millers, and grooms who
 handle cereals. Nodules of actinomycolosis are to be
 seen in the mouth, jaws, and tongue, while in the lungs
 the signs are mostly basic. The lesions frequently
 extend to the chest wall, in which abscesses form. The
 main thing to be sought for is the ray fungus in the
 sputum or in the pus from abscesses.

B. LESS RARE CONDITIONS SIMULATING PHTHISIS

We will now consider certain conditions, which are
 less rare as simulators of phthisis than the foregoing.
 As however, they are more easily recognised, their
 consideration will take up less space.

1. MALARIA

The regular intermittent varieties of malaria - tertian, quartan and quotidian - present little analogy to pulmonary tuberculosis, but two varieties exist, the one in temperate, the other in tropical climates, which present difficulties of diagnosis.

(a) Aestivo-Autumnal Fever.

During the later months of the year in temperate climates an irregular aestivo-autumnal fever is found, which may simulate early phthisis. The patient complains of headache, cough, dyspnoea, and pains in the back, and is conscious of having lost weight. Dyspepsia is present, the appetite is poor, and vomiting after food has occurred. The temperature is irregular, the chart showing an evening rise of varying height, but the classical stages of the malarial paroxysm are absent. In children diarrhoea is frequent. The pulse is soft and rapid, and on auscultation sonorous râles are heard over areas of the lung.

Differential Diagnosis. The patient lives, or has lived in a malarial country, and has history of malaria. The expression is dull and tired, and the facies resembles that of typhoid. If the case be of old standing the skin and conjunctiva are tinted a dirty yellow, while in phthisis the conjunctiva is pearly white. The cough and symptoms of pulmonary congestion came on suddenly, prior to which the patient was well. The râles accompanying the breath sounds are more diffuse than

those found in early phthisis, are rarely limited to one apex, but are liable to affect different lobes alternately, or may come and go. Tubercle bacilli are absent the blood shows an absence of the reactions of tuberculosis, and of leucocytosis, while malarial parasites are present. The condition is cleared up by quinine.

(b) Pneumonic type of Pernicious Malaria. In the bad malarial districts of the tropics a pneumonic type of pernicious malaria may resemble acute tubercular lobar pneumonia. This grave form of the fever was first described by Baccelli²³⁰ who recognised it to be of malarial causation, and a condition quite distinct from ordinary pneumonia, or from this disease supervening on malaria. The disease is of sudden onset, there being great dyspnoea, painful cough, with sputum which shows fluid or clotted blood. Percussion reveals dulness over one lung, and on auscultation there are fine moist rales, with coarse, sibilant, sonorous rhonchi. Thayer considers the disease to be an active pulmonary congestion due to blocking of the capillaries by the parasites, in which case it resembles malarial cachexia of the brain.

This type of malaria may be distinguished from acute tubercular lobar pneumonia, or other pneumonic conditions, by the history of old malaria, the sudden onset the great collapse, the fast pulse and subnormal temperature, and the presence of 'crescent' bodies in the blood. The bacilli and blood reactions of tubercle will be absent, and the disease will react to large doses of

quinine and immediate change of scene. Any intermittent fever that resists quinine is not tuberculous.

2. SEPTICAEMIA

There are certain low forms of septicaemia, due to general infection by pyogenic organisms, which simulate early phthisis. There is a daily chill, rigor, and fever the temperature rising to 103° F. or 104° F. and followed by profuse sweats. The face is bile tinged, and the tongue is dark. There may be a history of a previous suppurating process in the body, and there is marked leucocytosis, which is not present in early phthisis.

3. BRONCHO-PNEUMONIA

The great difficulty of distinguishing between phthisis and broncho-pneumonia, arises from the fact that phthisis is broncho-pneumonia, due to the tubercle bacillus. The difficulty is greatest in deciding the nature of broncho-pneumonia, which follows measles and whooping-cough in children. As Osler ²³¹ remarks "there are many cases of broncho-pneumonia in children, which time alone enables us to distinguish from tuberculosis."

The following points assist the differential diagnosis:

1. True broncho-pneumonia should clear up by the end of three weeks. Tuberculous broncho-pneumonia does not clear up.
2. The symptoms of true broncho-pneumonia are more acute than are those of the tuberculous variety.

3. The fever is higher in true broncho-pneumonia.
4. The pulse is less rapid and more in accord with the fever in true broncho-pneumonia.
In the tuberculous variety the pulse is very rapid compared to the temperature.
5. Tubercle bacilli are not found in true broncho-pneumonia.
6. The pulmonary lesions are more evenly distributed in true broncho-pneumonia than in tuberculous broncho-pneumonia, where they are more localised at the apices.
7. The blood reactions of tuberculosis will be present or absent according to the nature of the case.
8. A tubercular patient does not look so ill as one with true broncho-pneumonia.

4. BRONCHITIS

Simple acute bronchitis presents no analogy to phthisis. The percussion note tends to be hyper-resonant loud wheezing rhonchi are distributed all over the chest, and there are no crepitant rales.

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Gee, however, has shown that in certain cases bronchitis tends to be unilateral, and there are certain cases of phthisis which are preceded by attacks of bronchitis, which are probably of a tuberculous nature.

The following points help to distinguish tuberculous bronchitis from simple bronchitis.

1. The attack lasts much longer.
2. There is a hectic temperature, night sweating and loss of weight.
3. The signs tend to be localised to one apex.
4. There is diminished "Tidal breathing".

5. There may be slight dulness and increased vocal resonance.
6. Crepitations may be heard.
7. If tubercle bacilli are present, the diagnosis is clear from the start, but in the great majority of cases these are not found at this stage, and the diagnosis by some of the methods of blood reactions should be resorted to.

5. PLEURISY

When we remember that pleurisy invariably accompanies phthisis, that it is an early manifestation of the disease, and that the majority of all pleurisies are tuberculous, the importance of the early recognition of this condition cannot be over-estimated.

We suspect phthisis when in the course of an acute pleurisy, we heard crepitant rales at the apex, which however may be due to the congestion of the lung above the pleural effusion, and also when the convalescence of the patient is slow.

The following table gives the chief points in the differential diagnosis.

Tuberculous Pleurisy	Simple Pleurisy
1. Disease is more chronic.	More acute
2. Hectic temperature	High temperature
3. Effusion may be bloody	Usually purulent
4. Injected into guinea-pig causes tubercle.	Does not
5. Effusion contains lymphocytes	Mostly polynuclears

Tuberculous Pleurisy	Simple Pleurisy
6. Methylene blue is slowly absorbed from the pleural cavity.	Rapidly absorbed
7. Excess of chlorides in urine	Not marked.
8. Blood reactions of tubercle are present	Absent.

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Fernani believes in the following test (Mayer's) to determine the presence of polynuclear leucocytes in the blood, which is in favour of simple or purulent pleurisy. Two drops of blood are collected in a pipette and diluted with distilled water until the red colour disappears. A 50% solution of tincture of guaiac is added, and if there is polymorphonuclear leucocytosis a blue ring appears.

On this Lindsay writes:

6. PULMONARY COLLAPSE

This is to be diagnosed from phthisis by the history of its onset, usually following acute bronchitis, broncho-pneumonia, tumours or aneurism pressing on the bronchi, or foreign bodies in the bronchi.

There is no fever, and dyspnoea is the main symptom, which is absent in phthisis.

There is local retraction, weak breath sounds, and diminished vocal resonance. Collapse affects the bases more frequently than the apices.

7. BRONCHIECTASIS

This may occur late in phthisis when there is no doubt as to the diagnosis. As due to other causes it is diagnosed by the absence of tubercle bacilli in the sputum, which is very foetid, and is put up at intervals. The physical signs are those of a cavity, and are usually basic in site.

8. PULMONARY EMBOLISM

The acute onset, the great dyspnoea, the presence of mitral disease, thrombosis, or recent delivery, serve to distinguish this condition from phthisis.

9. ASTHMA

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On this Lindsay writes:

"A limited and retrocedent tubercular lesion of one apex is attended by fibroid changes and bronchiectasis. Attacks of dyspnoea occur, and the case is mistaken for one of asthma.

In such cases the differential diagnosis is usually easy. The history of the two types of case is quite typical. There is more or less debility or emaciation. The temperature may assist. Tubercle bacilli may be found in the sputum but are often absent. Localised signs at one or both apices, however slight, can be recognised.

CHAPTER X.

The bronchiectatic changes in the neighbourhood of the apical lesion may produce misleading physical signs.

It should be a rule to examine the sputum for tubercle bacilli in cases of supposed asthma, and to exercise special care when the patient is young and when debility or emaciation is present."

10. ANAEMIA

Anaemia is a symptom of phthisis, but there are several other conditions which may have to be excluded as the possible cause - the chief being: syphilis, malaria, septicaemia, toxæmia, Bright's Disease, chlorosis, pernicious anaemia, and malignant disease.

CHAPTER X.

THE INFLUENCE OF SYPHILIS, MALARIA, AND DIABETES
ON PHTHISIS.

1. The Influence of Syphilis on Phthisis.

In dealing with the influence of syphilis on phthisis, it is necessary to distinguish between recent and far off venereal infection, as the prognosis depends to a large extent on this differentiation.

A. Recent Infection.

By recent infection we mean syphilis which has not yet passed the secondary period, and this may complicate phthisis in two ways: - (a) a phthisical patient may become syphilitic, or (b) a syphilitic patient may develop phthisis. In both cases the prognosis is extremely grave.

(a) A phthisical patient becoming syphilitic.

All authorities are agreed that such a dual infection presents a prognosis of extreme gravity, governed, however, by two factors: - the state of the pulmonary lesion and the virulence of the syphilitic infection.

The case is serious for reasons direct and indirect. It may usually be assumed that a phthisical patient who contracts syphilis has not been living in the manner generally recognised to be essential for the cure of the pulmonary lesion. Further there is added to the impairment of function and the general toxaemia of tuberculosis, the systemic infection of syphilis, manifest in anaemia, debility, and depression. When we remember the ravages

of syphilis in the healthy subject, it is little wonder that in phthisis it should inhibit the resistance, light up the disease, and hurry on the fatal termination. Such is the view generally enunciated by competent authorities.

On the other hand, Monteverdi ²³⁵ believes that syphilis exercises a favourable influence on phthisis, and Emile Sergent ²³⁶, without going so far, holds that this combination has been regarded with undue gravity, so far as prognosis is concerned.

In view of such diverse opinions, it is obvious that there is some variable factor in the case, which more or less governs the prognosis; and this is apparently the state of the pulmonary lesion. In the first place, it is comparatively rare for a phthisical patient to become syphilitic, for if his phthisis is sufficiently advanced to be recognised, he is usually careful to avoid the chance of such infection.

In the struggle between the organism and the tubercle bacillus, the decisive advance for either side takes place in the early months of infection, and for a patient whose apex is in a state of tuberculous catarrh, where rest and strength might turn the balance of repair in his favour, for such a one to become syphilitic is utterly disastrous. So all authorities, even those who uphold the idea that syphilis may ultimately in some obscure way assist in repair, are agreed that secondary syphilis aggravates all the symptoms of phthisis.

If now we turn to Sergent's ²³⁷ cases, we find that in two, personally observed by himself, there was a recovery from phthisis after 14 and 16 years respectively,

notwithstanding the fact that they had also acquired syphilis. There is one striking point about both these patients, and that is, they developed syphilis two and four years respectively, after the commencement of their phthisis. Now phthisis of two and four years' standing is chronic phthisis, and it is easy to see that such cases becoming syphilitic, and where the syphilis is treated, stand a chance of recovery, as the pulmonary lesion is sufficiently under control to enable them to weather the lowering influence of secondary syphilis.

Furthermore, such patients would avoid the excesses which resulted in syphilis, and which, quite apart from this disease, must have exercised a baneful influence on their phthisis.

Not only so, but a phthisical patient becoming syphilitic, the efforts to combat the pulmonary disease will be redoubled.

A study of those cases reported in the literature leads one to the belief that the governing factor in the prognosis is the age and state of the pulmonary lesion - the more recent the phthisis the more grave the prognosis.

(b) A Syphilitic who becomes Phthisical.

We here consider the course of phthisis commencing in a patient with recent syphilis, that is in one who has not yet passed the secondary stage, for it is obvious that phthisis will run a very different course, according to whether it starts in a patient debilitated by the severe toxæmia of secondary syphilis, or in one who has established by treatment a resistance to venereal disease

of years past.

The following cases seen by the writer illustrate the gravity of phthisis occurring early in the course of syphilis.

CASE I.

A lad, aged 17, contracted syphilis in 1904. He was seen early and put on Hutchinson's pills. In spite of treatment he suffered severely from secondary symptoms and at the end of the year in 1905, although mercury was pressed, he had ulceration and destruction of the palate, a lesion which may be considered to belong to Hutchinson's "intermediate period." At this time he was in extremely low health, and developed cough, and tubercle bacilli were found in the sputum. He now rapidly went down-hill, and when seen by me in 1907 had both lungs involved in every lobe. He died two months later.

CASE II.

E.J., aet 29, third officer on trading steamer, complains of cough, sweats, and loss of appetite. He has been at sea since 17, and there is no family history of phthisis.

Five months ago he contracted syphilis, and three months later his appetite disappeared, and he commenced to suffer from cough and sweats, both of which were worse at night. He is certain that prior to this time he never suffered from any of these symptoms.

At date of examination, two months after the commencement of symptoms of phthisis, there is complete consolidation of the right upper lobe, with bronchial

breathing, accompanied by medium moist râles, with increased vocal resonance and fremitus. Over the left apex behind, pleuritic friction, broncho-vesicular breathing, and crepitations are heard. The pulse is 90.

These cases are examples of Rapid Phthisis occurring early in syphilis. A similar condition has been reported by Renon ²³⁸, where phthisis developed nine months after infection with syphilis, while Sergeant ²³⁹ mentions three cases of galloping phthisis, commencing during the secondary period, but also two cases, where the pulmonary disease apparently did not gain virulence from the pre-existing syphilis.

It would appear that the prognosis of phthisis appearing early in syphilis is in general one of extreme gravity, yet modified by two factors. Firstly, by the virulence of the syphilis, for as Hutchinson ²⁴⁰ remarks "A very large number of those who suffer from constitutional syphilis pass through the secondary stage with very little disturbance of general health. They scarcely know that they are ill." It is certain that in such a one the prognosis will be more hopeful than in the debauched, languid, anaemic subject who is saturated with the syphilitic poison. Secondly, an equally decisive factor in prognosis is the age of the syphilis, for as in a phthisical patient becoming syphilitic, the more recent the phthisis the more grave the prognosis, so in a syphilitic becoming phthisical, the more recent the syphilis, the more serious appears the ultimate issue.

B. Far off Syphilis.

A good deal of the difference of opinion regarding the course of phthisis in a syphilitic has arisen from the failure to distinguish between syphilis acquired a short time before the patient became phthisical, and syphilis which has spent its force years before. In recent syphilis when phthisis breaks out, the prognosis is always one of extreme gravity, but it would seem that if the syphilis be far off in time from the outbreak of phthisis, that the latter disease runs a peculiarly chronic course. This fact is illustrated by the following cases seen by the writer.

CASE I.

F.M., aet 46, complains of cough and sweats. No family history of phthisis.

At the age of 24 he contracted syphilis. For the past two years he has suffered from cough. For the last year he has lost weight, and five months ago began to expectorate. Two months ago night sweats appeared, with loss of appetite and constipation.

He shows chronic hypertrophic osteo-arthritis of the fingers, with slight enlargement of the internal condyles of the femur. The ring and little finger of the left hand are quite blue in colour. If they are rubbed they become red for a time, but soon return to the venous tint. This and the condition of the pulse indicates arterio-sclerosis.

The sputum contains tubercle bacilli.

The right apex shows diminished "Tidal breathing,"

is dull on percussion, and gives bronchial breathing with an occasional dry click.

CASE II.

A.H., aged 46, a native of Finland, and an officer in the mercantile marine, was seen by me on January 1st, 1908, complaining of cough, headache, loss of appetite, and pains in the legs.

Family History. - His mother died of phthisis, aged 47, when he was eleven years old, and some years later his father died of heart failure. He has one younger brother, who has always enjoyed good health.

Patient's History. - When 16 years of age A.H. went to sea, and has followed this profession ever since, having risen to hold a master's certificate. At the age of 22 he contracted syphilis, and was treated by inunction, being given fifteen frictions with mercuric ointment, under which the primary lesion disappeared, and the patient believed himself to be cured. Five years later, however, at the age of 27, his skin became covered with blotches and papules, about which he consulted an English doctor in the United States, who put him on mercury pills for three months. The skin lesions disappeared, and he has not seen any manifestations of syphilis since.

At the age of 42, twenty years after contracting syphilis, he began to feel generally weak, was easily tired, and the appetite was diminishing. He continued in this state off and on for some time, and two years later, when 44 years of age, he further noticed a difficulty of walking in the dark, which he describes "as if I was putting my foot on a step which wasn't there;" and

at this time he also commenced to suffer from pains in the legs, and from slight cough in the morning. Being in Finland at this time, he consulted a physician in Helsingfors, who told him that he was suffering from a nervous disease, and advised a course of mercury. Two days later, with regard to the loss of appetite, which had now increased to nausea and vomiting after breakfast, he saw another physician, who found tubercle bacilli in the sputum, and prescribed residence in a sanatorium. He was extremely surprised to find himself phthisical, and the same day consulted a professor of the University of Helsingfors, who confirmed both diagnoses. At this time the patient felt very weak, and entered Nummela Sanatorium, where he received ten injections of mercury, on the instructions of his physicians. He resided at Nummela for four months; on leaving, his weight had increased from 59 kilos to 75 kilos, the nausea, sickness, and cough having entirely disappeared. Five months later his weight rose to 84 kilos.

Six months ago the patient noticed that the cough, which at the commencement of his illness had attracted little attention, was now returning with increased frequency, and for this he saw a doctor in Germany, who prescribed the iodides of sodium and calcium, from which, however, no benefit was derived.

Present condition. - The patient is a well developed man of 46, weighing 76 kilos. He complains of cough, headache, loss of appetite, and pains in the legs. Temperature is normal.

The cough is most frequent in the morning, and his sleep is undisturbed by this. During the whole course of his illness he has never suffered from night sweats nor from fever. The chest is well developed, and there is no diminution of "tidal breathing" on either side. Percussion and vocal fremitus were normal throughout. On auscultation wheezing rhonchi were heard during expiration over both lungs, but in only one spot could any alteration in the breath sounds be detected, and this in the upper lobe of the left lung over an area corresponding to a dollar, situated in the middle of the anterior maxillary fold. Over this very limited area the breathing was bronchial, vocal resonance was increased, but no accompaniments could be heard. The sputum has been white and frothy for the past week, but prior to this was greyish white. A small piece of solid substance when examined contained large quantities of tubercle bacilli.

The appetite is poor, he has nausea after food, and is constipated. The liver is not enlarged.

The heart is normal in size and function. The radial arteries appear thickened and fibrous. The pulse is regular, frequency 70, the wave is full and well sustained, but the arterial tension is high. The urine contains no abnormal constituents, The spleen is not enlarged, but the inguinal and posterior occipital glands show adenopathy.

The patient is an intelligent man, takes considerable interest in his case, and has the nervous temperament. He has now suffered from pains in the legs for two

years intermittently, and describes the sensation as a dull pain, with now and then sharp twitches running from the hip to the knee, sometimes in the reverse direction. He has still the uncomfortable sensation when walking in the dark of being uncertain of feeling the ground, and his feet are often numb and cold. Both knee-jerks are absent, but neither paraesthesia, allocheiria, nor Romberg's sign is present. The gait is not ataxic, the legs are well developed, and hypotonia cannot be elicited. The left pupil is smaller than the right, and both eyes show the Argyll-Robertson pupil. *a few years ago tubercu-*

There is little doubt but that this patient has suffered from chronic phthisis for four years, and it appears that the pulmonary lesion of which a small part is now apparent in the left upper lobe, has its centre at the root of the left lung, possibly in enlarged bronchial glands, which by irritation of the sympathetic chain have brought about the contraction of the left pupil. Recently the patient has had chronic bronchitis which, from the absence of fever, may have been a super-added non-tuberculous infection. In any case this cleared up in a few days. One may here touch for a moment on the purely theoretical possibility of the phthisis having supervened on a tertiary lesion in the lung. In a patient who has had syphilis, and who in after years becomes phthisical, there is always the possibility of a dual infection, although diagnostic certitude is impossible. On this chance Sergent and Hutchinson advise that such patients be put on mercury, and this practice seems worthy of trial. In this case, however, it is improbable that a

tertiary lesion existed, as there were no pulmonary symptoms prior to the onset of phthisis, and as the presence of locomotor ataxia apparently precludes the formation of gummata. At the same time, if there still be syphilis in the system, such treatment would be beneficial.

The patient has all his life been a great milk drinker. He states that in no other country he has visited does milk enter so largely into the general diet as in Finland, which is chiefly a pastoral country and ships seven thousand casks of butter every week to Britain. He informs me that up to a few years ago tuberculosis was rife among the cattle, but that now there is an efficient inspection of milk and butter by Government chemists and bacteriologists. As a drinker of milk he has taken it all over the world. At the same time, as it might naturally be supposed that a life at sea would have precluded all prolonged contact with phthisis, it must be stated that six years ago, two years before the onset of phthisis, he began to carry a number of consumptives for two months in the summer on a cruise round the Mediterranean, and was thus for two summers in succession in contact with tuberculosis.

These cases seem to indicate that once the secondary period is passed syphilis does not appear to exercise a malign influence on phthisis. Thus Sergeant ²⁴¹ records two cases which became phthisical three and four years respectively after contracting syphilis and where the pulmonary lesion assumed a chronic form, and five similar cases where the phthisis appeared from thirteen to thirty four years after syphilis. To explain such cases Sergeant ²⁴²

has promulgated the theory of the "terrain syphilitique", which he describes as a "state of body resulting from old and worn-out syphilis, and which can be transmitted to descendants." He considers that this state is favourable to the development of tuberculosis, but that, "if tuberculosis is very frequent in old syphilitics, it is particularly torpid and benign in such subjects." Until it be statistically proved that the incidence of phthisis in old syphilitics is greater than among normal persons of the same age, the predisposition of old syphilitics to pulmonary tubercle will remain theoretical. On the other hand, Landouzy, Stieffel, and others have also held that tubercle in the subjects of old syphilis runs a peculiarly benign course, and the cause of this beneficial tendency to fibrosis of the pulmonary lesion is a question of extreme interest.

Handford ²⁴³ considered that the fibroid tendency of tubercle occurring in subjects of arterio-sclerosis was due to the high blood pressure and not to the sclerosis. Landouzy ²⁴⁴ holds it due to the sclerosis, and Sergent ²⁴⁵ believes it is caused by sclerosis, aided by a peculiarly active reaction to inflammation, which is one attribute of the "terrain syphilitique."

In the case under consideration phthisis appeared twenty years after the syphilitic infection, and is running a chronic and benign course. If we look to evidences of sclerosis outside the lung, these are to be found in the condition of the arteries, although there are no signs of aortitis or nephritis; and also in the condition of the spinal cord. It is little wonder, then, in an

area of the lung where the tissues not only share the general incitement to sclerosis, but are also subject to the irritation of the tubercle bacillus, that fibrosis should be active. Further, there is little doubt but that the active hyperaemia in the pulmonary lesion, present during "periods of high blood pressure," will also augment this process. Thus syphilis, one great cause of arterio-sclerosis, whereby life is shortened, affords a slight reprieve from a disease which might otherwise have been fatal. With regard to the course of phthisis following syphilis, it appears to be true that the more ancient the syphilis the more hopeful the prognosis.

2. The Influence of Malaria on Phthisis.

During last century there was a belief that malaria or syphilis were antagonistic to phthisis. Such was held by Boudin²⁴⁶. It is difficult at first to see how these ideas of malaria or syphilis, acting as a cure of phthisis, came into being, but it is probable that in the case of malaria at least it arose from the benefit of the enforced rest of convalescence on a chronic case of phthisis. Thus the writer saw a man who has suffered from chronic phthisis for the last twenty years. Twelve years ago, his phthisis appeared to be particularly bad, with cough, expectoration, and fever. On going to the Sierra to recruit, he acquired severe malaria, for which he was sent to hospital, to be treated by rest in bed, quinine, and tonics for a month, after which the symptoms of phthisis appeared to have vanished for a time.

When malaria occurs in the ordinary course of phthisis,

it is easily recognised and treated, as the symptoms of phthisis are eclipsed for the time being by the inter-current disease, but in patients with the dual affection presenting themselves for the first time, the diagnosis is more difficult, for the malaria apparently augments the gravity of the pulmonary condition. Geppener ²⁴⁷ has described a case of the two diseases, and Thayer ²⁴⁸ reports this combination to be frequent along the Eastern States of America. Marchiafava ²⁴⁹ believes chronic malarial fever with cachexia to predispose to tubercle. Certain it is that in the South of Spain malaria ranks with syphilis as the forerunner of phthisis.

3. The Influence of Diabetes on Phthisis.

Phthisis is liable to occur in the course of severe or of mild diabetes.

A patient who has suffered for months from diabetes, with polydipsia, polyphagia, and polyuria, drinking it may be litres of fluid per diem, and passing hundreds of grams of sugar in the urine, is suddenly discovered to be tubercular. The disease is insidious in its onset, simulating a simple chronic bronchitis which comes and goes, never clearing up. Fever and night sweats may be absent, and the sputum but slight in amount and confined to the morning. The patient's strength now rapidly declines, he becomes thin, the sputum takes on the characteristic appearance, and abounds with tubercle bacilli. In spite of its slow and insidious onset, the pulmonary lesion, when once declared, runs the course of Rapid Phthisis, destroying life in a few months.

In other cases, however, the diabetes is milder, - the amount of sugar small, the appetite and thirst increased but not excessive, eczema, gingivitis and boils are absent - but these also are liable to become tubercular, in fact lung mischief may bring a patient for consultation, who is unaware of having diabetes. Hanot²⁵⁰ has recorded the case of a diabetic, who became tubercular, yet who only passed a fifteenth of a gram of sugar per diem. In these cases also, phthisis comes on insidiously, by attacks of bronchitis off and on for months, a little sputum in the morning, gradually getting thicker and it may be tinged with blood, by the appetite, once increased, failing, and lastly by the gross signs of Rapid Phthisis.

It is thus clear that pulmonary tuberculosis is not exclusively the concomitant of intense diabetes, or the termination of diabetic cachexia, as it frequently occurs in robust patients, in whom glycosuria is slight or even overlooked. At the onset of the tubercular infection the clinical condition alters for the worse, and a patient who has supported for months or years the drain of a glycosuria, more or less intense, gets weak and loses weight. It may be treatment is blamed for the change, while the treacherous bronchitis escapes notice, whereas in reality the patient is but showing the rapid emaciation of phthisis.

Rapid Phthisis is not the only form of tuberculosis which attacks the diabetic patient. Dieulafoy²⁵¹ reports a case where death occurred in a few weeks from acute tubercular broncho-pneumonia, and Letulle²⁵² had a case

of acute military tuberculosis in a man of thirty six who had suffered for years from diabetes. Nor, on the other hand does the variety of diabetes, - nervous, traumatic, hepatic, or pancreatic - alter the liability to tuberculosis, for the case is reported of a boy of fifteen who developed traumatic diabetes as the result of a railway accident and died of Rapid Phthisis. ²⁵³

It was once supposed that haemoptysis was less frequent in the tuberculosis of diabetes than in chronic ulcerative phthisis, but this view is not borne out by recorded cases, and haemorrhage may occur at the beginning or end of the disease, from pulmonary congestion or from ulceration of a vessel.

The prognosis in the dual disease is one of extreme gravity, irrespective of the stage of the pulmonary lesion. Jacoud ²⁵⁴ wrote "I have had many occasions of studying this form of phthisis, but I have never seen a single case where a favourable prognosis could be given." Lasèque ²⁵⁵ held a more optimistic view, but by cruel irony he died of the two diseases.

As to prophylaxis, every diabetic patient should be told of the grave risk and the serious consequences of infection, and must lead an absolutely hygienic life, avoiding to the last detail all sources of danger.

The Treatment of Diabetic Phthisis.

In treating diabetic phthisis, it has been considered necessary to treat the phthisis at the cost of the diabetes, but Thorspecken ²⁵⁶ has recently shown that such treatment is against the best interests of the

patient, and that in many cases the pulmonary lesion commences to improve once the urine has been rendered sugar free. The diet of such a patient should then be carefully watched to find the limits of assimilation.

The first observer of the curious deformity of the fingers known as Pulmonary Hypertrophic Osteo-Arthropathy was Hippocrates, ²⁵⁷ who remarked of the nails in phthisic patients, "Tabidis unguis contractanter - tabidis unguis adunci," but this clinical observation was forgotten until Pigeau ²⁵⁸ drew attention to it in 1832, after whichrousseau ²⁵⁹ described it in the following year. In 1849 Marie and Souss-leits ²⁶⁰ published their classical account of the phenomenon.

The following is the description of Pulmonary Hypertrophic Osteo-Arthropathy, as observed by the writer ²⁶¹ in a child of six, where a diagnosis of Rapid Phthisis was made. We were first advised of the very extreme rarity of this phenomenon in children by Dr George A. Gibbon, who kindly wrote us on the case, and a perusal of the French and English literature leads us to the further belief that not only is this the youngest recorded case of the disease, but also the first instance to be noted where it occurred in conjunction with rapid pulmonary tuberculosis. As seen from the appended plates, the osseous system presents an absolutely typical picture of pulmonary hypertrophic osteo-arthropathy. The frontal eminences are well marked, but the superior maxillary bones are normal. (Plate I.) In the upper extremities there are typical abnormalities of the fingers. These

CHAPTER XI.

THE RELATION OF PULMONARY HYPERTROPHIC OSTEO-
ARTHROPATHY TO PHTHISIS.

The first observer of the curious deformity of the fingers known as Pulmonary Hypertrophic Osteo-Arthropathy was Hippocrates,²⁵⁷ who remarked of the nails in phthisic patients, "Tabidis ungues contrahuntur - tabidis ungues adunci," but this clinical observation was forgotten until Pigeau²⁵⁸ drew attention to it in 1832, after which Trousseau²⁵⁹ described it in the following year. In 1889 Marie and Souza-Leite²⁶⁰ published their classical account of the phenomenon.

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appear lengthened compared with the size of the hand, the proximal and middle phalanges are normal, but the distal phalanges of all the digits are curved antero-posteriorly and thickened, the nails being large and rounded, growing beyond the pulp laterally and over the tip of the finger. They each resemble "a flattened drumstick," to quote Marie's ²⁶² picturesque description (Plate II.) The deformities in the lower extremities are more marked (Plate III, Fig. 1). There is great enlargement of the condyles of the femurs, particularly of the internal condyles, which stand out prominent. The kneecap is not enlarged, but is carried out in front of the condyles. The whole articulation appears enlarged, and but for the angularity of the deformity and the absence of fluid one is forcibly reminded of Charcot's joint by the large mass standing out from wasted muscles. The tibia and fibula are not enlarged, but the hypertrophy of the internal condyle has brought about a condition of genu valgus, and has produced a compensatory concave inward curve of both tibia. The terminal phalanges of the toes present a similar deformity to that of the fingers, and equally well marked. (Plate III).

The etiology and pathology of this condition are quite unknown. Thayer's ²⁶³ collection of fifty five cases from the literature showed - forty three following pulmonary affections, three following heart disease, three following syphilis, two following chronic diarrhoea, one following spinal caries, and three from unknown causes.

Trousseau ²⁶⁴ noted that it was most frequent

in patients showing the second and third stages of pulmonary tubercle, and that it seldom exists in abdominal tuberculosis, unless a pulmonary lesion is also present. He also observed it, however, in bronchitis, empyaema, emphysema, hydatid of the lung, and in asthma, but considered it mainly to be a diagnostic sign of phthisis. "It must be remembered, however, that it is principally in cases of phthisis that it is met with, and that the curving of the nail is the more marked, the more advanced is the stage of phthisical disease."

The same physician states that while in most cases the onset of the deformity is slow, there are some where it is produced with great rapidity, "the patient suffering pain from the change that is going on."

Arnold ²⁶⁵, Marie ²⁶², and Thomson ²⁶⁶ have described the lesions as being due to hyperostosis, osteophytes being deposited on the surfaces of the bones in response to chronic irritation of the periosteum or the marrow by toxins in the blood, absorbed from the pulmonary disease. Thorburn ²⁶⁷ considers it to be a benign type of osseous and articular tuberculosis. Certain cases have been recorded by Batty Shaw and Higham Cooper ²⁶⁸, Florard ²⁶⁹, and Decloux and Lippmann ²⁷⁰, where the most careful examination failed to discover any pulmonary lesion. In view of this, it is of interest to note that in 1902, before the Medical Society of the Paris Hospitals, Thoinot and Dellamarre ²⁷¹ showed a patient with classical deformities, in whom ordinary examination had suggested the absence of any abnormalities in the lungs, but where the X-rays revealed the presence of an opaque mass in the

middle lobe of the right lung, which they suggest may have been a hydatid cyst. Béclère, who made the X-ray examination, said that in these rare cases where no lesion was found, the examination was probably at fault, and were X-rays used it was probable that a profound lesion would be discovered. Certain it is that a deep lesion may exist in either lung and defy diagnosis by ordinary methods.

Much experimental work is required on this subject, but it may be a demonstration will be forthcoming that this hyperosteitis is due to the stimulation of the osteophytes by the toxins of tubercle. Marcozzi²⁷² in a recent work on the actions of the toxins of the tubercle bacillus, which, if confirmed, will throw considerable light on the whole pathology of tuberculosis, has stated that he has observed inflammatory and hypertrophic phenomena of the cellular elements of the testicle entirely due to the stimulus of diluted toxins in the blood. If we grant the existence of toxins in the blood capable of producing inflammatory and hypertrophic phenomena, it is not strange that their influence should be most manifest in those situations where the blood flow is greatest. It is one of the paradoxes of physiology that the volume of blood in the capillaries is greater than that in the arteries, and if the capillary stream is slow, the parts supplied show the greater metabolism. The terminal phalanges are nourished by a circulation which is purely capillary, and whose action on the metabolism of these tissues is characterised by two facts - firstly, by the appearance of the primary ossification centres of the

distal phalange in the upper extremity at the ninth week of foetal life, in the lower extremity at the twelfth week, and which further have been shown by Lambertz ²⁷³ to be the first of the phalangeal centres to ossify; secondly, by the growth of the nail during life. Again, at the lower end of the femur, while growth is proceeding, the ossifying cartilage is supplied by capillaries from the perichondrium, periosteum, and from the medullary arteries, being the largest and most vascular ossifying cartilage in the body. The hypothesis suggested here is that in the presence of tuberculous toxins in the blood, capable in certain dilutions of inducing inflammatory phenomena, the osseophytes in those areas are stimulated to action by the amount of toxin with which they are brought into contact, through the large capillary blood supply, whereby there results an excessive and pathological formation of bone.

14. Urinary complications.

15. Mammary gland.

Tuberculosis of the larynx is a frequent complication of all cases of phthisis, occurring in a third of all cases. It is most usual in advanced cases, although it is found quite early in the disease, and may in cases precede the pulmonary lesion.

The first symptom is hoarseness of the voice, and soon the cough loses its explosive element, becoming prolonged and wheezing. Now eruptions of the disease may give rise to laryngeal oedema. Swallowing may be painful or impossible, especially when the epiglottis is

CHAPTER XII.

THE COMPLICATIONS OF PHTHISIS

1. Phthisis Laryngea.
2. Pneumonia.
3. Pleurisy.
4. Acute Miliary Tuberculosis.
5. Gangrene of the Lung.
6. Tuberculosis of Bronchial glands.
7. Pneumothorax.
8. Phthisical Diarrhoea.
9. Fistula in ano.
10. Thrush
11. Pityriasis vesicolor.
12. Swelling of the feet.
13. Meningitis.
14. Urinary Complications.
15. Mammary gland.

1. Tuberculosis of the larynx is a frequent complication of all cases of phthisis, occurring in a third of all cases. It is most usual in advanced cases, although it is found quite early in the disease, and may in cases precede the pulmonary lesion.

The first symptom is huskiness of the voice, and soon the cough loses its explosive element, becoming prolonged and wheezing. Exacerbations of the disease may give rise to laryngeal stridor. Swallowing may be painful or impossible, especially when the epiglottis is

involved, preventing its proper closure during swallowing, so that food may be coughed into the posterior nares.

When huskiness of the voice raises a suspicion of the onset of this condition, and the larynx is found to be swollen, the treatment is to ease the cough with opiates, and apply a mustard blister to the throat, to relieve the congestion.

If the larynx is infiltrated, use a laryngeal spray of 5% Menthol in Paroleine, and if ulcers be present they must be brushed with a 20% solution of lactic acid.

If there be pain on swallowing, this may be relieved by gargling or spraying the throat with a $\frac{1}{2}$ % solution of cocaine.

The patient may need tracheotomy.

2. Pneumonia.

This is a frequent termination of a case of chronic phthisis. The form is either lobar pneumonia or bronchopneumonia, and it is probable that the tubercle bacillus is usually the exciting cause.

3. Pleurisy.

Pleurisy of some description is the invariable accompaniment of phthisis. A dry pleurisy often occurs early, and is a useful process, for the adhesions ensure rest to the lung, and prevent the later appearance of pneumothorax. Pleurisy with effusion usually develops later in the course of phthisis. The effusion may be serous, but is more usually bloody or purulent. The effusion may be encapsulated by adhesions, which adds greatly to the difficulty of diagnosis.

4. Acute Miliary Tuberculosis.

This arises from the general infection of the blood stream with tubercle bacilli. In a few days the lungs may become riddled with thousands of minute grey translucent tubercles, which rapidly kill the patient.

5. Gangrene of the Lung.

This occasionally occurs, being due to necrosis of the fibrous tissue around a cavity.

6. Tuberculosis of Bronchial glands.

As we have shown elsewhere, this is a frequent complication of phthisis, especially in children. It is a disputed point if it is secondary to the pulmonary disease, or is present before the latter commences. Probably this last view is correct. As a rule no definite symptoms are produced, unless the glands are large, and suppuration has occurred, when pressure symptoms result. Pressure on the trachea may produce tracheal stridor, or there may be pressure on the left recurrent laryngeal nerve, or on the sympathetic chain, which will cause an alteration in the pupil. Should suppuration occur the glands may burst into the larynx or trachea, or very rarely into the pulmonary artery or the aorta.

Should there be tuberculosis of the bronchial glands, without well marked pulmonary changes, the condition is extremely difficult to recognise. The main points from which assistance is to be obtained are given by Young ²⁷⁴ as follows: - "An apparently causeless but progressive condition of ill health associated with a distressing paroxysmal cough, like that of whooping cough in character,

and worse at night, should excite suspicion. The children may or may not present the so-called tuberculous appearance to be subsequently described, but they become anaemic, fretful, and depressed, and night sweats are common. With regard to physical signs, on inspection it is said that puffiness of the face may occur as in pertussis, and there may be some dilatation of the veins over the upper part of the chest, and a marked growth of downy hairs on the back, but neither of these signs is in my experience of any value. On palpation in the episternal notch, some glandular enlargement may be felt in rare cases. On percussion, there may be dullness over the manubrium or to one side of it, or at the back beside the upper dorsal spine, especially on the right side. On auscultation, a permanent alteration of inspiration at one apex is regarded by Grancher as sufficient to indicate this condition. This author described three stages in this alteration, but it is probable that he attached too great importance to the auscultation of the breath sounds. The signs described are weakening of inspiration and later bronchial breathing with bronchophony. It is stated by D'Espine that in the normal child bronchophony ceases at the level of the spine of the seventh cervical vertebra, while in these cases it may be heard down to the level of the fourth or fifth dorsal spine. This I have been unable to confirm. Occasionally there may be weak breathing over the whole of one lobe, but even in these cases it is well to remember the functional irregularity of the diaphragmatic movements of children, which can be readily observed with the X-rays, and to

examine the chest on several occasions to see if the alteration is a permanent one. A further auscultatory sign is that described by Dr Eustace Smith, namely, a systolic murmur heard over the manubrium sterni when the head is held far backwards. In my experience it is not a sign of any diagnostic value. Radiographic examination of the chest may sometimes afford confirmatory evidence of disease of these glands, but is not to be relied upon unless the enlargement is marked. The co-existence of several of these signs, especially if one of them be dulness in the interscapular region, with a positive tuberculin reaction, renders the diagnosis highly probable."

7. Pneumothorax.

Pneumothorax is a frequent late complication in chronic phthisis, and is due to a small cavity bursting into the pleural sac. After symptoms of extreme dyspnoea, death usually occurs in twenty four to forty eight hours, *but in a few cases the accident is apparently followed by some improvement.*

8. Phthisical Diarrhoea is so frequent a complication of later phthisis that the patient should be warned of the possibility of its occurrence. It is due to tuberculous ulcerations of the intestines, particularly of the colon, or to waxy degeneration of the small intestine. The ulcerative variety is generally caused by swallowing the sputum, a habit which should be carefully checked in children and in young adults, who will often rather swallow the sputum than let it be known that they are expectorating. Waxy disease of the intestine is generally associated with amyloid disease of the spleen

and kidneys. The stools are very foetid.

If the diarrhoea is not checked, the patient soon loses his strength.

Treatment. Put the patient on a diet of milk with equal parts of lime water. Peptonised milk is also useful. Check the diarrhoea with Bismuth and opium, (Formula III) or Chalk mixture and Opium (Formula IV.), or lead and opium pills (Formula V.) If there be ulcerations in the large intestine give suppositories of morphia $\frac{1}{4}$ gr. every eight hours, or enemata of starch and morphia (Formula VI.)

9. Fistula in ano.

This condition usually results from ischio-rectal abscess. If the phthisis is advanced, it is not worth while putting the patient under the strain of an operation, although this should be done in early cases. In advanced cases relieve pain by causing soft motions with a teaspoonful of sulphur at bedtime.

10. Thrush.

This is usually seen in far advanced cases, as snow-white patches, on the lips, tongue, palate, and larynx. The fungus is easily killed by painting the patches with boroglyceride and glycerine (Formula VII.)

11. Pityriasis Vesicolor.

A skin lesion due to the microsporon furfur, which grows best in those areas, where perspiration tends to collect, the front of the chest and armpits. It produces patches of liver brown discoloration, surrounded by a

paler area, and is readily destroyed by salicylic ointment.

12. Swelling of the feet.

This is regarded by Wyllie ²⁷⁵ as a sign of evil omen, preceding death by a few days or a week. As it disturbs the mind, and destroys the hope of phthisis, the legs should be bandaged and kept out of sight.

13. Meningitis.

Death may occur from basilar or cerebro-spinal meningitis. Meningeal tubercles may also cause aphasia or hemiplegia. Peripheral neuritis affecting the extensor muscles of the arm or leg has also been observed.

14. Urinary complications.

Amyloid degeneration of the kidney may occur late in chronic phthisis, producing an increase in the quantity of urine, with albumen and late casts.

Pus in the urine may be due to tuberculous disease of the bladder or kidney, but is rare as a complication of phthisis.

15. ^{mm} Mammary gland.

Allot ²⁷⁶ described a curious non-tuberculous mastitis in males suffering from phthisis, ^{which} ~~and~~ may only occur on the affected side.

THE PROGNOSIS OF PHTHISIS.

The following points are those which influence the prognosis in phthisis.

- a. Heredity
- b. Age
- c. Sex
- d. Occupation
- e. Body Habit
- f. Stage of Disease
- g. Result of treatment
- h. Digestion
- i. Site of lesions
- j. Fever
- k. Pulse
- l. Blood pressure
- m. Complications
- n. Appearance of Sputum
- o. The Urine
- p. Habits.

Every person who becomes infected with the tubercle bacilli does not of necessity succumb to the disease. That in many a slight lesion undergoes a natural cure before it has given rise to symptoms is proved by the fact that post mortem examinations indicate that the majority of persons dying of all diseases have at one time or another had a focus of infection in their bodies. The number of these according to different authorities is set

out in the following table.

Authority	Percentage of Tubercle in all post mortems
Osler	7.5% pulmonary
Heitler	4.7% pulmonary
Bollinger	27% all varieties
Massini	39% "
Harris	38.8% "
Bouchard	75% "
Rebbert	100% "

Clinically, it is found that a certain number of cases, in which the disease gave rise to signs and symptoms, which left no doubt as to the diagnosis, there is an absolute cure, which is due to the formation of fibrous tissue around the lesion and the impregnation of the caseous area with lime salts.

Such cases as these, however, do not constitute the majority of those met with in practice, a point which it is well to keep in mind in view of certain optimistic writings on the subject. At the same time, although phthisis is always a serious and grave disease, the prognosis to-day is far better than it was a generation ago, on account of modern methods of treatment. That is to say, a relative cure, which means a cessation of the lesions so long as the patient is leading a hygienic life, and a lengthening of the duration of the disease, are facts of common knowledge.

There is no greater justification of the part which hygiene has commenced to play in human life than facts such as these, which also indicate that the keynote of

Science is Hope, with which we should start and carry out the treatment of phthisis in every case.

While it is impossible, and extremely imprudent, to lay down mathematic rules, based on types, as the probable duration and termination of the disease, there are certain points which, taken collectively, assist us in forming an opinion as to whether the case will do well or otherwise. These factors have to be considered separately and collectively, for no two cases of phthisis are actually the same.

a. Heredity

While heredity plays a part in the predisposition to phthisis, it is not proved that a history of phthisis in the family will influence one way or another the course of the disease when once declared. The results of Sanatoria treatment show that permanent arrest is not more frequent in those with no family history than it is in those with a history of phthisis.

The family history with regard to longevity, however, exercises a powerful influence on prognosis. The longevity should be gathered from the ages of the parents and grandparents, and there is no doubt that a patient coming of a long lived family inherits a stronger constitution than one of a short lived family. On the other hand, a family history of alcoholism or insanity will tend to make the prognosis more grave. Hermann²⁷⁷ considers that the presence of gout in a family is, to a certain extent, antagonistic to tuberculosis. A good idea of the value of a family history of phthisis is gathered from the following table by which, according to Wyllie²⁷⁵,

Insurance Companies are influenced in the selection of a "life" at age 25.

"If mother died of phthisis add on 7 to 8 years	
If father " " " " 6 to 7 "	
If both " " " " 18 -20 or reject	
If 1 brother)	} may be passed
If 1 sister)	
If 2 brothers died of phthisis add on)	} 7 to 8 years
If 2 sisters " " " ")	
If 2 sisters " " " ")	
If brother and sister " ")	

b. Age

The acute and subacute forms of phthisis are met with in very young and very old people, but the majority of cases of phthisis in young adults are chronic cases, so that they show the greater number of permanent cures. The results at Davos ²⁷⁸ illustrate this. Between the ages of 16 and 20 the permanent cures amount to 79.1%, and between the ages of 20 to 30, they amount to 75.9%, while the number decreases each successive decade until between 60 and 60 only 33.3% of permanent cures are obtained.

c. Sex.

More women die of phthisis between the ages of 15 and 25 than do men, and they also die younger of the disease than do men. It is probable that this is not due to any real sexual difference, but to their more sedentary habits, and to the aggravating influences of pregnancy and lactation on the disease.

d. Occupation.

We have already considered those occupations which predispose to phthisis, and where the patient is unable to leave them, the prognosis will be grave. At the same time if we can justly blame the occupation as a real factor in the causation of his disease, and this be completely removed, the prognosis will be more favourable than in the case of a patient who was previously engaged in a healthy occupation. Thus the writer has noted that phthisis breaking out in sailors usually runs a virulent course, and in these the prognosis is grave.

With occupation is to be considered the social status of the patient, for prognosis is better in the rich than in the poor, as the former seek medical advice early, and are able to take advantage of the best methods of treatment. In the rich we have early diagnosis and prompt treatment; in the poor late diagnosis and indifferent treatment.

c. Body Habit.

It is difficult to approximate the influence of body habit on prognosis, for as West ²⁷⁹ remarks, "Galloping phthisis may develop in a person previously robust, and on the other hand, in a weakly, delicate person phthisis may last for years." Apart from a patient's appearance, it is useful to know if his constitution be strong or weak. Hermann ²⁷⁷ gives an extremely practical rule for discovering this. Those with weak constitutions, feel better, work better, and eat better in warm weather, while those who thrive in the colder months have strong

constitutions. At Davos 278, 62.2 per cent of permanent arrests were got in well nourished and fairly powerful individuals, 42.2 per cent in slender persons, and only 32.2 per cent in those of a definitely phthisical appearance.

f. Stage of Disease.

The prognosis is best in the first stage, and worst in the third, but in general patients in the second stage may have an excellent chance. The writer is here referring to pathological stages as giving a better idea of the nature of the lesions than can be gathered from any anatomical classification.

With regard to onset, most writers are agreed that an acute onset is unfavourable.

Kidd ²⁸⁰ writes, "An acute onset is commonly followed by progressive invasion of both lungs, and has the gravest significance."

Fox ²⁸¹ writes "An acute pneumonic invasion with rapid consolidation of the apex rarely escapes softening and excavation."

Lindsay ²⁸² writes "an acute onset is, in general, decidedly unfavourable."

These views as to the acute onset are undoubtedly correct, for such cases are generally not cases of Chronic Phthisis, but of Rapid Phthisis.

The following views have been expressed with regard to the insidious onset.

Kidd ²⁸⁰ writes, "An insidious, bronchitis, or haemoptoic onset is more favourable."

West ²⁸³ writes, "The most unsatisfactory cases are those in which the onset has been insidious, and where the patient has been losing health, flesh, and strength, without obvious cause."

Lindsay ²⁸² writes "The insidious onset has no definite prognostic significance."

As chronic phthisis nearly always begins with an insidious onset, this latter is obviously more favourable than the acute onset of Rapid Phthisis.

Speaking strictly of chronic phthisis, the writer believes from a limited experience that the most favourable cases are those with a history of cough without expectoration, and dry pleurisy.

g. Result of Treatment.

If treatment results in a disappearance of tubercle bacilli from the sputum, the prognosis is good, as also it is when the sputum and moist sounds cease simultaneously under treatment. The Davos ²⁷⁸ results have shown, however, that the prognosis is bad in those cases where the expectoration ceases, but the moist sounds continue, and that of such accompaniments clicking rales are worst. The increase of weight under treatment is one of the most certain guides as to the progress of the case, and should treatment result in a rapid increase of weight, the prognosis is good. On the other hand a case that remains stationary under treatment has a bad prognosis.

h. Digestion.

A good digestion is one of the most important points in a favourable prognosis. Such patients as take their

food well and assimilate it, have an excellent chance of recovery. On the other a poor appetite, and dyspepsia which does not yield to treatment, are most unfavourable signs, as little can be done in the way of treatment when nutrition is disorganised.

i. Site of the Lesions.

It is stated that the prognosis is more favourable in phthisis affecting the apices than in phthisis of the bases. A good deal depends on the extent of the lesion, for the more tissue involved the sooner will the disease spread to the neighbouring lobe.

j. Fever

The higher the fever and the greater the swing of the chart, the more serious the prognosis. At the same time, if the fever be due to excessive exercise or mental excitement, and disappears when these are removed, the prognosis is more hopeful. One of the gravest signs met with in the course of phthisis is a temperature, which does not fall with rest in bed, and this augurs ill for the patient. The reason for this is not far to seek. When a patient is taking excessive exercise, the increased blood flow through the lungs causes the absorption into the system of a large amount of toxins, which disturb the heat-regulating centre. If, however, there be high temperature, without excessive exercise to account for this absorption, it means that the disease in the lungs is constantly producing by its activity large quantities of toxins, which are an indication of its virulence.

k. The Pulse.

A rapid pulse is a most unfavourable sign, and so also is a latile pulse, that is, a pulse which gains 20 or 40 beats from slight mental excitation, and may be produced by a question regarding haemoptysis.

l. Blood Pressure.

In 1891, Marfan ²⁸⁴ showed that arterial tension is nearly always lowered in pulmonary tuberculosis, and this fact has been since verified by Papillon, ²⁸⁵ and by Potain ²⁸⁶. Marfan ²⁸⁷ now finds that the state of the arterial tension is of extreme value in the prognosis of phthisis. With Potain's Sphygmomanometer the normal arterial tension is 15 to 18. When the tension in phthisis is normal or above normal, the prognosis is generally good. The following are the tabulated results on which this statement is based.

TABLE I.

Arterial Tension	Number of Patients	Course of Phthisis	Remarks
Habitually above normal (19 - 25)	6	6 favourable	1 death from apoplexy, 1 from interstitial nephritis
Habitually normal (15 - 18)	22	21 favourable, 1 unfavourable	In bad case presure fell 2 months before death
Low at first (12 - 13) but higher later (16 - 19)	3	3 favourable	
Normal or above normal at first (15 - 20) but low later (11 - 13)	4	4 unfavourable	1 alcoholic 1 diabetic 1 morpho-maniac

TABLE II.

The arterial pressure of 33 phthisical patients prior to their cure or amelioration

	Number	Per cent
Patients habitually having a normal tension	21	63.6
Patients habitually having hyper-tension	6	18.1
Patients having tension low at first, normal later.	3	9.
Patients having a constant low tension.	3	9.

In general Marfan finds that in chronic phthisis, hyper-tension is favourable so far as the cure of phthisis is concerned, while low tension is a bad sign. Further, if the tension at the start of phthisis be low, but later rise to normal or above normal, the prognosis is favourable, but if the arterial tension fall during the disease, the outlook is very grave. He finds that with haemoptysis, if the tension be normal or high, the prognosis is good, but that low arterial tension with haemoptysis is usually associated with galloping consumption.

He finds that the arthritic constitution, manifest in piles, eczema, asthma, or calculus, is associated with normal tension in youth, and with high tension after middle life. If phthisis occurs in such a patient, and the arterial pressure falls, there is grave reason to fear a fatal termination. On the other hand, if the tension remain high, the patient may ultimately die of arterio-sclerosis, but not usually from the pulmonary lesion.

In diabetic phthisis if the tension be low the prognosis is grave, but if it remain high, or become high

after first being lowered, the future is more hopeful.

It will thus be seen that the estimation of the arterial tension is one of our best guides in the prognosis of phthisis.

The following are Marfan's conclusions.

"In pulmonary tuberculosis the arterial tension is generally lowered. When it is normal or above normal, one is able to foresee a favourable termination, as it indicates that the disease is susceptible of cure or of amelioration. This rule has few exceptions, and the estimation of the arterial tension is one of the most certain means of knowing the curable forms of phthisis.

The lowering of arterial tension has usually a bad significance, and indicates progress of the lesions, but this rule is less applicable than the preceding one, and does not preclude the possibility of a persistent amelioration or even a clinical cure."

m. Complications.

With the exception of dry pleurisy, the appearance of complications indicating the spread of the bacillus to other organs is extremely unfavourable. An especially grave significance is to be attached to amyloid disease, ulceration of the bowels, laryngeal phthisis, and swelling of the feet.

n. Appearance of the Sputum.

Lowenstein ²⁸⁸ has studied the significance of tubercle bacilli in the polymorphonuclear leucocytes in the sputum. From observations on 56 cases he finds, (1) that tubercle bacilli are present in leucocytes in 10% of all

cases of phthisis, occurring in chronic cases and in those with a tendency to recovery, and (2) that the disposition of bacilli in the leucocytes points to their rapid disappearance from the sputum

o. The Urine.

Erhlich announced in 1883, that in hopeless cases of pulmonary tuberculosis the drazo-reaction appeared early and lasted until death, and was thus a useful factor in prognosis. Recently, however, Dmitrenko ²⁸⁹ has studied the reaction in 300 cases of phthisis. He finds that it appears very rarely at the beginning of the disease, that it may disappear as the patient's state improves, that it may disappear some weeks before death, and that it may be completely absent in grave forms of the disease. It has no relation to temperature.

p. Habits.

The importance of habits in prognosis cannot be over-estimated, and habits which lead to excesses will counterbalance any other favourable factors in prognosis.

While absolute certainty in prognosis is as impossible as it is unwise, the patient and his friends, if they are to undergo sacrifices, change their mode of living, seek other climates, and spend time and money in treatment, very naturally expect to have some indication of what the ultimate result is to be. By a careful consideration of the foregoing factors, we are able to say with some degree of certainty whether -

1. The case is hopeless.
2. The illness may be prolonged by treatment.

3. The disease may be ameliorated by treatment.
4. A relative cure is to be obtained.
5. An absolute cure may be reasonably expected.

As to time, there is only one condition where a fatal termination can be accurately foretold, and that is when we find the lungs involved by active disease in every lobe. In such cases, it is a safe pronouncement that the patient will not live a month.

In this disease, general treatment is of little avail until they are alleviated. In this chapter we will discuss the treatment of the following symptoms of phthisis.

1. Dyspepsia.
2. Cough.
3. Night Sweats.
4. Haemoptysis.
5. Pyrexia.
6. Debility.
7. Pleurisy.

1. Dyspepsia.

Dyspepsia is one of the most important symptoms of phthisis so far as treatment is concerned. So long as it is present, alimentation is disturbed, and we are unable to raise the patient's resistance and so combat the progress of the disease.

We have seen that in early phthisis there is nervous dyspepsia, which is characterised by a slightly fevered tongue, with loss of appetite, and "burping" after meals. Later there is atonic dyspepsia with pain during meals, and vomiting after food. These may go together and there is

 THE SYMPTOMATIC TREATMENT OF PHTHISIS

There is a principle of Medicine, more generally professed than practised, to treat the disease not the symptoms. The treatment of phthisis constitutes one great exception to this rule, for once symptoms have arisen in this disease, general treatment is of little avail until they are alleviated. In this chapter we will discuss the treatment of the following symptoms of phthisis.

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2. Cough.

3. Night Sweats,

4. Haemoptysis.

5. Pyrexia.

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1. Dyspepsia.

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We have seen that in early phthisis there is nervous dyspepsia, which is manifest in a slightly furred tongue, with loss of appetite, and "hawking" after meals. Later there is atonic dyspepsia with pale flabby tongue, and vomiting after food. This may go further until there is

chronic gastritis, with intolerance of all food, and vomiting.

There is an intimate connection between the dyspepsia of phthisis and vomiting. The writer believes that the syndrome of phthisical vomiting is in most cases primarily gastric. Food ingested into an irritable or inflamed stomach stimulates the vagus nerve and induces a dilation of the pulmonary capillaries. This in its turn aggravates the irritation of the tuberculous foci, thus causing coughing, which terminates in vomiting.

The appetite is generally poor, although the fictitious appetite of later phthisis occurs with a temperature of 102° - 103° F.

When dyspepsia is present it is bad practice to ignore it, and absolutely barbarous to stuff the patient with food which he is incapable of digesting, and which only aggravates his general condition. The accounts one has read of the over-feeding in German Sanatoria, and of patients swallowing four pounds of meat in the day, are enough in themselves to nauseate a healthy adult, to say nothing of the actual effect on a phthisical dyspeptic.

In the treatment of dyspepsia, the most useful agent is rhubarb and soda, given fifteen minutes before meals (Formula VIII) and there are few cases of the nervous dyspepsia of phthisis where an immediate and beneficial result is not obtained. The food should be light, nourishing and appetising.

If chronic gastritis is present, the patient should be placed on milk and lime water alone for several days.

Pari passu with the improvement of appetite, the

patient may be encouraged to eat more frequently.

2. Cough.

As pointed out by Douglas Powell,²⁹⁰ cough removes the products of disease, so in the majority of cases of phthisis there is no necessity to treat this symptom. When a patient only coughs at certain intervals in the day, say in the morning and late afternoon, and this cough is easy and accompanied by sputum, it were an absolute error to check it by drugs, as the sputum instead of being expectorated would lie in the bronchioles, causing further irritation and spread of the lesions. A phthisical patient knows instinctively the value of cough, as is apparent to anyone who has been in the vicinity of a phthisis ward before the patients turn in for the night. They make an effort to cough, and so clear their lungs for the night, and are able to sleep. Yet there are times when the cough requires treatment.

If in the morning, the patient has an irritating cough after breakfast, and has difficulty in bringing up the sputum, this will be eased by giving him a cup of hot tea the first thing in the morning, and allowing him to rise for breakfast half an hour later. On no account is morphia to be given for this.

If this irritating cough accompanied by difficult expectoration be present during the day, the pulmonary secretion may be eased by an expectorant cough mixture (Formula II). Should the cough be dry, irritating, and hacking, with no expectoration, it points to the cause as due to the irritation of the pulmonary lesions, and

mild sedatives such as the balsams, aromatic oils, and spirits of chloroform should be tried (Formula IX). This cough will often disappear with rest in bed, and cod liver oil.

When the accumulation of mucus in basic cavities induces a trying and ineffectual cough, Mitchell Bruce finds that inverting the patient will ease his condition.

When the cough is loose, paroxysmal, and frequent, accompanied by large quantities of fluid expectoration, the cause is generally bronchial, and inhalations of benzoin, tar, or 2% formalin are indicated (Formulae X and XI).

We have now mentioned five varieties of cough which are frequently seen in phthisis, yet for which no morphia has been prescribed. The indiscriminate prescribing of cough mixtures containing morphia for phthisical patients, is no doubt very gratifying in its immediate effects to all concerned, but on no ground is it defensible. It locks up the secretions in the lung, induces decomposition of the waste products of pulmonary catarrh, thereby further damaging the tissues, deranges digestion and is apt to induce the morphia habit. It is difficult not to write too strongly on this, for the writer has seen the habit induced by this means in those who did not know they were taking morphia, but would clamour for a particular mixture which contained it.

If the cough is present at night so that sleep is prevented, or during the day to an extent which is weakening to the patient, it is best at first to try the hydrocyanic mixture (Formula XII) and only in the event

of this failing to resort to morphia.

At the end of phthisis, it is a different story. Here there is no drug to give the mental and physical rest of opium, which robs death of half its terrors. It may be given as the Brompton Mixture (Formula XIII), but a mixture containing Heroin is preferable, (Formula XIV)

3. Night Sweats,

As in the treatment of cough, there is too great a tendency to use morphia off-hand, so also the treatment of night sweats by atropine is to be discouraged. Night sweats are due to weakness and toxic infection, the toxins most likely being those of other pyogenic organisms than of the tubercle bacillus alone, for sweats are not the inevitable accompaniment of phthisis. It is thus possible to combat the causes of night sweats by fresh air, generous diet, and by inhalations of the essential oils, particularly oil of pine, which seem to retard the growth of pyogenic bacteria, although they do not influence the tubercle bacillus.

Further, it is possible to do much to improve the hygiene of the skin by sponging the body daily with tepid water to which there is added a little of the crude eau de Cologne. This proceeding is at once beneficial and pleasant.

The routine use of liniments and vesicants is to be utterly deprecated. As Lindsay 291 truly remarks: "There are few sadder sights in practice than that of a consumptive's chest plastered over with iodine, and in a state of positive filth - a sight, unhappily, by no means rare. It can only be called barbarous."

Should the methods of hygiene fail, we may resort to drugs, and of these the simplest and most effective is an ounce of whiskey in warm milk half an hour before bedtime. It is generally supposed, and undoubtedly is the case, that a hot whiskey at bedtime is a sudorific, but this does not hold good in phthisis, and it has the further advantage of being a soporific. If this fails, a pill of quinine, digitalis and opium is always effective. (Formula XV)

4. Haemoptysis.

The first question to ask in a case of haemoptysis should be "Is this the first?" If this is so, and the haemorrhage be from the congestion of early lesions in the lung, the danger is not immediate but remote, as it is unlikely that the patient will lose a large quantity of blood. Further, the treatment of early haemoptysis differs from that of later haemorrhage.

(a) Early Haemorrhage. The first thing is to assure the patient and relatives of the absence of danger, and to prevent panic. If the patient be in a state of mental excitement, an eighth of a grain of morphia may be injected. Jalap should be given immediately to cause brisk purgation, which lowers the blood pressure. To raise the coagulative power of the blood, calcium chloride in doses of 20 grains every two hours is indicated.

The patient should go to bed immediately in a well aired, darkened room, and all conversation forbidden. He must remain in bed at least for a week. A slight degree of faintness may be present from the nervous shock,

but, as this lowers the blood pressure, it does not generally call for treatment. No food must be given for several hours, and even then all proteid is to be avoided for a day or two. If the haemorrhage persists in alarming quantities, morphia should be pressed. The remote dangers of haemoptysis have already been considered.

(b) Later Haemorrhage. Here there is danger of immediate death from syncope or suffocation, and it is necessary to recognise immediately which is to be feared, as the treatment of the two is different.

Death from syncope results from the loss of a large quantity of blood, which the patient coughs up. The immediate treatment is to arrest the haemorrhage. The writer has seen good results from the inhalation of 5 minims of amyl nitrite, a glass capsule being broken in a handkerchief under the patient's nose and mouth. To maintain a reduced blood pressure $\frac{1}{4}$ - $\frac{1}{2}$ gr. of morphia is immediately given with a hypodermic. Absolute rest is essential, the patient remaining in bed, and using the bed pan. Purgation is dangerous.

Ice to the chest or sucked is merely a placeto, and recent work on uterine haemorrhage shows that it is not a haemostatic.

Adrenalin has recently been recommended, but on what grounds it is difficult to see. If applied locally it has an immediate haemostatic effect on blood vessels. In the lung, however, it is impossible to apply it locally, and when given internally it raises the blood pressure all over the body, and so increases the haemorrhage.

Ergot, tannic acid, gallic acid, acetate of lead, given internally exercise no haemostatic action on the pulmonary vessels, and are relics of the past.

The danger from suffocation is recognisable when the patient puts up no blood, but shows the signs of haemorrhage - clammy brow, pale drawn face, dilated pupils, cold extremities, dyspnoea, and faintness. He should be told at once "to cough it up", and if unable to do this, a nauseating dose of Ipecacuanha is given.

In early haemorrhage the patient, if ignorant, need not be told of his disease for a day or two, but the shock of the first symptom over, the whole question of diagnosis and treatment must be considered. It is nothing short of malpraxis to tell a patient that he "has strained his lung cycling."

5. Pyrexia.

The treatment of pyrexia is rest in bed. As it is often due to mixed infection, antiseptic inhalations will do good. If pyrexia resists this treatment, the case is hopeless. The usual antipyretics do little good, but Mitchell Bruce recommends 2 - 3 gr. doses of Phenacetin every hour for three consecutive hours, to be given daily if the patient is relieved. In some cases quinine does good by its tonic and antiseptic action.

6. Debility.

This is the result of toxæmia, and is to be treated by rest and nourishment in the form of tonics and malt. Arsenic and iron in the debility of phthisis has the high authority of Trousseau, ²⁹² and the writer has seen re-

markable results follow its use. (Formula XVI)

7. Pleurisy

As we have seen pleurisy is a beneficial symptom, which may be explained to the patient to ease his mind. For the relief of pain, the writer has found strapping of the chest with sticking plaster to be more efficacious, if well applied, than liniments or counter irritation.

C H A P T E R X V .

THE OPEN AIR TREATMENT.

Since the earliest days of Medicine as an Art down to comparatively recent times, the whole treatment of phthisis was based on erroneous principles. The disease was regarded as an "inflammation" for which a low diet and antiphlogistic drugs were prescribed. It was often thought that bad air might have something to do in its causation, but it occurred to none that fresh air was as important in the treatment as foul air was in the aetiology. Here and there throughout the centuries, unconscious references are made to elements of the open air treatment, thus Hippocrates advised long rides on horseback, Celsus considered sea voyages beneficial, and LaennaC recommended a life by the sea shore.

The idea of the open air treatment of phthisis was born in the Highlands of Scotland 1747 when the unknown pioneer wrote "A letter from a Physician in the Highlands to his friends in London." The message was ignored in London, but in Scotland the idea was not allowed to die, for in the early part of the nineteenth century, the Parish Minister of Erskine ²⁹³, Dr Stewart, who held the high and ancient office of priest and physician, was carrying out the open air treatment on lines which differ in no particular from the present hygienic treatment of phthisis, as may be gathered from

the following account written by one of his patients.

"In the old established practice, Dr Stewart contends that the effect is pursued, and not the cause; and that irritation of pulse and quickness of circulation, which are the effects of weakness, are treated as if they proceeded from inflammation, until the patient is reduced to a skeleton, and all the powers of the constitution are destroyed; thus co-operating with, and increasing, a disorder of which the destructive tendency and the ravaging powers are but too well known; but admitting that this old practice succeeds, and that the life is preserved, it is still but a system of palliation, for the extreme precaution necessary against the fear of taking cold makes the patient a complete hot-house plant, and implies a state of constant bondage. The climate cannot be brought to suit the constitution, therefore the constitution must be hardened to bear the climate, which can only be done by restoring the frame to its original healthy tone and vigour

"It is Dr Stewart's urgent wish that the patients should be for many hours daily in the open air - cautioning, however, avoiding fatigue - either on horseback or on foot, or in an open carriage, which last he relies upon as being least likely to tire the patient. The diet should consist of plain meat, broth, pudding, fish, or any course of simple but nutritious food, according as the patient feels inclined

"Dr Stewart is anxious to explain that his system partakes not in the most remote degree, of the Brownonian

or cramming system; he is altogether hostile to it, or to any undue means of giving strength by false stimuli he only prescribes that moderate quantity of refreshing food which nature and reason point as wholesome, and those early and regular hours which are most efficacious to digestion."

In the early years of the eighteenth century, other writers now and then urged the adoption of the open air treatment, but it was not until 1840, that George Bodington²⁹⁴ laid down the principles of this treatment and founded the first Sanatoria in the world, amid a storm of opposition and ridicule from the powers that were. The clinical acumen and scientific instinct which characterised his work may be gathered from the following passages of his book.

"Sir James Clarke rather sarcastically alludes to what he terms the beef steak and porter system, which he decidedly condemns, apparently guided by the "phlogiston" theory. I could never recommend porter and beef steaks to any person suffering from tubercular consumption - not from any preconceived notion of "phlogiston" but on accord of its very grossness and unfitness for a consumptive patient. On the other hand neither could I recommend to such an one, from a prejudice in favour of the aforesaid theory of 'phlogiston', a meagre diet of vegetables, beer and water, aided by tartarized antimony, etc. I should recommend to one thus consuming away under the influence of this wasting disease, a

nutritious diet of milk, fresh animal and farinaceous food, aided by the stimulus of a proper quantum of wine having regard to the general state and condition of the patient. If this is to be called the "beef steak and porter system" then I am guilty of patronising it, but to my mind it rather has the character of a preservative system, whilst the wasting plan is as much entitled to be called the destructive one."

And again:

"The only gas fit for the lungs is the pure atmosphere freely administered, without fear; its privation is the most constant and frequent cause of the progress of the disease. To live in and breathe freely the open air, without being deterred by the wind or weather, is one important and essential remedy in averting its progress, one about which there appears to have generally prevailed a groundless alarm lest the consumptive patient should take cold. Thus, one of the essential measures necessary for the cure of this fatal disease is neglected from the fear of suffering or increasing another disease of trifling importance. No two diseases can be more distinct from one another than consumption and catarrh. It is the latter only which might be caught by exposure to atmospheric causes; with the former they have nothing to do. Farmers, shepherds, ploughmen, etc., are rarely liable to consumption living constantly in the open air, whilst the inhabitants of towns and persons living much in close rooms within doors are its victims. The habits

of these latter ought, in the treatment of the disease, to be made to resemble as much as possible those of the former class as respects air and exercise, in order to effect a cure. How little does the plan of shutting up patients in close rooms accord with this simple and obvious principle."

"The equal temperature so much considered and said to be necessary should be that of the external air instead of that so commonly employed, the warmth of a close room.

"The common hospital in a large town is the most unfit place imaginable for consumptive patients."

These views, to which modern Science gives her unqualified approval, were in their time the subjects of sarcasm and ridicule to drive the patients from Bodington's Sanatoria, which was later filled with lunatics. He was the indignant prophet of his age, and the mind of the seer is manifest in what he wrote ^{to} his son ²⁹⁵ "I often think that, when I am dead and buried perhaps the profession will be more disposed to do me some justice than whilst I lived." He died in 1882, the year in which Koch discovered the bacterial nature of phthisis.

While in his own country the prophet was not accepted, it was otherwise abroad. In Germany, Hermann Brehmer, attracted and convinced by the writings of Bodington, was an active convert to the open air treatment, and in 1854 founded a small Sanatoria in Silesia,

while in 1859 he built the now famous Sanatoria of Gorbardsdorf in Upper Silesia, where the following rules were carried out

1. A life spent in the open air under conditions which give immunity from tuberculosis.
2. Complete freedom from any debilitating circumstances or anything which may lead to an exacerbation of the disease.
3. Methodical hill climbing as an exercise, when the condition of the patient renders this desirable.
4. An abundant dietary in which milk, fatty foods and vegetables occupy an important place.
5. Various hydro-therapeutic measures.
6. Constant and unremitting medical supervision.

Among Brehmer's earliest patients was Dr Peter Dettweiler, who later founded the world famous Sanatoria of Falkenstein in the Taunus Mountains.

After Germany, the movement spread to America, where the pioneer is Edward Trudeau. In 1873, he was sent by Dr. Loomis to the Adirondack Desert in the hope of prolonging his life, and there he later founded the famous Adirondack Cottage Sanatoria, of which he is now the distinguished chief.

The open air treatment of phthisis is based on the fact that as there is no specific cure of the disease, the method of saving a patient's life is to raise his resistance to the infection, whereby a natural cure may

result. To attain this end, the patient leads a hygienic life in conformity with nature, of which the three main elements are:

1. Fresh Air.
2. Nourishing Food.
3. Regulated exercise and rest.

1. FRESH AIR

This is the first requirement of the treatment, and the patient is removed from the stuffy atmosphere of the ill-ventilated sitting room and bedroom to the pure air of the mountain, of the sea, of the desert. It is not difficult to understand the vast alteration such a change will make on a patient, when we remember that an adult inspires some twenty-eight thousand times in twenty-four hours.

The main qualities of the air a consumptive should breathe are that it be pure, dry and calm, which thus obviates dust and cold. Such qualities are to be found in the air of the country, not in the smoke-laden atmosphere of towns. So far as purity of the air is concerned, it is to be obtained in most country places in the British Isles. Compared to purity of air, changes of temperature are a minor consideration. If there be a great fall of temperature in the evening, it means that the number of hours the patient can breathe fresh air outside is less than the number he could spend in the open air if such a change were not present. This will be more fully entered upon in the Chapter on

climate, but even if, as occurs in most places in this country, there be a great fall of temperature at night, the patient can still breathe pure air in his house.

There is a great distinction, ill understood by the majority of lay persons, between fresh air and draughts. Nothing is worse than sitting in a draught, even if it be a draught of pure air, for one side of the body is cooled whilst the other is not, likewise the practice which the writer has seen at some Sanatoria, of making the patient sit in a room, with the windows wide open and a perfect gale of wind blowing, is a pure fetish, and in advanced cases may be absolute cruelty.

How then may a patient at home undergo the open-air treatment? The rooms of English houses are little adapted for this purpose on account of their faulty construction, so far as ventilation is concerned, for the room is either stuffy or draughty. The whole house must be constantly ventilated with fresh air, and to attain this it is a good plan to have a board 6 inches deep placed below the lower sash of every window in the house. This means that the window is open between the middle sashes, and the current of air is directed towards the ceiling of the room. In mild summer weather, the windows may be thrown open, but the above plan is very serviceable at nights, and in winter.

In most cases, it is extremely useful to send the patient to a Sanatoria for a longer or shorter period, where he will be inculcated with the principles of the

open air method, and will carry them out in his own home. This refers to cases which cannot afford to undergo the entire cure at a Sanatoria. Those who have returned cured are careful to carry out the details in most cases, as a means of retaining their health. The patient should spend as many hours as the weather permits in the open air, and Osler recommends that he have a calendar on which the hours of sunshine may be ticked off. When sitting outside, the patient must avoid draughts, and for this purpose the Liège-halle or moveable summer-house is extremely useful.

As to hours, these will vary according to the situation of the patient, but he should make a rule of retiring early, as sitting late is associated with a hot atmosphere and tobacco smoke. His bedroom should be well ventilated before he turns in, and if the night is cold, he may undress before a fire, at which his night things have been warmed. A hot drink of milk will often induce sleep, and if not he should read soporific literature, of which it is not difficult to select a few works.

If the morning be damp, the patient should dress before a fire, and rise by eight in order to get a fresh atmosphere.

All rooms used by the patient during the day must be well ventilated, and not too cold.

2. NOURISHING FOOD.

No definite rules need be laid down as to the exact nature of the patient's food, as it will depend to a great extent on what he has been used to. A good

general rule is that the patient should have if possible a better and more generous diet than the one he was on prior to his illness. In any diet milk should have an important part, and ought to be boiled, unless the cow is known to be healthy by the tuberculin test, when it is best taken warm and fresh. To many patients the addition of a little lime water will make the milk perfectly digestible. To be avoided in general are greasy soups, cold meats, tinned meats, pork and sausages. A generous diet is one thing, to stuff the patient is another. This latter is indefensible. He may put on weight like the Strasbourg geese, but his strength is not improved. The main diet should be milk, eggs, butter, fish, chicken, game, mutton, vegetables, bacon, ham, well toasted bread and preserves.

3. REGULATED EXERCISE AND REST

The temperature is our unfailing guide as to exercise and rest. A patient with early phthisis may take any exercise in moderation, which does not raise his temperature. Golf, tennis, riding, motoring, fishing, and shooting are among the most suitable, but each must conform with the rule as to temperature. Cycling is not suitable, as it throws too great and sudden a strain on the heart and lungs. If we find that these exercises cause a rise of temperature, they must be stopped immediately, because it is impossible to graduate them with respect to temperature.

In these cases patients should follow the "cure de terrain" which has become widely known in Germany, and consists in muscular, cardiac, and respiratory exercises, induced by hill-climbing.

We begin the treatment by prescribing a definite walk. Walking on the flat is not the exercise it is reputed to be, being mostly mechanical, and muscles are more stimulated when their action is accompanied by effort. The patient should therefore walk up a hill, for here the muscles are consciously exercised to the end of the climb, and further a different group come into play on ascending and descending, as anyone who has climbed hills must have noticed the great rest to the hamstring muscles when the descent begins.

The exercise is easily regulated, and affects the heart and lungs.

It has been stated that the heart in phthisis is smaller than normal, but whether this be so or not, there is no doubt but that it is weakened by the toxins in the blood, and secondly by the obstruction to the pulmonary circulation.

In many phthisical patients the chest is narrow, and in all the capacity is diminished.

The effect of climbing is to induce deep breathing and to stimulate the heart and circulation, and it is superior to mechanical chest exercises in the following particulars. First, the chest movements are reflex, and depend on the amount of energy expended. Secondly,

the movements are natural, and there is not the extreme expiration which mechanical gymnastics induce, and which cannot but be harmful to a damaged lung. Thirdly, the exercise lasts much longer, and it is impossible to breathe mechanically for more than a few minutes without fatigue.

If the exercise causes fever, it is to be diminished and there are some cases where it must be commenced on a level surface.

Climbing is also suitable for two small but recognisable groups of phthisical patients.

(a) Those who suffer from dyspnoea, from a mechanical cause in the lung - such as fibrosis, emphysema, and old pleuritic adhesions. In these patients the dyspnoea is due to the diminished "Tidal Breathing" from the small area of available pulmonary tissue. These cases are greatly improved by the exercises, although they may be astonished at first by the treatment suggested, as they easily become breathless.

(b) The obese phthisical patient, and the cardiac case with a superimposed phthisis, are also suitable for the "cure de terrain" although its effects are to be watched most carefully.

The treatment by climbing has the great advantage that it can be carried out in most country places, but certain precautions are necessary.

The patient should never feel tired after the exercise. If he does, it has been excessive, and must be reduced.

The climbing must be methodical and progressive carried out at definite hours and increased week by week.

It should commence with short walks up slight inclines, the length and gradient being gradually increased.

The patient must breathe through the nose, and avoid talking, as this will cause hurried and unequal breathing.

Arriving at the top of the ascent, he should at once descend, and on no account sit down. On reaching the foot of the hill he should put on an overcoat and return to the house for a tepid sponging and change of clothes. The temperature should be taken at once, as it may be of only slight duration, but is an indication of excessive exercise, as also is loss of weight, except in the obese.

There is a clinical as well as a physical difference between climbing and ordinary respiratory gymnastics, as the latter have been known to produce bad results in phthisical patients. Thus Mantoux²⁹⁷ has seen this followed by increased cough, catarrh and haemoptysis. Yet he holds that respiratory gymnastics are useful in cases where climbing is impossible, as in convalescent pleurisy with a fluctuating temperature and where rest is indicated. In these cases he believes that respiratory gymnastics help to fill out the atelectic lung by the introduction of air.

Climbing, as it increases the pulmonary circulation has the further advantage of flooding the diseased areas of the lung with rich blood containing the free

opsonins. Not only so, but small quantities of bacterial débris will be absorbed into the general circulation, where they will stimulate the production of fresh opsonins. It is obvious that if the exercise be excessive too much of this bacterial débris will be absorbed, so causing a lowering of the amount of opsonins present. The writer has seen short exercise cause a marked rise in the opsonic index. In view of the danger of too great auto-inoculation, it is well that on this point the temperature is as reliable a guide to excessive exercise as is the opsonic index.

Points of detail in the open air treatment are; the hygiene of the skin, which is best ensured by the tepid bath in the morning, and a sponging with tepid water, to which crude Eau de Cologne has been added, after exercise or before retiring; the avoidance of all excitements and worries, and constant medical supervision.

The following is a suggestion of the routine management of a case of phthisis:

7.a.m. Fire lit in patient's bedroom if morning is cold.

7.30 a.m. Cup of tea infused with milk.

8 a.m. Patient rises, tepid bath, and dresses in front of fire.

8.45.a.m. Rhubarb and soda, if directed, then walk for 15 minutes in open air if weather is fine.

9.a.m. Breakfast. Allowed: Fruit, porridge, tea, coffee, or cocoa, made with milk, buttered toast, pure

marmalade or home made jams, fresh eggs, boiled, scrambled, or poached, fresh fish, omelettes, bacon and eggs, or cold ham. Avoid: sausage, cold meat, and new bread.

Tonic or specific.

9.30 - 10 a.m. Sit for half an hour reading papers or letters.

10 - 12 a.m. Out of doors. Exercise in some form or sitting.

12.30 a.m. Lunch - Avoid greasy soups. Take soup, fish, chicken, white farinaceous puddings, fresh milk.

1. - 3.30 p.m. Out of doors. If exercise causes perspiration change flannels and sponge.

4.p.m. Afternoon tea made with milk.

4 - 6.15 p.m. Rest outside or inside, according to weather.

6.15 p.m. Rhubarb and soda, if required.

6.30 p.m. Dinner, followed by tonic or specific.

9.p.m. Retire for night.

If patient is used to alcohol, he may continue it at meals in small quantities but is better without it. He should cease smoking.

CHAPTER XVI

SPECIFIC MEASURES IN THE TREATMENT OF PHTHISIS

There is no specific for phthisis, and of the hundred drugs which phthisical patients have swallowed, there is not one which has cured the disease. In speaking, therefore, of specific drugs, we refer to those which are used in the belief that they exert a noxious influence on the tubercle bacillus. Such drugs are given by -

1. Inhalation,
2. Ingestion,
3. Intravenous injection,
4. Intratracheal injection,

and among the most frequently used by these methods are -

1. Creosote,
2. Carbolic acid,
3. Thymol,
4. Formalin,
5. Essential oils,
6. Cod liver oil.

The internal administration of sulphur has been tried by the writer.

1. Inhalation.

It is extremely doubtful if any inhalation can reach the alveoli in sufficiently concentrated a form to exercise any deterring influence on the tubercle bacillus.

Nor is this strange when we remember that the respiratory exchange takes place not in the alveoli but in the larger

bronchi, so that any inhalation must become considerably diluted before it reaches the terminal alveoli.

The subject has been experimentally investigated by Murrell ²⁹⁸. He found that on passing the vapour of oil of Cinnamon or Peppermint over cultures of tubercle bacilli, their growth was not retarded in the slightest degree. On the other hand, a 6% solution of Formalin, (*Formula X1.*) which in itself is a 40% solution of formaldehyde, was found to kill the tubercle bacilli after a short exposure to its fumes. A patient is able to inhale a 6% solution of formalin (1-16), by ~~bubbling~~ ^{breathing} the air to be inspired through *a mask.*

Murrell tried the effect of two such daily inhalations, lasting from 10 minutes to half an hour, on fourteen cases of phthisis, some of which were advanced cases, and obtained an improvement in twelve cases. This treatment was first proposed by Cervello ²⁹⁹ in 1899.

Admitting the lethal properties of Formalin to the tubercle bacillus, it is improbable that it reaches the deep foci of disease in the lungs, although it is clinically certain that the inhalation of such drugs is beneficial in checking the progress of secondary infections.

2. Ingestion.

Kobert ³⁰⁰ found that the ingestion of oil of camphor or cinnamic acetol produced a leucocytosis, from which he deduced a favourable influence on phthisis. A prolonged trial of these drugs, however, did not bear out this anticipation.

Creosote and its allies are the most used of the internal specifics in phthisis. It is certain that their action as intestinal antiseptics will promote nutrition

by preventing the appearance of the irritating products of decomposition, but how far they act on the lung is an extremely doubtful question. It has been proved to have no influence in checking the processes of tuberculosis in animals.

Creosote may be given in doses of from 1 to 20 minims thrice daily after food, which is also the dose of its ally guaiacol. This latter may be given, as recommended by Philip in the following alcoholic solution.

℞ Guaiacol 1 part
Alcohol (90%) 20 parts
Aquam 180 parts

If dyspnoea is present, or there be an objection to the taste and smell of these drugs, Duotal (carbonate of guaiacol), a white tasteless crystalline powder, is given in doses of 5 - 25 grs. daily. Philip uses a pill of Duotal and Arsenic.

℞ Duotal gr. IV
Acid Arsenios. gr. $\frac{1}{60}$
Fiat pilula.

Sig. One to three thrice daily after food.

Creosotal (carbonate of creosote) is a brown viscous liquid, the smell of which is much less marked than that of creosote. It is taken in some fluid after food in doses of from 3 - 30 minims thrice daily.

With regard to the curative action of these drugs, authorities differ. Philip³⁰⁰ finds they exercise a beneficial influence in chronic phthisis.

"Prolonged use of creosote and numerous allied bodies, has led me to the conclusion that they do exert a beneficial - even curative - influence in more chronic forms of pulmonary tuberculosis. It is less easy to be

categoric as to the nature of this influence. Experimental observations on animals have failed to show that creosote is certainly inimical to the development of the tubercle bacillus. Yet there can be little doubt clinically that the symptoms and course of the disease are modified - in some cases most-favourably - by its exhibition. More particularly, cough and expectoration are often reduced remarkably; or, putting it otherwise, secretion is lessened, and thereby the need for cough removed. I have frequently noted the gradual, and sometimes complete, disappearance of a copious, bacillus-containing sputum follow the continuous, prolonged use of creosote. Laryngeal, tracheal, and bronchial manifestations are influenced favourably. In like manner, the gastro-intestinal tract benefits. Appetite and digestion improve. Complicating diarrhoea is frequently relieved. Contrary effects which are occasionally observed, such as irritation of the alimentary tract, are probably referable to the use of impure or cruder kinds of creosote. Such disturbing effects have not occurred to any extent in my experience, when pure beechwood creosote is administered. Still more rarely does renal disturbance occur. None the less, it is wise to keep an eye on the urine."

On the other hand, Lindsay³⁰¹ writes: "Creosote and uaiacol have now had a prolonged trial in phthisis. Their use was evidently suggested by the hypothesis that these agents might in some way assist to antagonise the action of tubercle bacilli, render the tissues more resistant, perhaps promote fibroid change. A large number of observers of the highest eminence have expressed them-

selves as favourably impressed with the results of this treatment. I confess that, after a long trial of these remedies, I have seen nothing to justify the high place which they have held in the treatment of phthisis. I am inclined to suspect that creosote and guaiacol have shone by reflected light. Their general adoption as remedies in phthisis was almost coincident with the wide recognition of the value of systematised hygiene in this disease, and the better results obtained in recent years have been sometimes attributed in part to medicaments which, I believe, have had little real efficacy. No doubt, creosote sometimes exercises a favourable influence upon gastric derangements. That it, in any sense, renders the lungs or the tissues generally immune to the action of bacilli is a most doubtful proposition. In recent years I have gradually discarded creosote and guaiacol - reluctantly, because I could not ignore the weight of evidence in their favour - but I feel certain that the results of treatment have been at least as good without, as with, these remedies."

Other less frequently used drugs of this nature are - Styraol (cinnamate of guaiacol), a white crystalline, insoluble powder, without taste or smell. Dose 10 grs. three times a day.

Benzosol (benzoate of guaiacol), a white crystalline sparingly soluble powder, with slight taste and smell. Dose 4 - 12 grs. thrice daily.

Thiocol (ortho-guaiacol-sulphonate of potassium), a white crystalline, soluble powder, without smell, and with a faint saline taste. Dose 5-30 grs. t.i.d.

Cod liver oil. While the tonic and nutritive properties of cod liver oil are well recognised, a specific action on tuberculosis has not been proved. A series of experiments on young pigs have been made by inoculating them with tuberculosis, then feeding one set with cod liver oil, while others received ordinary food as a 'control.' Naturally, those fed with the oil gained more weight than the others, but as there was no control to show the normal increase of weight in a young healthy pig fed on garbage, the experiment is worthless.

The writer has found that cod liver oil is absorbed into the blood. In making opsonic estimations, it was noticed that the blood of phthisical patients when drawn into Wright's capsule settled more quickly than normal blood, that is to say, the red and white cells began to sink before the blood had coagulated leaving a clear area of plasma at the upper part of the tube. In normal blood, the cells do not sink much before coagulation occurs.

At first the writer believed that either this was due to delayed coagulation of the blood in phthisis, or that he had discovered the long sought condition of true hydraemia.

Experiment showed that coagulation was not delayed, but that the specific gravity of the blood was lowered. A series of 25 estimations showed that this lowering of specific gravity coincided with a fall in the haemoglobin, which is naturally diminished in the anaemia of phthisis. The following table shows the specific gravity, and the percentage of haemoglobin found.

It will be seen that in most cases the haemoglobin is higher than it should be with the corresponding specific gravity.

Even if the two coincided, they do not explain the phenomena observed in the capsule, for if the low specific gravity was due to the diminution of haemoglobin in the red cells, these would tend to float rather than to sink. The experiment was further complicated by the presence of leucocytosis in some cases, which would tend to raise the specific gravity. Eventually by elimination everything pointed to cod liver oil. It was found that the sinking of red cells when blood is drawn into a capsule is only got in those patients who are taking oil, and by taking cod liver oil emulsion, the writer produced the phenomena in his own blood.

TABLE

Specific Gravity	Haemoglobin
Normal 1049 - 50	90
Phthisis.	
1 1048	70
2 1046	60
3 1047	70
4 1046	70
5 1048	80
6 1047	80
7 1046	60
8 1046	70
9 1048	70
10 1045	60
11 1047	70
12 1046	70
13 1046	60
14 1047	70
15 1048	80
16 1046	70
17 1046	70
18 1047	60
19 1047	70
20 1046	60
21 1046	70
22 1046	60
23 1048	80
24 1046	70
25 1046	60

3 and 4. The introduction of drugs by intravenous and intratracheal injection has been occasionally tried, has given uncertain results, and has not been generally adopted.

Sulphur in the Treatment of Phthisis.

On the knowledge that sulphur when taken internally is excreted by the bowels, kidneys, skin and lungs, the writer tried the administration of this drug in a series of cases of phthisis, in the hope that the constant excretion of SO_2 by the lungs might exercise an antiseptic action on the growth of the tubercle bacillus. In going over the literature a reference, which was mislaid, was found which showed that two physicians in the early part of the eighteenth century had tried the effect of rectal injections of SO_2 , which they claimed was beneficial. In this instance the sulphur, a teaspoonful three times a day, after meals, was given by the mouth. The patients were all in later stages of the disease, being mostly advanced cases. Precipitated sulphur was the only drug or tonic given. As the patients were attending an outpatient clinique, it was not possible to take the temperature. The following were the clinical results.

CASE I.

A.E., female, aet 23. Mother died of phthisis and diabetes. Has been ill for four years. Has cough, spit, and haemoptysis 8 days ago, to the amount of half a pint. Pain in the left side. Has no appetite, is constipated, but menstruates regularly. Great night sweats, but as a rule sleeps well. Tubercle bacilli in sputum abundant.

Pulse 120. Weight 39 kilos. Right lung is normal.

The upper lobe of the left lung is consolidated, dull on percussion, with bronchial breathing, moist rales, and increased vocal resonance on auscultation. Inspiration is hoarse over the lower lobe, accompanied by dry clicking rales.

One month later, Patient has 4 motions per diem. Condition is worse, Pulse 130, weight 38 kilos. There are moist rales in the lower left lobe, and hoarse breathing over the right apex.

Two months later. Patient died in the interval.

CASE II.

A.G., railway man, aet. 40, complains of pain in the throat on swallowing. No family history. Had pneumonia two years ago, and one year ago "an influenza". Has been ill $1\frac{1}{2}$ years. Has had cough, and 10 ounces of watery sputum per diem. No haemoptysis. Appetite bad, and has lost weight. Sleeps well, but wakes with perspiration. Pulse 100, weight 47.4 kilos. Tubercle bacilli present.

The right upper lobe shows signs of consolidation, with an area of amphoric breathing below the outer third of the clavicle. Inspiration is hoarse over the left upper lobe.

Larynx. Vocal cords are swollen and oedematous, the false cords and the aryteno-epiglottidean folds being covered with tuberculous granulations.

One month later. Sputum very much diminished. No change in the pulmonary signs. Pulse 92. Weight 46 kilos. 3 motions per diem.

Two months later. Much worse. Daily vomiting after food. Pulse 82. Weight 44.6 kilos.

CASE III.

M.G. female, unmarried, aet. 26, complains of cough and pain. No family or previous history. Has been ill for three months. Has a great deal of cough, with sputum, which was tinged with blood a week ago. The appetite is poor, and there is vomiting after food. She has lost weight and is unable to work. The bowels are constipated, and she has not menstruated for three months. There are profuse night sweats. Tubercle bacilli are present. Pulse 96. Weight 45 kilos.

The breathing is broncho-vesicular over the upper left lobe, with loud rales and clicks on both inspiration and expiration. There are a few dry inspiratory clicks over the right apex.

Two months later. There is complete consolidation of the left lung, with bronchial breathing, increased vocal resonance, and fremitus. There is also consolidation of the right apex. Four motions per diem. Pulse 80, weight 43.2 kilos.

CASE IV.

F.G.R. clerk, aet. 26, complains of cough and weakness. No family history. Had influenza three years ago. Has been ill for one year. Coughs day and night, with a little sputum in the morning. No haemoptysis. He has pain in the stomach after food, and vomits in the evening after coughing. He has lost weight, and is easily tired. Bowels are regular. Sleep is disturbed by cough and night

sweats. Sputum contains tubercle bacilli. Pulse 74, weight 48.2 kilos.

There is dulness over the entirety of the upper right lobe, with bronchial breathing, increased vocal fremitus and resonance, with moist rales. At the apex of the lower lobe behind, there is hoarse broncho-vesicular breathing, with dry inspiratory clicks.

One month later. Cough is less and vomiting has ceased. No change in the lungs. He has difficulty in getting up sputum, which was tinged with blood three days ago. Sweats are diminished. Pulse 68, weight 48.6 kilos. 5 motions per diem.

Two months later. Disease has attacked left apex, where dry inspiratory clicks are audible. Pulse 64. Weight 48 kilos.

CASE V.

M.I. Fisherman, aet. 26, complains of being tired and hoarse. Two uncles died of phthisis. Has been ill for one year. Cough is frequent, with a good deal of sputum. No haemoptysis. He sleeps well, but has great night sweats. Appetite is good. Tubercle bacilli are present in the sputum. Pulse 125. Weight 47 kilos.

The right upper lobe shows broncho-vesicular breathing, with cog-wheel inspiration. No accompaniments.

At the apex of the lower lobe behind, there is dulness, bronchial breathing with moist rales, and increased vocal resonance and fremitus.

One months later. Has had 24 motions in the day. Lung condition unchanged. Sulphur reduced to 5 grs. t.i.d. Pulse 120. Weight 46.7 kilos.

Two months later. Disease has involved the left lung. There is broncho-vesicular breathing with fine crepitations over the upper lobe of the left lung. Pulse 90, weight 46 kilos.

CASE VI.

J.M.M., lad, aet 16, complains of weakness and cough. No family history. Has been ill for one year. Has cough, accompanied by pain in the front of the chest. Sputum in the morning. Appetite is good, and the bowels are regular. No night sweats. Tubercle bacilli are present. Pulse 130. Weight .22 kilos.

Over both apices, the breathing is broncho-vesicular, with dry inspiratory crepitations. The inspiration is hoarse over the apex of the lower right lobe.

One month later. The breathing is still broncho-vesicular at the apices, with medium moist rales, which are also heard at the apex of the lower right lobe. Pulse 100. Weight 21.5 kilos.

From these results it is certain that sulphur has absolutely no influence in checking the progress of the disease. They show, however, that a new therapeutic quality must be credited to it - that it slows the heart.

Other Specific Measures.

Hydropathy.

Winternitz 302 in 1899 advocated the treatment of phthisis by hydropathy in the form of cold douches. This treatment has yielded no results.

Artificial Pneumothorax

Fortanini ³⁰³ has advocated this method of treatment since 1882, and claims to obtain an absolute anatomical cure. It has not been tried in this country by recognised authorities, yet it may be worthy of trial, as every now and then a note appears in the medical press from someone who claims to have cured phthisis by this "new method".

The method consists in the injection into the pleural cavity of from 1000 to 1500 c.c. of sterilised air, sufficient to cause complete immobilisation of the lung, which is to be maintained by the further injection of 100 to 200 c.c. daily or on alternate days, and later by monthly injections of 200 to 300 c.c. The necessity for injecting air becomes less frequent, for the lung loses its expansibility, and further air is less readily absorbed, as the respiratory surface is diminished. Fortanini has not yet tried the method in early cases of phthisis, but recommends that it be practised in cases that do not yield to ordinary treatment. After a cure is obtained it has to be determined whether the pneumothorax should be continued, or whether the air should be allowed to be absorbed and the lung to expand.

When the whole of one lung is attacked, but the other remains healthy, he considers that the pneumothorax should be indefinitely maintained, as at present it has not been determined what length of time is necessary for permanent immobilisation of the lung by pneumothorax. If both lungs are affected, the pneumothorax is to be kept up on the side most affected, to allow the best chance of the healing and expansion of the other lung.

He finds that as the method means restoration to health, patients readily submit to the treatment.

Fortanini believes that the difference in rapidity between pulmonary and surgical tuberculosis, is due to the constant changes of pressure in the lungs. Pneumothorax rests the lung, and obliterates cavities, the walls of which come into contact and are united by fibrous tissue, which is associated with the disappearance of bacilli from the sputum, which finally ceases.

It is useless in cases where both lungs are greatly affected, and also where the pleural cavity is obliterated by adhesions.

Accepting the above statements with all reserve, one may yet believe, in view of the occasional improvement which follows the natural appearance of pneumothorax in phthisis, that the method is worthy of trial.

In this chapter we have only considered those specific measures which have been seriously tried in this and other countries. From time to time, the wildest proposals are put forward, such as the daily inhalation of ether, and other schemes, which add to the gaiety of the profession.

CHAPTER XVII.

KOCH'S TUBERCULIN T.R. IN THE TREATMENT OF PHTHISIS

Tuberculin T.R. is a pale opalescent fluid, l.c.c of which contains 10 mgr. of the debris of Tubercle bacilli in a 20% solution of glycerine. It is prepared by filtering the bacilli from a bouillon culture, washing them in normal saline, centrifuging, pouring off the supernatant fluid, grinding down the bacilli with ground glass, washing again and suspending the bacterial debris in a 20% solution of glycerine. This gives a solution which is free from the exotoxins of old tuberculin, and only contains the endo-toxins which are combined with the protoplasm of the bacilli.

The writer has investigated the properties of this Tuberculin, firstly with regard to its effect on the Opsonic Index ³⁰⁴, and secondarily as to its value in the treatment of phthisis.

It has been frequently stated that the injection of a therapeutic dose of tuberculin is invariably followed by a fall in the opsonic index lasting from three days to three weeks. This period of low opsonic index is generally known as the negative phase and its duration varies with the amount injected. This negative phase is followed by a period of high opsonic index, which is known as the positive phase, and lasts for a few days, after which the index returns to about its original level.

It is generally believed that by reducing the dose to the minimum that will produce any effect the negative phase may be reduced and the positive phase correspondingly increased. The doses which have been most frequently used to produce these results range from $\frac{1}{1000}$ to $\frac{1}{100}$ milligramme or even more.

With a view to gaining more precise knowledge of the effect of a therapeutic injection a phthisical patient was injected with $\frac{1}{100}$ mgr. of Koch's new tuberculin (T.R.) and the opsonic index estimated hourly for several hours with the following result.

C H A R T I

1907.	Index.
Feb. 16th	0.75
" 17th 9.a.m. (injected 1/100 mgr)	0.75
" 17th 10.a.m.	0.75
" 11.a.m.	0.97
" 1.p.m.	1.5
" 2.p.m.	1.3
" 3.p.m.	0.78
" 19th	0.5.
" 20th	0.4
" 22nd	1.5
" 23rd	1.38
" 27th	1.39
" 28th	0.76
" 29th	0.8

It will be observed that a rapid rise of the opsonic index occurred within two hours of the injection; it rose to a maximum in four hours, fell to its original level in six hours, and was succeeded by the usual negative and positive phases. This initial rise may be called the pre-negative phase and has hitherto been overlooked

owing to the fact that indices are rarely taken more than once a day. This pre-negative phase has been independently observed and recorded by Clive Riviere ³⁰⁵.

In order to explain these facts it is necessary to assume that when tuberculin is injected into the body the opsonins present in the blood are absorbed by the bacillary debris. Fresh opsonins are then produced and as rapidly absorbed and it is thus that the negative phase is produced. But in accordance with the law of regeneration more opsonins are produced than suffice to replace those absorbed and sooner or later a point is reached at which the tuberculin is saturated. More and more opsonins are produced until the index begins to rise and the positive phase occurs. This theory will explain the negative and positive phases but not the initial rise. To explain this phase we must assume that at the moment of injection a minute fraction of the dose found its way immediately into the circulation, probably owing to rupture of some capillaries. This minute dose absorbed sufficient opsonin to set in motion the mechanism of immunity but not enough to cause more than an exceedingly minute and transitory fall in the index. The index accordingly rose rapidly. The great bulk, however, of the injection remained at the site of inoculation until by dilatation of the capillaries and transudation of lymph it too was swept into the general circulation. This occurring some hours later the excess of opsonins produced were again absorbed and the negative phase set in.

If this explanation be correct, then it follows that if a sufficiently small dose be given the negative phase will be absent or so slight and transitory as to be negligible and the injection will be followed by an immediate rise. The writer tested the truth of this explanation in five patients on 14 different occasions by injecting a dose of one-millionth of a cubic centimetre of tuberculin in a bulk of 1.7. minims. On every occasion the injection was followed by an immediate positive phase lasting from two to four days, after which the index resumed nearly its former level and the negative phase was absent. In one case, that of a child aged ten years, the positive phase continued to the sixth day.

C H A R T I I

<u>Day</u>	<u>Index</u>	<u>Dose</u>
1st	0.44	0
2nd	0.5	$\frac{1}{1,000,000}$ c.c. T.R.
3rd	1.1.	0
4th	0.95	0
5th	0.7	0
6th	0.75	$\frac{1}{1,000,000}$ c.c. T.R.
7th	1.0	0
8th	1.0	0
9th	1.1.	0
10th	0.35	$\frac{1}{1,000,000}$ c.c. T.R.
11th	0.81	0
12th	0.8	0
13th	0.5	0
14th	0.55	0

The next question which arose was whether by repeated injections one positive phase could be superimposed upon another, and if so how far could the process be carried.

With this object a dose of one-millionth of a cubic centimetre of tuberculin was injected into two patients on six successive days with the following results.

CHARTS III and IV

	Dose	Index	Dose	Index
1st Day	0	0.66	0	0.75
2nd day	1	0.65	1	0.5
3rd day	1	1.2	1	0.9
4th day	1	1.49	1	1.37
5th day	1		1	
6th day	1	1.4	1	1.8
7th day	1	0.59	1	0.6
8th day	0	0.56	1	0.6
9th day	0	0.68	1	0.7

It will be seen that the first three doses were followed by successive rises in Case A. and the first four in Case B. In both cases the fifth injection was followed by a rapid fall to below the original level. Here the rise was higher and more prolonged than any previously produced in the same patients by a single injection and it seems reasonable to assume that this was due to a super-imposition of positive phases. It is, however, obvious that this piling up of positive phases is only possible within very narrow limits. The mechanism of immunisation is one of delicate adjustment and is easily deranged. Apparently a very slight excess of

tuberculin suffices to turn the scale and convert an over-production of opsonins into a deficit.

The writer repeatedly tried the effect of injecting tuberculin according to the method suggested by Weber's law. This method has for its foundation the fact that in some classes of phenomena in order to produce effects increasing in arithmetical progression it is necessary to use stimuli increasing in geometrical progression. This method appeared to be successful in certain cases but the results are inconclusive. As an instance of the result produced by these means the following observations may be taken.

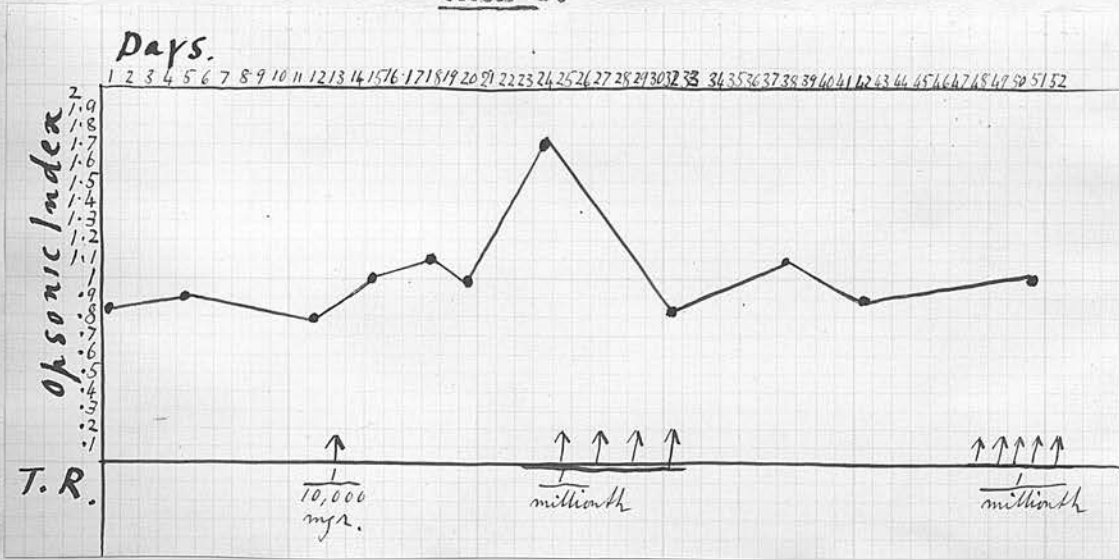
C H A R T V.

	Index	Dose millionths of 1 mgr. T.R.
1st Day	1.5.	0
2nd "		160
3rd "		320
4th "		640
5th "		1280
6th "		2560
7th "	0.82	5000
8th "		10000
9th "	1.0	0
10th "	1.1	0
11th "	1.75	0
12th "		0
13th "	1.5	0
14th "	1.72	0
15th "	1.55	0
16th "	2.6	0
17th "	1.18	0
18th "	1.12	0
19th "		0
20th "	1.62	0
21st "	1.22	0
22nd "	0.86	0

From this table it would appear that it is possible by rapidly increasing doses to raise the opsonic index for a considerable period. One is not, however, justified in stating this as a fact universally true.

These results are taken from observations on thirty-two tuberculous patients over a period of six months, during which time nearly 500 opsonic estimations were made. In general, we may conclude that if a high opsonic index is associated with a beneficial action on the lesions, it is necessary to estimate the opsonic index as a guide to the dosage of Tuberculin, lest the index is depressed instead of raised.

The following cases show the dosage of Tuberculin, the Opsonic Index, and the influence of this treatment on the weight of the patients. No reference is made to the lesions, as the patients were all early cases, and deductions from alterations in the signs over a short period are quite unreliable.

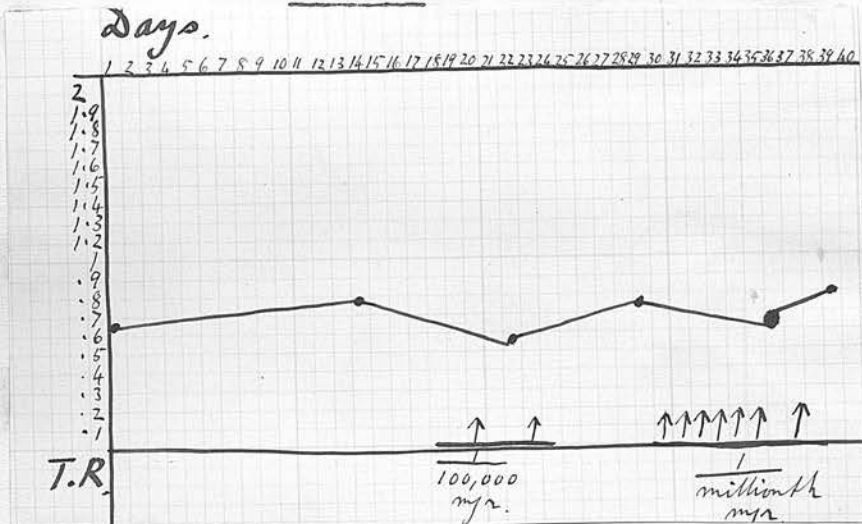
CASE I.

Number of days under treatment - 52.

Weighed at start - 9 st. 13½ lbs.

Weighed at end - 9 st 12½ lbs

Loss of weight ¾ lb.

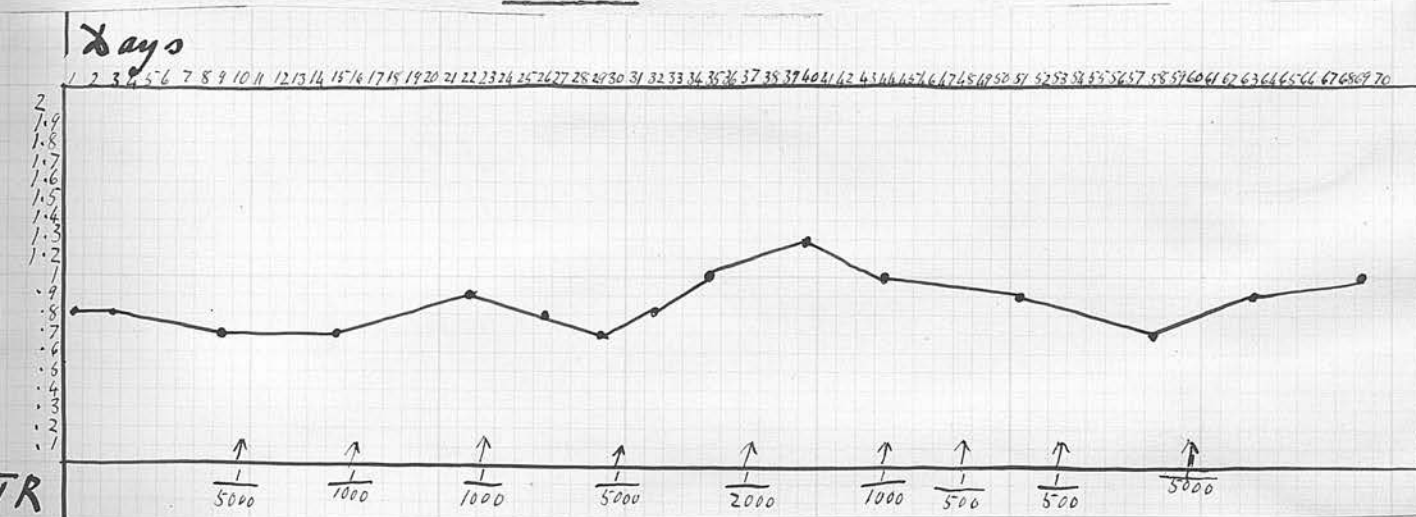
CASE II

Number of days under treatment 42

Weighed at start - 9 st 11½ lbs.

Weighed at end - 9 st. 13 lbs.

Gain in weight 1½ lbs.

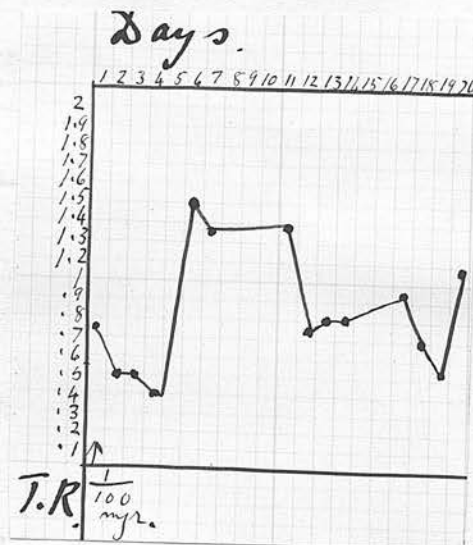
CASE III

Number of days under treatment - 70

Weighed at start - 8 st. 2½ lbs.

Weighed at end - 8 st. 13¼ lbs.

Gain in weight 10¾ lbs.

CASE IV

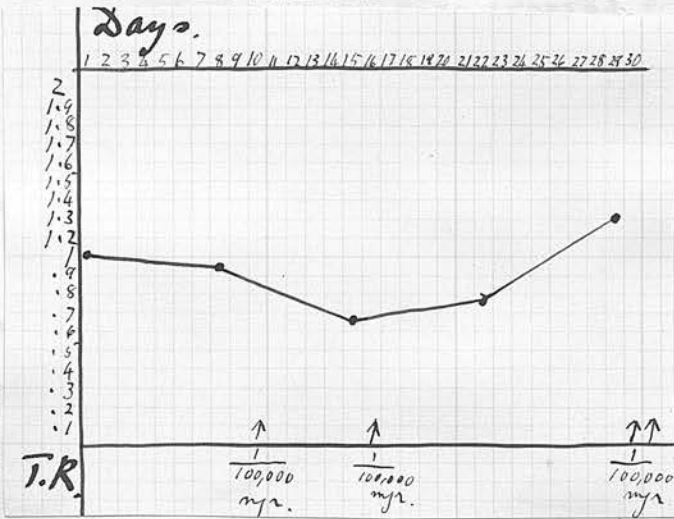
Number of days under treatment - 20

Weighed at start - 8 st. 6 lbs.

Weighed at end - 8 stone 8½ lbs.

Gain in weight 2½ lbs.

CASE V.



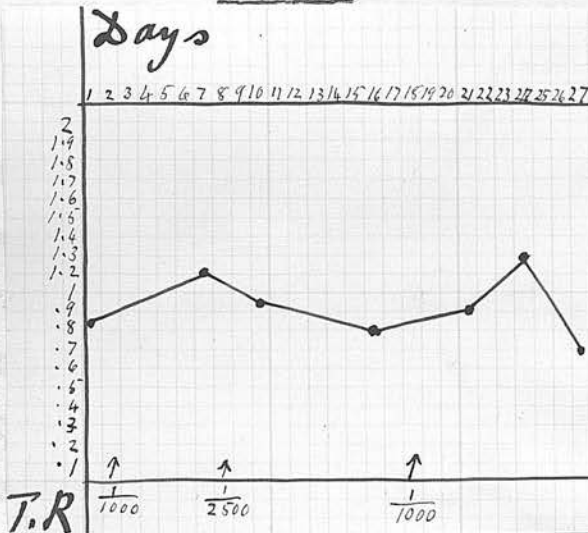
Number of days under treatment - 31.

Weight at start - 7 st 2 lbs.

Weight at end - 7 st. 2 lbs.

No gain or loss.

CASE VI



Number of days under treatment - 37

Weight at start - 10 st. 3 lbs.

Weight at end - 10 st. 5½ lbs.

Gain in Weight 2½ lbs.

These six were selected cases in the first stage of phthisis, and were kept in bed during the treatment.

In the dosage of Tuberculin, the opsonic index was the guide, and most of the cases were treated for much longer periods than are given in the above charts, which are selected as covering the period between two successive weighings. The total gain in weight of six cases in 252 days was $15\frac{1}{2}$ lbs. As a control we take the loss or gain in weight of 21 patients in all stages of phthisis over varying periods, and who were not treated with Tuberculin.

A comparison of the results is seen from the following tables.

TABLE I.

Patients treated with Tuberculin.

Case	Days	Gain	Loss	Average per month
1.	52		$\frac{3}{4}$ lb	.41 lb -
2.	42	$1\frac{1}{2}$ lbs		1.07 lbs
3.	70	$10\frac{3}{4}$ lbs		4.6 lbs
4.	20	$1\frac{1}{2}$ lbs		2.2 lbs
5.	31			
6.	37	$2\frac{1}{2}$ lbs		2.03 lbs
6	252	$15\frac{1}{2}$ lbs		Average gain per month per case 1.58 lbs

TABLE II

Patients not treated with Tuberculin

Case	Days	Gain	Loss	Average per month
1.	48		5 lbs	3.12 lbs - .
2.	39		2 lbs	1.53 lbs - .
3.	20		7½ lbs	11.15 lbs - .
4.	27	16¼ lbs		18 lbs +
5.	27	1 lb.		1.1. lb +
6.	14	5½ lbs		11.25 lbs +
7.	37	17¼ lbs		13.98 lbs +
8.	83	2½ lbs		.77 lb +
9.	27	¾ lb		.83 lb +
10.	88	14¾ lbs		5.02 lbs +
11.	9	2¼ lbs		7.5 +
12.	36	4¼ lbs		3.54 +
13.	77	9¼ lbs		3.62 +
14.	28	1 lb		1.1. +
15.	28	5 lbs		5.32 lbs +
16.	42	9 lbs		6.42 lbs +
17.	53	18¾ lbs		10.61 lbs +
18.	18	6¾ lbs		11.25 lbs +
19.	34	7¼ lbs		6.39 lbs +
20.	33	23¼ lbs		21.13 lbs +
21.	27	7 lbs		7.7 lbs +
21.	895	10. st 11 lbs		Average gain per month per case 5.22 lbs.

When we remember that those treated with Tuberculin were selected cases, the results, as seen by the weights, are extremely disappointing. Further, it was found that a rise in the Opsonic Index had no influence on the temperature, and with regard to this treatment in general the writer has come to the following conclusions:

It is impossible to control the Opsonic Index by injections of Tuberculin T.R., as auto-inoculation of bacterial debris is constantly going on in the lung, and is increased by mental or physical exertion, depending on the increased blood flow through the pulmonary circulation.

If the injection of bacterial debris was a specific for phthisis, the disease would be non-existent, for whenever the bacilli settles in the lung, auto-inoculation of bacterial debris commences, which makes a high index one of the earliest signs of the disease. In those who do not develop phthisis in spite of the bacilli entering the body, it is probable that this auto-inoculation causes a large enough production of opsonins to overcome the infection at the start. If nature is incapable of doing this, it is not to be attained by mechanical means.

In surgical tuberculosis, in which Tuberculin T.R. yields excellent results, there is no auto-inoculation from the lesions, as is manifest by the absence of fever in this form of the disease. Here the injection of

small doses of the bacilli produces an increased amount of opsonins in the blood, which find their way to the local lesion.

We know less of the effect of Tuberculin in phthisis if the opsonic index be not taken than if it be estimated and the labour of doing this is sufficient to preclude the method coming into general use.

If it be given at all, it should be where the lesions are quiescent, and in doses of from $\frac{1}{100,000}$ mgr to $\frac{1}{10,000}$ mgr., the opsonic index being estimated *pari passu* with the injections, which should not be more frequent than one per week.

This is the method of vaccine treatment in phthisis as outlined by Wright ³⁰⁶ who suggests that auto-inoculation be controlled by rest and by increasing the coaguability of the blood, after which the amount of opsonins around the lesions are increased by graduated exercises and by reducing the coaguability of the blood. Wright ³⁰⁷ and Paramore have shown that Calcium lactate in doses of 15 grs three times a day increases the coagulative power of the blood, which can also be diminished by citric acid.

CHAPTER XVIII.

OTHER VACCINES, TUBERCULINES AND SERUMS IN TREATMENT

Acquired immunity to a bacterial disease may be active or passive. Active immunity may be attained by the introduction of a modified form of the disease into the body. Such was the method of Jenner, who found vaccinia to give immunity from small-pox. So far as tuberculosis is concerned, vaccination has only been practised on the bovines, being first tried in 1901 by Von Behring. The results have not been certain.

Immunity may also be acquired by the introduction of small doses of bacterial toxin into the body. According to Pliny, King Mithridates attained an immunity to certain poisons by this method, and in modern days it has given us the anti-diphtheritic serum. The toxins of the Tubercle Bacillus in the form of varieties of Tuberculin have been given to phthisical patients in the hope of inducing such an immunity in their systems. There are, however, two great differences between the action of diphtheria toxin on the horse and that of Tuberculin on a phthisical patient. In the one instance we are dealing with a healthy animal, in the other with a body already containing the bacteria and toxins. A horse immune to diphtheria toxin is also immune to the diphtheria bacillus, which only acts by virtue of its

toxins. This dual immunity is not to be attained by the use of Tuberculin, the tubercle bacillus has a general and a local action.

The theory of the action of Tuberculin and Vaccines rests on the following considerations.

It has been proved that the dead or living tubercle bacillus, or its toxins, when introduced into the animal organism produce specific protecting substances. These are of the nature of anti-toxin, bacteriolytic, and agglutinating bodies, which can be demonstrated in the tissues of the organism.

These specific bodies are found in the blood plasma in the leucocytes, in the tissues, in exudations, in milk and in eggs. It is held that if the bacillus or toxin be introduced into the organism of a tuberculous patient, the same specific substances will be produced, and so exercise a healing influence.

That is to say by introducing small doses of the toxins and bacilli with which the patient is already saturated, it is hoped to produce an immunity. We may call it Mithridatisation, but essentially it is homoeopathy. The chief criticism of this theory is the answer to the question: "How does phthisis arise?" It arises by the tubercle bacillus settling in a body incapable of producing protective substances in sufficient amount to overcome the infection. How then, if the healthy organism was powerless to produce enough anti-bodies are the diseased tissues to elaborate these specific elements?

The following preparations of the bodies and toxins

of the tubercle bacillus have been used in accordance with the above theory.

1. Koch's Tuberculin. O.
2. Koch's Tuberculin T.R.
3. Koch's Bacillary Emulsion.
4. Klebs Tc. - Selenin
5. Hirschfelder's "Oxy-tuberculin".
6. Ruck's Tuberculin.
7. Von Behring's Tulase.
8. Raw's Bovine Tuberculin.

The second variety of acquired immunity is passive immunity. This is obtained by introducing into the system of the diseased animal, the serum of another animal which has been highly immunised to the disease. The great victory in combating disease by this method is illustrated by the anti-diphtheritic serum. The following serums, obtained by immunising animals against the tubercle bacillus, have been tried in the treatment of phthisis.

1. Carroll's.
2. Maragliano's.
3. Marmoreck's.

We now proceed to a more detailed consideration of these methods of treatment.

OLD TUBERCULIN.

Koch's Old Tuberculin is a solution of the exotoxins of the tubercle bacillus, prepared from a bouillon culture of the bacillus. When first introduced the idea was to induce inflammatory, degeneration, and neurotic changes at the seat of the lesions. This it undoubtedly did with disastrous results to the patients.

Its more recent advocates told that the injection of small doses, will cause anti-toxic substances to be formed in the blood. Thus Sahli ³⁰⁸ does not regard its action as specific, in the sense that anti-diphtheritic serum is specific, but holds it is able to mithridatise or immunise the phthisical patient to a certain degree against the toxins of the bacillus.

He believes a tuberculous focus is healed by a purely local process consisting of the neutralisation of the tubercular toxins by substances formed in the damaged lung from protoplasm of the blood lymph and cellular products of tuberculous tissue. These substances saturate the tubercle bacillus, and change it from a parasite to a saphrophyte, after which comes leucocytosis, increased oxidisation from increased circulation, and the production of fibrous tissue at the periphery of the lesion.

Sahli regards these substances as specific and formed locally, and believes their action is stimulated by Tuberculin, as this produces anti-bodies in the system, which means a cessation of the loss of appetite, general

weakness, diminished nutrition, defective circulation and fever, all of which are manifestations of the toxins of the bacillus, and preclude the local action of repair.

Mithridatisation immunises the organism against the toxins, abolishes the general derangements and permits a local cure to be effected. Sahli also thinks that it may increase the natural faculty of the organism to produce anti-bodies, which combine with the toxins. He claims to have obtained favourable results in early cases without fever.

It is generally admitted that Tuberculin should only be given in early quiescent cases without fever.

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The principles of its use are stated by Helm as follows:

1. The tuberculin treatment can only be employed in the case of patients who are free from fever, whose strength is well-preserved and in whom the destruction of the lung-tissue is not too far-advanced. To submit patients who are subject to feverishness and bleeding to a course of tuberculin treatment is justifiable only where the physician has had many years of specialistic experience. Patients with heart-complaints, diabetes, kidney and nerve troubles are to be excluded from the tuberculin treatment.

2. Before commencing the tuberculin treatment, the temperature of the body must be measured every three hours for several days. Temperatures between 37.0 and

4. The tuberculin treatment is best administered and 37.5° Celsius are not to be considered insignificant. The sputum must moreover be examined for tubercular bacilli, and the urine for albumen and sugar.

3. After the injection has been made, the temperature must be measured every 3 hours until the cessation of the reaction, which may last from 2 to 3 days; it is possible for the fever reactions to delay their appearance, and care must be taken that reactions which set in during the night do not escape observation. In addition to the fever reaction the local reaction must also be watched, the latter frequently lasts several days longer than the former.

4. The most extreme caution must be observed in the matter of increasing the dose; if the reaction has been strong, the dose should not be increased at the next injection, but should be repeated in the same measure, or even diminished in strength. It is impossible to draw up a table regulating the doses of tuberculin and the period which must elapse between the separate injections. Each patient must be individually treated.

5. In cases where abnormal sensitiveness can be observed, and where the feverish reactions are of long duration, the tuberculin treatment must for a time be abandoned; whether a second attempt may safely be made depends before all upon the general health of the patient (as shown by the temperature, the weight of the body or by the results of an examination of the blood)-

6. The tuberculin treatment is best administered in stages i.e. with interruptions of weeks or months, and must be several times repeated. Each fresh stage must begin with a diagnostical injection.

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Trudeau one of the best known advocates of Tuberculin in phthisis, relies on this method by producing well marked toxin immunity by the use of small and increasing doses, so establishing an immunity to the doses, and preventing the aggravation of the disease which too rapid an increase in dosage is apt to produce.

He judges the dose, intervals of injections, and length of treatment, entirely by the clinical manifestations of each case, in which all marked or general reactions are avoided. His initial dose is $\frac{1}{1000}$ mgr of old Tuberculin, a gradually increasing amount being given every three days or every six days, and where there is no reaction the treatment lasts for six months at the end of which time the patient is able to tolerate an injection of 1 c.c. of the pure filtrate.

311

The following solutions, as used by Deny's are extremely useful for dosage. Each solution consists of 1 c.c. No. 1 contains $\frac{1}{10000}$ mgr (for febrile cases only). No 2 $\frac{1}{1000}$ mgr. No III $\frac{1}{100}$ mgr. No IV $\frac{1}{10}$ mgr. No. V. 1 mgr. No XI 10 mgr No VII 100 mgr., and No VIII is pure filtrate. At each injection, the dose is increased by .1 c.c. so that the increase in the first ten injections is by $\frac{1}{10,000}$ mgr for the next ten injections by $\frac{1}{1000}$ and afterwards by $\frac{1}{100}$ mgr until 1 c.c. of the pure filtrate is given.

If intolerance is shown by fever, local reactions, or general impairment, the injections are stopped until these signs have disappeared, and on commencing, one half of the last dose should be used. If reactions occur, the treatment may be prolonged for a year.

Trudeau concludes:

"Chronic and incipient cases respond well to treatment but in acute cases it is well to wait until a partial arrest shows itself under rest and open-air treatment before beginning the use of tuberculin. Cases with long-continued increasing fever, and cases with extensive lesions or intestinal or other complications, or very feeble and badly-nourished cases, as well as those showing marked intolerance, are unsuitable for treatment. The production of tuberculin immunity by this mild clinical method influences favourably the course of chronic tuberculosis, prolongs life, and often aborts a commencing infection."

312

Roemisch, who has extensively used Tuberculin, finds as follows:

"The cases which are best adapted for the treatment are chronic, mostly advanced cases which for some time show no tendency of improvement, but which previously showed a diminution of the limits of disease and an absence of local reaction. In one patient whom he treated in 1900 and again in 1902, a permanent cure was attained in spite of extensive disease of the right lung. The patient is now objectively and subjectively well. A second case reports of himself that he is quite well,

add of this patient Roemisch states that the result has surprised him not a little. Four other cases are quoted to show definite lasting cures of phthisis by means of tuberculin. In all these six cases the conditions of the cure was unfavourable. He also reports of seven less severe cases, treated before 1902, all of which have lost all signs of disease. One of these cases, however, offers an anatomical confirmation for the cure of the disease. The patient committed suicide, and the post-mortem examination which was performed by Turban at Davos, revealed firmly-scarred tissue, without any signs of active tuberculosis. In treating his cases, he aims at obtaining a mild local reaction without any severe general disturbance. If one injects doses which are too small, the whole treatment does no good, while if one exceeds a dose which is just sufficient to produce the mild reaction and especially if one repeats this dose several times; one runs the risk of producing great damage. The signs that the limit is nearly reached include feeling languid, loss of appetite, disturbance of sleep, various forms of pain, and increased quantity of sputum. Slight elevation of temperature even if this is only one or two tenths of a degree centigrade, must be taken note of if the temperature has previously been steady. When such symptoms occur, Roemisch repeats the same dose instead of increasing it, or if the signs are unmistakably present he gives a smaller dose. He believes that the therapeutic effects gained when small doses produce a slight increase of râles are highly important. He has come to the conclusion that a certain number of cases which resist all

other forms of treatment often do well under the tuberculin treatment, and regards improvement up to a certain point, and then persistence of the signs of disease, as an indication for the treatment. Tuberculin does not render open-air treatment and other means superfluous, but must be regarded as a very valuable addition to the therapeutic agents which we have at our disposal against pulmonary tuberculosis."

313

The following is a summary of the results and opinions of other authorities with Tuberculin.

Von Ruck in 2000 cases had "satisfactory results" Pottenger with 400 cases cured 92% in "I Stage", 68% in "II Stage" and 8% in the "III Stage".

At Massachusetts State Sanatoria of 58 cases treated, a third were "apparently improved."

Rosenberg finds Tuberculin makes the "duration of treatment shorter".

Porter gave Tuberculin to 50 patients "some of whom were harmed and a few did well."

Miller found of 25 patients treated, 5 did well 2 poorly.

314

The following are Trudeau's tabulated results of those cases, who were treated for a period of over three months, and whose sputum contained tubercle bacilli at the start.

A comparison of 185 patients treated with tuberculin and 864 cases treated without tuberculin showed on dis-

charge the following:

	<u>Incipient</u>			<u>Advanced</u>		
	Apparently cured	Disease arrested.	Active	Apparently cured	Disease arrested.	Active
Treated with tuberculin.	56%	34%	10%	27%	55%	18%
Treated without tuberculin.	50%	38%	11%	6%	51%	43%

A comparison of 135 patients treated with tuberculin and 690 treated without it, showed:

	<u>Incipient</u>		<u>Advanced.</u>	
	Living	Dead	Living	Dead
Treated with tuberculin	79%	21%	61%	39%
Treated without tuberculin	63%	37%	36%	64%

We may conclude that Tuberculin is beneficial in raising the resistance of latent phthisis, if given by skilled hands, under extreme precautions:

OXY-TUBERCULIN

Hirschfelder in 1897 announced the discovery of this Tuberculin, prepared from old tuberculin by hydrogen peroxide and heat. It has neither given results nor is it used at present.

KLEB'S Tc. - SELTENIN.

315
Klebs states that this treatment "is based upon the recognition of the fact that there exist in the various derivatives of the tuberculin cultures harm-

ful as well as beneficial stuffs, toxin and sozin. By the removal of the former - by means of sodium bismuth iodid - an innocuous, bactericidic substance, viz: tuberculocidin (Tc) was produced.

The efficacy of Tc in the case of animals as well as of men, consists in the destruction of the tubercular bacilli, even where these have already produced high-grade tuberculous changes.

Since every case of human tuberculosis represents a mixed infection the Tc treatment has - since 1900 - been materially improved by the utilisation of other bactericidic substances in conjunction with the tuberculocidic - especially of those acting upon the most ordinary concomitant of tuberculous infection, the diplococcus semilunaris mitri (D.K. catarrhalis Pfeiffer), namely of Selinin, a solution of bodies of cocci, deprived of their toxines by $H_2 O_2$.

These two substances, Tc and selenin, when used internally, stomachically or rectally, may be administered in small quantities (2 ccm Tc and 4 ccm sel., once a day) and will suffice to bring about a complete cure in all cases (100%), in the first stage of the disease - tuberculosis of the lymphatic glands, scrofula. In the second stage - non-ulcerous tuberculosis of the organs, so-called close tuberculosis - it succeeded in achieving cures to the extent of 90% - several of which had been determined as tuberculosis by six years' continuous observations - and 10% almost complete cures.

For severe cases of ulcerous tuberculosis, characterised by the appearance of free tubercular bacilli - stages III and IV - this treatment is not always sufficient.

"Larger doses" here must be administered, up to 40 and 50 ccm Tc 1% and 40 cmm selenin 1% daily (-4 to 5 ccm Tc 10% and 4 ccm selenin 10% -) which, when administered stomachically, are readily assimilated and usually have an anti-febrile effect.

In individual, especially refractory cases, this treatment must be supplemented by the intercurrent introduction - likewise rectal or stomachic - of an immunising substance, tubercle protein Hpp - Hydrogenio peroxydato paratum."

This preparation is at present on its trial in Germany, but it is unlikely the results will be startling.

RUCK'S TUBERCULIN.

In 1898, Ruck prepared a watery extract of tubercle bacilli, by extracting the bacilli with water, ether, and alcohol, then drying, pulverising, and re-dissolving. Such a solution contains the soluble endo-toxins of the bacillus. It is claimed in America that Von Behring's "Tulase" is prepared in an identical manner.

VON BEHRING'S "TULASE"

316

Von Behring described "Tulase" as follows "Tulase" is a clear liquid, containing none of the protoplasm of the bacilli, colourable by Ziehl or Gram's stain. It

is made by treating the bacilli with chloral. After this treatment the Tc of the bacilli becomes so modified that on being subcutaneously or intravenously injected, or even on being injected, the cells of the body transform it into the hypothetical substance T.X., which confers an immunity against tuberculosis, and a hyper-sensitivity to old Tuberculine."

It is impossible to conceive what exactly this substance is, and it has yielded no results.

RAW'S BOVINE TUBERCULIN

317
Raw believes that bacilli of the Human type produce phthisis, ulceration of the intestines, and abdominal glands, while bacilli of the bovine type produce Tubercular Peritonitis, Tuberculosis of the lymphatic glands, Tubercular bones and joints, Tubercular Meningitis and Lupus.
He also assumes that bovine and human bacilli have an antagonistic action the one on the other, and by this explains the good results of Tuberculin T.R. on surgical tubercle. On this same assumption, he is treating phthisis with a bovine Tuberculin. This assumption is pure theory, and has no basis of experimental proof. If the bovine vaccine succeeds in phthisis, it is more likely to be because phthisis is a disease of bovine origin.

SERUMS.

Carroll's Serum was introduced in America, but has fallen into disuse.

Maragliano's Serum is chiefly employed in Italy. The 2nd Annual Report of the Phipps' Institute reports 15 cases, in which no noticeable results were obtained.

Marmoreck's Serum.

This has been used in Germany and America in Phthisis. 318
 Richer thinks it may give "some passive immunity". It has been found of value in Surgical Tuberculosis by 319 Hoppa and in Laryngeal Tuberculosis by Weil 320 but its use has been attended by symptoms of profound 321 toxaemia. (Baer).

INJECTION OF CHOLESTERIN

On the fact that cholesterin has an anti-toxic effect 322 on snake venom, Lemaine and Gerard tried the effect of cholesterin on monkeys inoculated with tubercle, and claim that it regards the process of tuberculisatation. A hospital patient in the last stages of phthisis was said to be greatly benefited, a statement which shows how the Spes Phthisica seems to hover about the treatment of the disease.

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CHAPTER XIX

ON SANATORIA.

Whatever be the position of Sanatoria as a factor in the cure and diminution of phthisis, they must receive the credit of having constituted, since 1894, a powerful demonstration of the value of hygienic methods. While it is impossible even to approximate to what extent Sanatoria treatment has prolonged the duration or diminished the incidence of phthisis, there is no doubt whatever of the immediate benefits of this treatment, so that thousands who pass through Sanatoria every year are convinced of their utility and determined to practise a hygienic mode of life. Further, as up to quite a recent date it was generally believed that sputum by its dissemination through the air constituted the chief means of propagating phthisis, the destruction of and precautions against expectoration, have been rigidly taught in these institutions. While doubts have arisen as to the aerogenous theory of infection, more evidence has accumulated to show the great dangers of contact of sputum with food, and therefore the spread of knowledge as to the dangers of expectoration to the community at large, is to be reckoned as aiding the prophylaxis of the disease.

Not only so, but as the result of treatment is far better in early cases, the rise of Sanatoria has stimulated the art of early diagnosis, and has thus indirectly influenced the curability of the disease.

The great ignorance which prevails generally as to hygienic conditions of life gives Sanatoria a utility of raising the general standard of health in the community, which cannot fail to be appreciated in those countries where they exist, as in Germany where thirty thousand persons pass through them every year.

We may consider the number and class of Sanatoria in England and Scotland, in relation to the amount of phthisis in the community.

The following table gives the name of the Sanatoria, the number of patients taken, the cost per bed, and the charge per week per patient.

TABLE I.

County	Sanatorium	No. of beds	Cost per bed	Charge per week
ENGLAND				
Bedfordshire	Daneswood	24	£1000	Free
Berkshire	Pinewood	60	£700	£3. 3.
Cheshire	Manchester Consumptive Hospital	50		18/6
"	Heswall	21	£500	Free
"	Liverpool	40	£375	12/6-£1.
"	Crossley	90	£600	Free -£2.2.
Cumberland	Blencathra	28	£240	30/- -£2.2
Devon	Dartmoor	8		£4. 4.
"	Devon & Cornwall	35	£370	£1.11.6
"	Dunston Park	10		£4. 4
"	Mildmay Home for Advanced cases	10		7/- -10/6.

County	Sanatoria	No. of beds	Cost per bed	Charge per week
ENGLAND (Continued)				
Devon	Western Hospital	40		7/6-12/6
"	Udul Torre	13		£4. 4
Durham	Belle Vue	20		£4. 4.
"	Durham	45		Free
Essex	Coppins Green	20	£80	25/-
"	Maldon	12	-	£4. 4.
Gloucestershire	"Birmingham"	40	£420	
"	Cotswold	33	-	£4. 4.
"	Painswick	12	-	£4. 4.
Hampshire	Alderney Manor	25	-	£4. 4.
"	Firs Home	20	-	10/6
"	Hahnemann Home	32	-	7/6 -21/-
"	Home Sanatorium	45	-	£4. 4.
"	Linford	24	-	-
"	Moorecote	10	-	£4. 4.
"	National Sanatorium	85	-	Free
"	Overton Hall	12	-	£4. 4.
"	St Joseph's Con- valescent Home	72	-	8/--12/-
"	Stourfield Park	40	-	£4. 4.
Isle of Wight	Royal National Hospital (Ventnor)	155	-	10/-
"	St Catherine's Home (Ventnor)	12	-	10/6
Kent	Benenden	68	£100	Free
"	M.A.B. East Cliff Home (Margate)	130	-	Free
"	Royal Sea Bathing Hospital (Margate)	162	-	22/-

County	Sanatorium	No. of beds	Cost per bed	Charge per week
ENGLAND (Continued)				
Kent	Victoria Home for Invalid Children (Margate)	46	-	5/-
"	Sandgate Homes		-	£4. 4.
Lancashire	Clayton Vale Small-pox Hospital	32	-	Free
"	Crossley "Home of Peace"	25	-	Free
"	Liverpool Hospital for Consumption	30	-	5/- -12/6
"	Manchester-Out-patient Department		-	-
"	Moor End, Sheffield	20	-	Free
London	Brompton	318	-	Free
"	City Road	80	-	Free
"	Free Home for the Dying		-	Free
"	Friedenheim Hospital	48	-	Free
"	Home for Consumptive Females	23	-	8/-
"	Hospital of St John and Elizabeth	17	-	Free & £4.4
"	Margaret Street Hospital		-	Free
"	Mount Vernon	140	-	Free
"	St Joseph's Hospital	48	-	5/6
"	St Luke's House	28	-	Free
"	St Peter's Home, Kilburn	12	-	5/- -21/-
"	Victoria Park	164	-	Free
Middlesex	Northwood	100	£1000	Free
Norfolk	The Beeches (Long Stretton)	6	-	£4. 4.

County	Sanatorium	No. of beds	Cost per bed	Charge per week
ENGLAND (Continued)				
Norfolk	The Children's Sanatorium, Holt	15	-	Free & 15/-
"	Kelling	51	£200	-
"	Mundesley	19	-	£4. 4.
"	Southrepps	7	-	£4. 4.
Northumberland	Barrasford	50	£270	-
Nottingham	Sherwood Forest	30	£220	Free & 31/-
Oxfordshire	Chilterns	30	-	£4. 4.
"	Kingswood	12	-	30/-
"	Maitland Cottage	24	-	10/--30/-
Somersetshire	Engel Home (Cheddar)	16	-	
"	Mendip Hills (Wells)	24	-	£4. 4.
"	Nordrach-on-Mendip (Bristol)	40	-	£4. 4.
"	St Michael's Home (Axbridge)	41	-	Free
Suffolk	East Anglian (Nayland)	35	-	£4. 4.
"	Maltings Farm (Nayland)	32	-	£1.1-£2.2
Surrey	Crooksbury Ridges (Farnham)	24	-	£4.4.
"	Frimley	108	£700	Free
"	Ockley	8	-	£4. 4.
"	Whitmead	20	-	£4. 4.
"	Woodhurst	13	-	£4. 4.
Sussex	Eversfield (St Leonards)	55	-	13/--25/-
"	Fairlight Hall (Hastings)	22	-	11/6- 15/6
"	King Edward VII (Midhurst)	100	-	£2. 2.
"	Millfield (Little-Hampton)	100	-	Free

County	Sanatorium	No. of beds	Cost per bed	Charge per week
Sussex	Rudgwick	13	-	£4. 4.
Wales (North)	Nordrach-in-Wales (Penmanmawr)	23	-	£4. 4.
"	Vale of Clwyd (Ruthin)	20	-	£4. 4.
Wales (West)	West Wales	28	£400	10/-
Westmorland	Meathop (Grange)	44	-	25/-
Wiltshire	Winsley	66	£485	£1.15-£2.10
Worcestershire	Knightwick (Worcester)	23	£130	-
	Midland Open Air (Belbroughton)	20	-	£4. 4.
Yorkshire	Bradford Poor Law (Skipton)	33	-	Free
"	Leeds (Selby) and Armley Home, Leeds	78	-	Free
"	Hull and East Riding	30	-	£1.

SCOTLAND

Aberdeenshire	Nordrach-on-Dee	50	-	£5. 5.
Forfarshire	Sidlaw	40	-	£2. 2.
Invernessshire	Grampian	-	-	£3.3 -£4.4
Kinrossshire	Ochil Hills	60	-	£4. 4.
Midlothian	Royal Victoria	100	-	- Free
Rosshire	Seaforth	50	-	-

Total number of beds for the treatment of phthisis in England and Scotland, 3,411.

It is a low estimate that 60,000 persons die every year from phthisis in the two countries, and if we take the duration of the disease as three years, it means

that 180,000 phthisical patients are living, while there is provision for only some 3,000 cases of all classes.

Immediate results of Sanatoria Treatment.

In judging the value of figures bearing on the immediate results of Sanatoria treatment, it is necessary that several facts be kept in mind. If cases sent to Sanatoria as early cases have not convincing evidence of phthisis, or have not been diagnosed by an expert, it is open to question if they were actually phthisical. It were better if they were grouped as "threatened". Further there is a regrettable want of uniformity in the nomenclature of the results of treatment, for while some Sanatoria speak of "cures", others have no more optimistic term than "very great improvement." One might suggest that some uniform terminology be adopted, and that used at the King's Sanatorium has the advantage of being clearly defined, and is as follows: -

Arrest. General health restored in every respect. Lung disease completely arrested; there being no physical signs present, or only such as are compatible with a completely healed lesion. No cough; no expectoration, or if found, free of tubercle bacilli.

Much Improved. General health completely restored. Physical signs in the lung, though much improved, not completely cleared up, e.g., perhaps limited to a few moist sounds on cough only.

Improved. General health, though improved, only imperfectly restored. Physical signs though less

marked than on admission, still present.

Stationary. No appreciable improvement in physical signs or in general health.

Worse. Deterioration of general or local condition or of both.

In the same way the want of uniformity in classifying the stages of phthisis is a hindrance to the true approximation of the results of treatment in early and advanced cases.

In many instances Turban's ³²³ classification is followed, this being: -

Stage I. Disease of slight severity affecting at most one lobe or two half lobes.

stage II. Disease of slight severity more extensive than Stage I. but affecting at most two lobes, or severe and affecting at most one lobe.

Stage III. All cases of greater extent and severity than Stage II.

By the term "slight severity" is implied disseminated foci manifested clinically by slight impairment of resonance, cough, or weak breathing, either vesicular, vesico-bronchial, or broncho-vesicular with fine and medium râles. By "severe" disease; compact consolidation and cavities recognised by great impairment of resonance, tympanitic note, very weak broncho-vesicular, bronchial or amphoric breathing with musical or toneless râles either medium or coarse. Simple pleuritic dulness if only a few centimetres in extent is to be neglected; if it be considerable, it should be specially named among

the complications, The extent of "one lobe" is always to be taken as equivalent to that of "two half lobes" and so on.

On the other hand, many writers refer to "early", "slight", "moderate", "advanced", "complicated", terms which are quite useless as accurate statements.

As a differential statement of the stages of chronic phthisis, Turban's classification is probably the best, but in the writer's opinion, it should be secondary to a clinical classification of phthisis, as Acute, Rapid, and Chronic Phthisis.

For statistical purposes in the following table, showing the results of treatment in various stages, it has been necessary to regard certain terms as synonymous. As regards results, the following terms have been considered interchangeable: -

<u>Cure</u>	Cure, absolute, provisional, relative; very great improvement, much or great, arrested.
<u>Improved.</u>	Slight improvement - stationary.
<u>Worse</u>	Unsuitable, hopeless, no improvement.

Also with regard to the stage of the disease, the following are synonymous: -

Stage I. Early or slight.

Stage II. Moderate, serious, chronic, more extensive, more advanced.

Stage III. Advanced, cavity, very bad, seriously complicated.

Such a compilation is open to serious objections, but it is the only way to obtain a general view and es-

mination of the results of Sanatorium treatment.

TABLE II.

Sanatoria or authority	No. of cases	No. of Years	Percentage of			
			Cure	Improved	Slight Improvement	Worse
Weber H. 324						
Stage I			74			
II			48			
III			19			
Turban 325						
(Davos) I	97		98.8			2
II	205		84.7			15
III	106		52.8			50
Morin 326						
(Switzerland)						
I			50	40		
II			10	67		
III			3.2	48		
Snow 327						
(Bournemouth)						
I			63			
II			40			
III			11			
Nottingham		5				
I	156		86.5	10.2	3.8	
II	191		45	31.9	8.8	9.4
III	39		10	55	15	19.

The next table shows the results of treatment at various Sanatoria of cases in all stages.

TABLE II.

Sanatoria	Years	Percentage of			
		Cure	Improved	Slight Improvement	Worse
Durham	6	79.9	13.8	1.2	5
Leeds	6	82.8		17.2	
Bowden	4	26.4	29.4	25.5	18.1

TABLE II (Continued)

Sanatoria	Years	Percentage of			Worse
		Cure	Improved	Slight Im- provement	
Devon & Cornwall	4	62.7	23.2	2.1	
Bradford Poor Law	2	61.9	15.2	21.7	
Crossley	2	35.9	26.7	32	5.2
Mount Vernon	6	34.7	32.4	25.9	4
Nottingham	5	57.1	28.2	7.5	7.2
Liverpool	5	64.6	21.7	12.9	-
Hull and East Riding	4	44.4	25.2	22.5	3.3
Heswall	4	48.7	33.7	17.1	.5
Daneswood	3	67.	22.6	6.1	3.3
Northwood	2	46.2	43.4	8.7	1.0
Winsley	2	62.9	23.6	12.6	-

It will be seen from these tables that the immediate results of treatment are extremely satisfactory, giving a percentage of "cures" in early cases in 50 to over 90. The figure will vary with the class of patients treated and the care with which suitable cases are selected.

It is far more important, however, to know in what percentage of cases a "lasting cure" is obtained. This is a difficult point on which to obtain information, as patients are not traced after leaving the Sanatoria, and as the results of enquiries into their after histories are not in conformity with the optimistic statements, which have been made as to this method of treatment.

Turban ³²⁵ wrote to 394 patients who had been treated in a period of seven years, with the following result: -

TABLE III.

	<u>Number</u>	<u>Per cent</u>
Patients treated in seven years	394	
Dead or did not reply	169	42.8
Improved since treatment	127	32.4
Stationary	60	15.2
Worse	30	7.6

This shows that while Turban got an immediate cure in 98.8% of early cases, he only got a lasting cure in all cases in 42.8%. Nor is this the entire truth of the matter, because the length of time since the patients' discharge from the Sanatoria is not stated.

If we take the average duration of the disease as three years, it is a fair test of the real value of Sanatoria "cures" to ask, what is the condition of patients three years after their discharge.

Where it has been possible to obtain statistics in answer to this question, they are a grave condemnation of the loose statements which are currently believed on this matter, and for which the profession as much as the public is to blame.

1901-1902	29	12	2	5	12	5	12.5
1902-1903	29	33	2	5	34	5	15.2
1903-1904	29	14	2	5	24	2	15.5
Totals	126	60	7	5	72	12	15.2

TABLE III.

DURHAM SANATORIUM

Condition of "early" cases three years after treatment.

Year	Cases discharged		Condition on April 30, 1907				
	Total	Returned to work	At work	At home	Dead	Lost sight of	Percentage at work in April 1907
1900-1901	16	14	6	-	4	6	37.5
1901-1902	300	23	11	1	9	9	36.7
1902-1903	41	30	10	3	20	8	24.4
1903-1904	59	46	46	4	11	15	49.2
Totals 4	146	113	56	8	44	38	39.2

Condition of "advanced" cases three years after treatment

Year	Cases discharged		Condition on April 30, 1907				
	Total	Returned to work	At work	At Home	Dead	Lost sight of	Percentage at work in April 1907
1900-1901	20	9	0	1	17	2	Nil
1901-1902	25	12	1	-	19	5	4.0
1902-1903	38	23	5	-	24	9	13.2
1903-1904	39	16	1	5	24	9	2.6
Totals 4	122	50	7	6	84	25	4.9

TABLE IV.

LIVERPOOL SANATORIUM (DELAMERE)

Table showing the condition in February 1904 of all patients who had left the sanatorium since September 1901.

Total discharged	In good health	In fair health	More or less invalids	Dead	No information obtained
238	113 or 47.5%	37 or 15.5%	36 or 15.1%	30 or 12.6%	22 or 9.2%

TABLE V.

DURHAM (STANHOPE) SANATORIUM

I. ALL CASES CONSIDERED TOGETHER

Year of discharge	Number discharged	Number at work on April 30th of each year since discharge							Percentage at work in April, 1907
		1901	1902	1903	1904	1905	1906	1907	
1900-1	36	23	18	15	14	15	12	6	16.7
1901-2	55	--	35	32	22	21	20	12	21.8
1902-3	79	-	-	53	36	32	25	15	19.0
1903-4	98	-	-	-	62	51	47	30	30.6

II. EARLY CASES SEPARATELY CONSIDERED

1900-1	16	14	12	12	10	12	10	6	37.5
1901-2	30	-	23	24	17	17	17	11	36.7
1902-3	41	-	-	30	26	23	16	10	24.4
1903-4	59	-	-	-	46	41	39	29	49.2

III. ADVANCED CASES SEPARATELY CONSIDERED.

1900-1	20	9	6	3	4	3	2	0	Nil
1901-2	25	-	12	8	5	4	3	1	4.0
1902-3	38	-	-	23	10	9	9	5	13.2
1903-4	39	-	-	-	16	10	8	1	2.6

A consideration of these figures indicates that in early cases of phthisis, we may hope to get a lasting cure in 30 to 40 per cent of all cases. Such facts will undoubtedly give rise to much disappointment when they are more generally recognised, but this were better than that the myth of 90% of declared early phthisis being curable should continue to raise false hopes. They also show the great responsibility which lies on those in whose hands is the power of detecting the disease in its early stages.

With regard to the selection of suitable cases for Sanatoria, Huggard ³²⁸ gives the following advice. "For all those people who are unable otherwise to secure suitable attention, hygienic arrangements and medical supervision, a sanatorium is obviously the best place. It is also the best place for the wilful, the thoughtless, and the impulsive. The great advantage of sanatorium life is that the patient is drilled into the practice of hygiene, and his task is made easier by having fellow-learners in the same discipline. He is made to take sufficient food, to rest and to exercise according to his condition; and he is more likely to have answered his daily prayer not to be led into temptation. In the sanatorium, too, the patient is under the immediate eye of the doctor; and the beginnings of new ailments are seen at once. Patients suffering from laryngeal tuberculosis are, also, as a rule best treated in a sanatorium, where their tendency to talk too much can be kept under control. In many cases, however, the cast-iron discipline of a sanatorium is unnecessary, in many others unavailing.

Where the needful hygienic and general conditions can be secured with the intelligent co-operation of the patient all the requirements of treatment can be carried out without recourse to the rigid system of a school. In some cases, also, it must be recognised that one ending only to the disease is inevitable. When this is the case, little is to be gained by drilling the patient. Discipline is out of place, and, except occasionally, the thermometer does not then afford information to compensate for the anxiety or the inconvenience occasioned by its use....The psychological element is important in the treatment of tuberculosis.....One great object of treatment in most cases will be to place the patient where there will be the greatest freedom from worry, fretting, irritation, depression, or excitement. This object may or may not be best secured in a sanatorium."

As the subject is often glibly accepted, it is well to remember Lindberg's dictum on this point. "Climatic treatment is not a complete therapeutics, and will only be a share if so interpreted. It is a means to an end, and is itself a powerful adjunct to hygienic and medicinal measures, and a substitute for them in cases of illness from which a patient has recovered, and a powerful remedy in cases of illness, and a powerful remedy in cases of illness, and a powerful remedy in cases of illness."

In the first instance, it is necessary to decide what cases, by the nature and degree of the illness, are suited for change of climate. These are the cases which, in the first place, of course, are the most serious.

C H A P T E R X X .

ON CLIMATES ABROAD

The value of certain climates in the treatment of phthisis has long been recognised, According to Weber, the Peruvians since unknown times were accustomed to sending consumptives to the mountain air of the Andes. In this hemisphere the advantages of certain climates in Europe in pulmonary disease were first pointed out by Brockman³²⁹ in 1843, and by Fuchs³³⁰ in 1853.

No climate in the world is a specific for Phthisis, they are only valuable adjutants in helping the patient to carry out the hygienic treatment. Change of scene facilitates change of habits, and while the pure air of some new climate will have a direct action on the patient's pulmonary lesions, the effect on his general life is of equal importance. As the subject is often glibly accepted, it is well to remember Lindsay's³³¹ dictum on this point. "Climatic treatment is not a complete therapeusis, and will only be a snare if so interpreted. It is a means to an end, not an end in itself; a powerful adjunct to hygienic and medicinal measures, not a substitute for them; a channel of escape from vicious habit and abnormal mode of life, not a mysterious remedy or an unfailing specific."

In the first instance, it is necessary to decide what cases, by the extent and nature of the lesion, are suited for change of climate. These are roughly cases in the first stage, quiescent cases, and very chronic cases.

By quiescent cases we mean those with the signs of old disease, not presently active, with no fever, and but little cough or expectoration. By very chronic cases we mean those in the third stage of phthisis, with good nutrition, no cachexia, and no complications.

As a guide to deciding on a change of climate, the following points should be carefully considered: Age, sex, single or married, occupation, means, extent of lesion, general health and habits, the condition of the larynx, bowels and kidneys.

A consideration of these ^{points} will guide ^{us} as to whether the patient be sent to a Sanatoria, to a climate abroad, on an ocean voyage, or be advised to emigrate.

A. Suitable for Mountain Climates.

1. Early Phthisis.
2. Slight disease at one or both apices with little fever.
3. Cases of slight disease with high fever, after this has been controlled at home.
4. Quiescent chronic disease of one lung.

B. Suitable for an Ocean Climate.

1. Early Phthisis.
2. Chronic cavity in one lung.

C. Suitable for Marine Climates.

1. Extensive disease with slight fever.
2. Extensive disease with slight catarrh.
3. Quiescent extensive disease.

D. Suitable for Inland Climates - plain or desert

1. Cases with albuminuria and diabetes.
2. Emphysema.

E. Unsuitable for Abroad.

1. Rapid Phthisis.
2. Advanced disease with fever.

The Climate of Mountains.

The climate of the mountain is characterised by low atmospheric pressure, dryness of the air, warmth, light and sunshine.

Such climates are stimulating and tonic, inducing an increased metabolism, manifest by greater respiratory movements, which strengthens the thoracic muscles and produces an increased oxygenation of the blood. This means an improvement of function of the skin, lungs, and kidneys, so that the patient eats better, feels better, and sleeps better than he did in denser air. According to Bruce³³² a residence at high altitudes gives an actual increase of chest measurement.

Hygienic measures in a mountain climate yield favourable results in the treatment of phthisis. Thus Weber³³³ obtained cures in 25% of cases, while Turban³³⁴ at Davos got 66.1% of absolute and relative recoveries.

The principal mountain climates in the world are: The Andes, which are three thousand metres above the sea, with a variation of temperature of but 4° Centigrade. The expense, the long journey, and the isolation are the drawbacks.

The Rocky Mountains and the Adirondacks are drier, than the Alps, but less equable than the Andes.

The Swiss Alps, where there are numerous stations at Arosa, Les Avants, Andermatt, St Moritz, Montana, Miesen and Leysin.

South Africa. The heat, dryness, dust, arid summer, and great changes of temperature are against the mountain climates of South Africa.

The Climate of Forests.

The soil of forests is moist, there is little movement of the air, and the temperature is equable. The climate of a forest, however, differs according to whether it be on the level or on a hillside.

Forests on the level are dark, being hidden from the light of the sun. The air is cold and damp, from the exhalations of rotting undergrowth. When the forest lies on a hillside, these disadvantages are obviated, and the climate rendered healthy. The hillside is well drained, and ventilation is perfect, as during the day the air moves up the hillside, and at night there is a downward current. Such a forest is sheltered from cold winds, and is better lit than the forest on the level, for the trees standing at different heights, light gets in between them. The air of a pine forest is charged with balsams and volatile oils.

Climate of Inland Plain or Desert.

The air of such climates is dry, warm, and sunny. There is little rain, and a "blue rejoicing sky." A fall of temperature occurs at night, but with such regularity that it may be provided for. The disadvantages of these climates are wind and dust.

Egypt is the chief of such climates, but against it is the short season, December to March, although at Alexandria it is prolonged to May, the dust storms, and the

great expense. Lindsay's ³³⁵ experience of Egypt is as follows: -

"Egypt has great attractions for the patient of ample means who can command every luxury, and whose condition does not preclude him from travelling about and enjoying the scenery and the antiquities. Such cases are, however, exceptional, and Egypt is not well adapted for ordinary cases of phthisis. In spite of the magnificent climate and in many places excellent accommodation, the conditions of life and the social milieu are not suitable for consumptives. The Nile voyage, however charming, has a very limited utility for phthisical cases. Dust is apt to be troublesome in Egypt. The season is short, as patients cannot stay with advantage when March is past. The choice of locality in Egypt lies between the following places: Cairo, Helouan, Mena House, Luxor, and Assouan. Cairo should be avoided by the consumptive. The hygiene is bad, and the social atmosphere unsuited to such cases, I have made only a very limited trial of Egypt in phthisical cases, and I give the foregoing opinions with some reserve. Dr Sandwith records that "after eighteen years of life in Egypt he has only as yet seen one case of a European living in the country becoming tubercular." ³³⁶ He admits, however, that the natives suffer largely from the disease. I should be inclined to limit the recommendation of Egypt to early and quiescent cases, especially in middle-aged patients where bronchitis or weak circulation or rheumatism is present, and in cases where the historic and antiquarian attractions of the country were likely to prove of special interest.

"The same remarks might apply, mutatis mutandis, to the interior of Algeria. Hammam R'Ihra is a good spring resort, but is not very suitable for winter. Biskra, on the edge of the Sahara, is recommended for cases where desert air and quiet are desired."

Other inland climates are Pau, Montreux, Meran, and California.

Marine Climates.

The climate of certain coasts has the advantage of warm, sunny, humid air. Such climates are apt to be damp, but the variation in temperature is less than is found inland.

The Riviera is perhaps the best known marine climate, and according to Theodore Williams ³³⁷ is beneficial in the following cases: -

- (a) Phthisis, in which inflammatory processes have played a large part in predisposing to the disease.
- (b) Strumous phthisis.
- (c) Laryngeal phthisis.
- (d) Unilateral tuberculisation rather than bilateral.
- (e) The large class of consumptives who, either from extent of disease, or feebleness of circulation, or advancing years, are unable to endure the rarified atmosphere and cold of the high altitudes. Most of these patients love warmth and cannot take enough exercise, when the thermometer is below zero, to maintain it.

The presence of Monte Carlo, and the change of the Riviera from a health resort into a social playground,

are two factors which greatly militate against its value for phthisical patients.

Other Marine Climates are: -

The Home Resorts: Ventnor, Bournemouth, Torquay, St Leonards, Dawlish, Sidmouth, Salcombe, Ilfracombe, Rostrevor, Queenstown, Glengariff.

The Canary Islands: Orotava, Santa Cruz, (Teneriffe and La Palma) Las Palmas, Güimar, La Laguna.

Madeira: The climate being warm and moist is apt to be relaxing rather than stimulating.

Sicilian resorts: Catania and Palermo.

Malaga and Algéiras: Of these Algéiras is preferable, the season being from February to June. The winter in the South of Spain is characterised by great variations in temperature.

Algiers and Tangiers are too oriental to be suitable for northern invalids.

The Climate of the Ocean.

This is marked by pure air, great movement of air, high percentage of ozone, sunshine and slight changes of temperature. There is freedom from injurious influences and mental rest.

Such were the advantages of ocean travel on the old "three decker", steering an easy course to sunnier climes, but they have mostly gone with the advent of the "ocean greyhound." The stuffy cabin of the modern steamer has replaced the roomy apartments of the sailing ship. The change from a temperate to a warm climate is comparative-

ly quick. Further, the life on a modern steamer is in no way suitable for a phthisical patient.

The tramp steamer is little better. The food and accommodation is indifferent, and there are too many days in port.

A voyage to Australia in a sailing ship is still practicable from some ports in the kingdom.

The ideal method of ocean travel is by private yacht under medical supervision.

Climates for the Poor.

Mountain Climates. Colorado.

Marine Climates. Tasmania, Florida and California.

Inland Climates, Orange River, Transvaal, Karoo? Natal, California, New South Wales, Queensland, and New Zealand.

CHAPTER XXI.

PHTHISIS AND MARRIAGE

In discussing the relation of phthisis to marriage, the question arises: "Is phthisis an inheritable disease?" Were we to use heredity in its strict sense, no bacterial disease can be inheritable, since a bacteria cannot be part of the germ-plasm. As Kantback ³³⁸ puts it "No specific infective disease is hereditary, if we use the term 'heredity' in the sense which Darwin and the biologists have given to it. If it appear congenitally it is simply communicated to the foetus by infection." It were better to put the question thus: "Is it possible for the child to be actually infected with the tubercle bacillus before birth, or is it only born with a predisposition to the disease?" Or as the French have more neatly expressed it, is it "hérédité de graine ou hérédité de terrain?"

Is placental transmission possible? On this question, we find two opinions diametrically opposed, some holding that predisposition, but not the actual bacilli, is all that the child can inherit, while others claim that as in syphilis, the father can transmit tubercle to his child, without infecting the mother, and that in the child the disease may remain latent for years.

Such are the rival theories. In considering the facts, one is sceptical of accepting deaths in very early infancy as absolute evidence of placental infection, for in a weak child, disease of extra-uterine origin is able to make extremely rapid progress.

The only definite proof of placental infection is the presence of tuberculosis in a dead foetus. The cases of this, reported prior to 1882, may be passed as it was not possible to demonstrate Koch's bacillus in the lesions. The following observations, however, are more definite. Sabaroud³³⁹ found tubercles in the liver and spleen of a child who died on the eleventh day, the mother being tuberculous, and Armani³⁴⁰ produced tuberculosis in rats by injecting them with the liver and spleen of a foetus from a tuberculous mother. Bar and Renon³⁴¹ found tubercle bacilli in the blood of the umbilical cord.

In the lower animals, especially in the bovines, placental transmission has been proved to occur. Cheveau³⁴², Bang³⁴³, Johne³⁴⁴, Malvez³⁴⁵, Bronwier³⁴⁶, and others have shown the existence of tuberculosis in the foetuses and new born calves of tuberculous cows. Renzi³⁴⁷ in the examination of 18 pregnant rats, found five instances of tuberculosis in the foetuses. Gartner³⁴⁸ has demonstrated tubercle bacilli in the eggs of tuberculous parrots, but not in those of the fowl.

It would appear from this that placental transmission is undoubtedly possible, but so far has not been

shown to be frequent in the human species. On the other hand, it is an incontestable fact that not one in a thousand cases of still-birth is investigated on this point.

Can the Sperm infect the Ovum? It has been shown by Landouzy³⁴⁹, Martin³⁵⁰, Wiegert³⁵¹, Jani³⁵², Bozzolo³⁵³ and Niepie³⁵⁴ that the semen of a tuberculous patient, in whom there is no apparent lesion of the genital organs, may, nevertheless, contain tubercle bacilli. It is a different question, however, if the tubercle bacillus can actually infect the ovum. Baumgarten³⁵⁵ artificially fertilised a doe rabbit with spermatozoa from a tuberculous buck rabbit, and later found a tubercle bacillus in an ovum. This observation while of extreme interest, hardly admits of any definite deduction, for while the bacillus might undoubtedly have lain there until the ovum became a foetus, it is also possible that the ovum might have destroyed the bacillus, or the bacillus the ovum.

In support of the theory of direct parental infection, Baumgarten adduces the fact that the tissues of the foetus and the new born offer considerable resistance to bacillary infection, and suggests that the bacilli may lie latent in the glands and bone marrow until some local injury or lowering of general resistance permits them to attack the tissues.

This view is borne out by the work of Landouzy³⁴⁹, Martin³⁵⁰, Birch-Hirschfeld³⁵⁶, and Schmorl³⁵⁷, who found that apparently healthy organs in fetuses of tuber-

culous mothers, contained tubercle bacilli, and by the
 experiment of Maffaci ³⁵⁸, who inoculated hen's eggs with
 various bacteria, and noted that the infection did not
 break out until some time after the eggs were hatched.

³⁵⁹ Roger ³⁵⁹ has pointed out that hereditary tuber-
 culosis differs from acquired tuberculosis, not in the
 pathology, but in the distribution of the lesions,
 as in the foetus and new born it is the liver that is
 invariably affected, which points to infection by
 the umbilical vein. He holds that tuberculosis
 of the liver in the infants of tuberculous parents in-
 dicates placental transmission provided there be no
 tuberculous lesions in the intestine to account for
 the condition.

It is certainly clear that tuberculosis in early
 infancy does occur, and mostly in the infants of tubercu-
 lous parents. Thus Leroux ³⁶⁰ has reported 23 cases of
 tuberculosis in infants under three months. As the proved
 cases of direct hereditary infection are extremely
 few, it seems more likely, however, that the off-spring
 of tuberculous parents escape with a predisposition to
 that disease, manifest in definite physical stigmata,
 thus rendering them an easy prey to the repeated ex-
 posures to infection which they are unable to avoid.

Although denied by Calmette, this is borne out by the
 researches of Schrieber ³⁶¹, Hatinel ³⁶², and Landouzy ³⁴⁹,
 who failed to obtain the tuberculin reaction in the
 children of tuberculous parents, yet it is possible
 that the bacilli may lie latent in the tissues for years,
 and unless the disease were active, no reaction would be
 obtained.

INHERITANCE OF A PREDISPOSITION

Virchow ³⁶³ denied the existence of congenital tuberculosis, on the strength of his own pathological investigations, and held that the infant is infected in the first few days of life.

This too, is the opinion of Hamilton ³⁶⁴, who writes "with extremely few exceptions - so few that they may almost be neglected - children are not born tubercular even of tubercular mothers, nor are the young animals born tubercular under like conditions."

This view is summed up in Debierre's aphorism: "On ne naît pas tuberculeux, on naît tuberculisable".

Whether congenital infection be denied or not, the inheritance of a predisposition to tubercle is generally admitted. It is a question of extreme interest to consider where lies the actual weakness in such a predisposed constitution. On this Hamilton writes "Most likely the particular vulnerability resides in the epithelial protective coverings of the body being too little resistant, too easily stimulated by external agencies, too readily penetrated by the parasite of the disease..... In support of this assertion are to be taken into account certain epithelial manifestations which accompany the tubercular habit - namely, the very dark or very light degree of colour of the hair, the overgrowth of hair in the bushy eyebrows and long eyelashes, and lastly, the occurrence of a tanago-like overgrowth in tubercular children along the spine and over the legs.

To my mind these all point to an anomaly of the epithelial type which is peculiar to the tubercular habit of body".

Whether the primary infection come from the lung or intestine, does not touch the validity of this view, as in either case we are dealing with epithelial structures.

A more disputed point, however, arises when we ask "What produces the Predisposition"? Some hold that the presence of the bacilli in the parents finds expression in a particular type of build in the offspring. A more probable explanation is that of Hamilton, whose dictum is as follows: "Where has the inherited strain come from? What is its ancestral history? Can it be generated by vicious surrounding? I question whether it can. No doubt, once in the blood, the particular habit may be fostered by every external agent which tends to deteriorate the natural powers of resistance. But will such external agencies tend to produce a particular colour of hair, a certain narrowness of chest, tallness of stature, and other peculiarities which are distinctive of the tubercular constitution? My conviction is that they will not, and that we must go much further back in the history of the human race to get at the explanation of the matter. My own impression is that these features are the lineal descendants of a variation which took place far back in our history, that the variation has occurred irrespective of surroundings or external agencies, and that its influence has been propagated in the descendants ever since. It may be a variation

which is common to many races, but one which apparently is intensely hereditary."

Assuming the origin of the predisposition, it is not unlikely that disease in the parents, favours a throw back in the offspring along an old inherited strain.

DANGERS OF MARRIAGE

We have seen in the foregoing that the children of phthisical parents are liable to be born with a predisposition to the disease, and that actual infection is possible although less common.

Such are the dangers to prospective offspring by the marriage of those tainted with tubercle, yet more immediate are the dangers to the parents themselves.

It is needless to say that no person affected with active phthisis should be permitted to marry, not only on the ground that it will aggravate the disease, but on the score of the most elementary conception of ethics.

Hutchinson's rule as to syphilis might well be applied to phthisis, that no person should marry until he or she has lived two years after all signs of pulmonary disease have disappeared.

When the disease is arrested, the question of marriage is more difficult to decide, and Kirchner ³⁶⁵ points out that the dangers are greater among the poor than among the rich.

The points to be borne in mind are summed up by ³⁶⁶ Osler as follows:

"The question of marriage of a person who has arrested or cured lung tuberculosis is more difficult to decide. In a male, the personal risk is not so great; and when the health and strength are good, the external environment favourable, and the family history not extremely bad the experiment - for it is such - is often successful, and many healthy and happy families are begotten under these circumstances. In women the question is complicated with that of child-bearing, which increases the risk enormously. With a localised lesion, absence of hereditary taint, good physique and favourable environment, marriage might be permitted. When tuberculosis has existed, however, in a girl whose family history is bad, whose chest expansion is slight, and whose physique is below the standard, the physician should, if possible, place his veto upon the marriage.

In women the position is further complicated by the chances of child-bearing, and as Dubois has remarked: "If a woman threatened with phthisis marries, she may bear the first accouchement well; a second, with difficulty, a third, never."

In view of this, when phthisis declares itself in a married woman, the question arises as to the advisability of inducing abortion in the event of her becoming pregnant.

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Armstel has recently made an exhaustive consideration of this question, and quotes the figures given by Maragliano as to the effect of pregnancy

upon an existing phthisis in 42 cases are very striking. The patients suffered in every instance from circumscribed tuberculosis, and the general condition at the beginning of pregnancy was good. Nine of the mothers died from phthisis during the puerperium, 7 died within three months from the time of labour, 9 died between the third and sixth months after labour, 10 between the sixth and ninth, 4 died after about a year in only 3 out of the 42 cases did the condition of the lungs remain stationary. The death-rate was thus 94 per cent; while in 188 cases of circumscribed phthisis in women not pregnant, the death-rate in the same period of twenty-one months was 18 per cent only. The chances of life for a child of a tuberculous mother are in any case bad. Friedmann's statistics with respect to 2984 patients suffering from phthisis showed that in 33 per cent of the cases there was a definite history of hereditary predisposition, while in only 25 per cent, could such a history be definitely negatived. *order to increase her chances of recovery,* but Armstel quotes 17 cases from his own practice in order to show the results to mother and child of allowing the pregnancy of a phthisical mother to go to term. In these cases the women all came from phthisical families; 10 of them were sound at the beginning of pregnancy; the others suffered in each case from a chronic form of phthisis and the prognosis apart from pregnancy would have been good. In every instance the phthisis made rapid progress during pregnancy, one woman died during labour, the others at intervals after labour which

varied from three days to eighteen months. Of the children, one died at birth, only four reached the age of 1 year, and only three that of two years. Thus seventeen mothers died, while three weakly children survived.

It seems probable that a large proportion of these women would have survived if abortion had been induced early in pregnancy; but it is also true that in many similar cases labour has gone on to term with results favourable both to mother and child, and therefore he dissents from the opinion of Maragliano that in every case in which pregnancy occurs in a woman suffering from phthisis abortion should be induced, and would judge each case on its merits. Another great distinction between the author and Maragliano is that the latter would induce abortion in these cases for the purpose of fighting tuberculosis, while the former would do it only in the interests of the mother.

Where the disease of the mother is curable Van Armstel would, as a rule, induce abortion as soon as possible in order to increase her chances of recovery, but when there is no hope of the mother's recovery he would let the pregnancy go to term in the interests of the child.

Armstel concludes as follows: (1) Pregnancy, labour, and the puerperium have a harmful effect upon an already existing phthisis. (2) Where a hereditary predisposition to tuberculosis exists pregnancy often causes the development of phthisis or of some other form of tuberculosis. (3) Germinative infection

does not occur, and placental infection rarely occurs, but the children of a tuberculous mother are weaker, and have less resistant power and less prospect of life both before and after birth than normal, and are pre-disposed to tuberculous disease, especially to phthisis.

(4) It is the duty of the doctor to warn any woman who suffers from acute or latent phthisis, and who proposes to marry, of the dangers which marriage will bring. (5) When the marriage has occurred the doctor should point out to the wife and husband the necessity and the usefulness of facultative sterility. (6) After conception has occurred the doctor should make clear the danger to the wife of pregnancy, and should advise that the pregnancy should be interrupted in her interests. (7) The indication for the induction of abortion is the more pressing the less far the phthisis has advanced, because of the increased chance of preserving a relatively hopeful case.

and the actual occurrence of infection in childhood may be gathered from the fact that the mortality from tuberculosis is highest during the first ten years of life, as has been shown in the con- sideration of age as a predisposing cause of tuberculosis.

It is probable that the strain thrown on the respiratory system during the years of rapid growth may account for the greater susceptibility of the child to tuberculosis than to pulmonary tuberculosis, and that the greater susceptibility of the child to tuberculosis is due to the fact that the infection of the respiratory system is more likely to be carried to the lungs in the case of the child than in the case of the adult.

C H A P T E R X X I I .

THE PROPHALAXIS OF PHTHISIS IN THE CHILD

The prophalaxis of phthisis in the child, particularly in those children predisposed to the disease, has received all too little attention in these days of educational "systems" and of variegated crusades against tuberculosis. ³⁶⁸ Eustace Smith remarks "Phthisis is a common disease in the child", and quite apart from the numbers who die in childhood from various forms of tuberculosis, it is certain that a vast number (50% ³⁶⁹ according to Schlossman) pass into adult life with the germ latent in their bodies, and are thereby destroyed at some later period, as during the critical years of adolescence, when the death rate from phthisis is high. That is to say the infection of the child means the disease in the adult, and the actual amount of infection in childhood may be gathered from the fact that the mortality from tuberculosis is highest during the first ten years of life, as has been shown in the consideration of age as a predisposing cause of tuberculosis

It is probable that the strain thrown on the alimentary system during the years of rapid growth may account for the greater susceptibility of the child to *Tabes mesenterica* than to pulmonary tuberculosis, and clinically it is certain that tubercular infection of the mesenteric glands not only may be chronic but also may be found post-mortem where no symptoms of such infection were

present in life. Further, we know that tubercle bacilli may be latent for years in the living body, and that in animals they proceed from the mesentery to the lung. From these facts we conclude that many children pass from childhood with the bacillus lying latent, ready to attack and destroy in later life when resistance is lowered, either by environment or on some hereditary defect becoming manifest. Quite apart, however, from the question of actual infection in childhood, it is obvious that prophylactic measures in the child will be followed by a diminution of phthisis, firstly, because predisposition will be remedied, and secondly, the individual rendered stronger when he enters the critical periods of life.

In view of this it is not surprising that the death rate from phthisis should remain unaffected by any preventitive measures, which fail to reach the primary sources of the evil - the predisposition and the infection of the child. Here, as in the question of treatment, we distinguish between the children of the rich and the children of the poor, and prophylactic treatment for the latter will be discussed in the relation of the State to phthisis.

A child born with a predisposition to tubercle, either on account of this disease in its forbears, or it may be from some of those more subtle disharmonies of heredity, is started in life with definite mental and physical stigmata.

Such children are often undersized, the head appearing large in proportion to the body, and are of

poor development. According to Eustace Smith the lungs are small, causing an alteration in the shape of the chest, apparent at the fourth or fifth year, and due to the adaptation of the thorax to its contents. Two varieties are seen, the shoulders may be narrow and sloping, the chest elongated, and the ribs abnormally oblique. Again, the scapulae may project back like wings, the alar or pterygoid chest - so that the more lateral position of the external borders of the scapulae gives an appearance of flatness and over broadness to the thorax, whose antero-posterior diameter is diminished, and is greater below than above. The facies of a predisposition to phthisis has already been dealt with, and appears the more marked in the child - silky glossy hair growing well forward on the temples, large eyes and pupils overhung with long eye-lashes, full lips, with well formed nose and malar ridges, and pale complexion. Yet more marked than the features is their expression, for as surely as syphilis is manifest by old features on young shoulders, so the taint of tubercle is marked by the expression of thought on a child's face - the face of the predestined, that is warned.

The mental attitude of these children is often striking, particularly when there co-exists the neuropathic temperament, for we find a hyper-sensitivity of the brain, not present in the normal child. A frequent commonplace is "How imaginative children are" and applied to the normal child this dog-eared remark is by no means true. Children are not imaginative, for

their life and play is but an imitation of things they have seen, they are actors not dramatists. A child will play for hours with a few chairs in the make-believe they form a train. Take away the chairs and the train disappears into nothing. The stage is cleared, and to resume the play the actor must find new properties. His theatre of life is small, ideas are but dawning, and to call forth his few precepts he must seek the assistance of external things. A child that will fill a train with passengers, speed them across continents, finally wrecking them in a calamitous collision, would stare in wonder were he asked to think about a train.

Nor is this strange when we remember the long road the child must travel - from a bundle of reflexes, to a "reasoning creature in being" for at birth the child is the most helpless animal in the Cosmos. The fossorial wasp leaves its shell to enter at once on the life of a mother it never knew. The foal will stagger to its feet a moment after birth, ready, as in ancient days, to follow the herd. But the child, in whose development is read the history of aeons - the amoeba to man - has only the power to feel and to move. Every perceived stimuli will call forth movements, some obviously purposive as the movements of the mouth in sucking, from the inborn memories of the long mammalian line. When these movements are influenced by contact with external objects and their sensory impressions registered on the brain, there follows a conception of the external world. The child has now the power to distinguish and

select, and is capable of conscious action, impossible without the memory of previous experience - as witness the decerebrated frog who lives for years, eating food placed in the mouth, springing when touched, but incapable of volition - for consciousness is the totality of previous experience. Even simple movements are of slow evolution - at the end of the second month the head can be raised, at the seventh the child can sit up, at one year it can stand ^{370.} Yet there is something more, for the act of selection is an act of will, and with this there dawns on the child the World of Idea, for in the absence of the actual object or movement, these are recalled by stimulation of the supreme centres by some allied object or movement, resulting in a mental concept or idea. ³⁷¹ Of this time Maudsley writes: "Transient hallucinations are probably common; when it stretches out its hand and makes futile grasps, it is not always grasping at a real object out of its reach it is sometimes mocked by a hallucination. The real world and the unreal world "not being yet distinguished, its life is as much in the one almost as the other, its natural talk when it is amusing itself is an incoherent prattle in which it does not distinguish its personality from things around it but speaks of itself in the third person as one of them." Last, is the power of inducing by an act of Will the mental equivalent of abstract Thought, and this we term imagination, the absolute creation of Thought, the latest and highest attribute of Mind.

From this it is clear that knowledge and imagination are by no means natural to the child, and the concentration, projection, and correlation of ideas are only laboriously to be attained.

The child predisposed to tubercle presents a very different mental attitude, for here there is mental activity far in advance of its age and experience. The mental characteristics of such children are most marked when they also present the neuropathic temperament, and have never been better described than by Maudsley³⁷² in his masterpiece "The Pathology of Mind". Precocious children of highly nervous temperament, especially those predisposed to meningeal tubercle create imaginary scenes and dramas which they see and deal with as actual events; instead of going to sleep perhaps when they go to bed, they lie awake prattling of visionary scenes as if real and they were taking an active part in them; so much so that their mothers are sometimes alarmed by their delirious chatter and think them light-headed. They are dreaming so to speak while wide-awake, and with them as with the dreamer, the notion is translated instantly into vivid sensory form; all the more easily, first, because there is no store of registered ideas to hold it in by their associations, and, secondly, because of its own vividness and intensity".

"Although this sort of delirious dramatisation is most apt to take place at night when outer objects are shut out by the darkness and quiet reigns around,

yet in less degree it may take place in the daytime; then the child cannot be trusted always to distinguish between facts and fancies, may be accused of inventing the story which it tells and perhaps punished as a liar. The truth is that it does not distinguish between the very vivid images of real things which its intense imaginations are and the perhaps less vivid images which realities appear to it. How can it if the unreal is more real to it than the real?"

These children have strange desires and fancies, unknown to the normal child. Often showing an absolute dread of child companions they will speedily make friends with their elders, and a child that shrieks with terror at the sight of another child in its house, will seize hold of stray dogs in the street without fear and apparently with impunity. Again a child that is timid to most events in life will wander away from its friends in an ecstasy of delight when the wind is blowing a gale.

At night they are subject to fearsome dreams and visions, not wholly the product of intestinal irritation. Thus Maudsley ³⁷³ tells of a scrofulous child who when awake at night, would shriek in a paroxysm of terror because it imagined there was something in the bed with it, and feared the moonlight because "it made so much noise". Such nightmares as these are not the nightmares of childhood - of a physical paralysis in face of impending danger in the form of trains, bulls or robbers - they are the nightmares of the indescribable fear of an unknown calamity, whereat the child will

wake, still frightened, trembling and wet with perspiration. Of these terrors Charles Lamb ³⁷⁴ has written in the telling words of sad sincerity: "Dear little T.H. who of all children has been brought up with the most scrupulous exclusion of every taint of superstition - who was never allowed to hear of goblin or apparition, or scarcely to be told of bad men, or to read or to hear of any distressing story - finds all this world of fear, from which he has been so rigidly excluded ab extra, in his own "thick-coming fancies" and from his little midnight pillow, this nurse child of optimism will start at shapes, unborrowed of tradition, in sweats to which the reveries of the cell-damned murderer are tranquility."

So extreme and grave a clinical picture is by no means present in all children with a predisposition to tubercle. Many such children have no night terrors, take a delight in plays and toys, and yet in these the danger signals are to be read in their build, expression, and precocity - as witness one child of six who learnt the alphabet without effort in two days. Further, they seem to be able to understand and to accept situations which are puzzling to their elders. For the portraiture of such a child the writer knows of no better than that in "Dombey and Son".

The quickness and precocity of these children are in most cases their undoing. So far from being recognised by their parents as the danger signals indicating a life in absolute harmony with nature whereby that lowered vitali-

ty which was their birthright may be repaired, their aptitude for knowledge, far beyond their years is encouraged, their hours are spent in crowded rooms, and a body once predisposed to tubercle is turned into a veritable hot-house for that disease.

With regard to prophylaxis we first consider the means of avoiding the infection of the child, and passing the question of intra-uterine transmission of bacilli as possible but extremely improbable, the sources of danger from the start of extra-uterine life are through the respiratory or alimentary systems. As to the former, it is interesting to note that those who uphold the aerogenous infection of the adult should admit its extreme rarity in the child, as proved by the more frequent lesions in the mesentery. Now as the child inspires the same air as the adult, consequently running the same risk of infection, one were justified to ask if the lungs in childhood have been proved to present a greater resistance to pathogenic organisms than in later life. Were such the case one would unhesitatingly accept this seeming incongruity in the air borne tubercle bacillus, but the fact remains that the child is more susceptible to other pulmonary diseases than is the adult. However, as all are agreed that respiratory infection plays but little part in the child, and as the measures against alimentary infection cover this as well, nothing further need be said. The danger through the alimentary system is by the food, either contaminated at its source, or before it reaches

The child should be brought up entirely in the
the child. the actual place depending upon circumstances.

Should phthisis be suspected or apparent in a mother, she must on no account be permitted to nurse the child, for the strain on her system will augment the disease, and the child is subjected to grave risk of infection either at the breast or by the preparation of its food by the mother, or by the contamination of playthings which it is apt to place to its mouth. A healthy wet-nurse should be selected to have entire charge, for there is little doubt that from every point of view the best start in life for any child is to be fed at the breast as proved by the classical instance of the great Siege of Paris when although the general mortality was appalling the infant mortality became extremely low, the mothers being forced to suckle their children. If breast feeding is impossible, and it be decided to rear the child on cow's milk, the purity of this must be assured, which also applies to the feeding of the child after weaning. The ideal condition, possible to a few in the country, is where a special cow, tested every few months by tuberculin, is reserved for the child, and the milk suitably diluted according to age. (Formula X V //) Otherwise Pasteurised milk should be used (Formula X V ///). or some of the brands of humanised milk on the market. Scrupulous care is to be taken with the cleaning of the feeding bottle, of modern make with no rubber tubing, and no phthisical person should have any hand in the preparation of the child's food, and for the same reason the child is kept apart from phthisical patients.

The child should be brought up entirely in the country, the actual place depending upon circumstances, but in choosing a district the following factors are to be borne in mind. A dry soil is essential, after which comes sunshine and bracing air. These conditions are most likely to be found along the south-east coast of England, or in the high mountain districts of Scotland and England (not in narrow or low-lying valleys). The house must be well situated, facing south-east, and the ventilation, drains, and water in perfect condition. The child should occupy a large, airy, room, looking south-east, with the windows open at the top, except in very severe weather, and at night the nurse should occupy an adjacent room.

It is generally believed that mountain districts are of greater value in the prophylaxis of phthisis, than climates by the sea. Weber ³⁷⁵ found that of 40 children coming from tuberculous families, but reared in mountain districts, only 4 developed tubercle, under the strain of the unfavourable conditions of later life. Mercier found that of the children of tuberculous parents only 3% of those sent to the country died, while 50% of their brothers and sisters who remained in towns died.

Clothing is to be warm and loose, stays and braces being absent, pure wool underclothing summer and winter and flannel nightgowns. The food must be simple and wholesome - porridge and milk, fresh eggs and butter, home-made preserves, ripe fruit, small quantities of minced meat or white flesh, fresh fish, potatoes, milk puddings

of rice, semolina, tapioca, brown bread, treacle, and milk chocolate as a sweetmeat. Fluids should be fresh milk, buttermilk, "tea and water", "tea and milk", cocoa and freshly-made lemonade. Meals must be regular breakfast at 8.30 a.m. light lunch at 11 a.m. dinner at 2 p.m., tea at 6 p.m. and no food given between meals. The maximum amount of sleep is necessary, and the best soporific for the child is to have spent the day in the open air. The kissing of the child by strangers should be prevented, as a dangerous proceeding.

The teeth and bowels demand special attention, as decaying in or about the teeth is a frequent cause of digestive troubles. The teeth should be cleaned night and morning, and periodically be examined by a dentist. The feverishness which accompanies even a slight degree of constipation in a child is a clinical indication of how easily the mechanism of assimilation is thrown out of gear. Castor oil is the best cathartic for children, and as an emulsion with *Marsch Mallows* is tasteless. (Formula XIX).

Certain diseases undoubtedly predispose a child to phthisis - measles, whooping cough, and catarrhal pneumonia - which weakens the system, and by chronic catarrh leaves a damaged area of pulmonary tissue, liable to infection by the tubercle bacillus, either inspired or carried thither from other parts of the body by the lymphatics. Enlarged tonsils in a state of chronic inflammation are said to offer a good absorbing surface to the tubercle bacillus, and to be associated with glands in the neck. When present they should be removed at

once as the mental and physical results of the operation are remarkable.

Catarrhal diseases of the respiratory system are to be avoided by hardening the child with a life in the open air, encouraging all amusements in which this is essential, and by chest exercises in moderation. The liability to chill is obviated by educating the skin to react to changes of temperature, and to this end the cold douche is useful. The child should be sponged with warm water every morning, in front of a fire in winter, in sunlight in summer - followed by a cold douche 65° F to 70° F., after which it is rapidly dried with a bath towel thrown around it. If the skin appears blue and cold, and the "red glow" absent, the water has been too cold.

Too much attention cannot be given to the selection of a suitable nurse, for the child's education begins at the breast, and the nurse chosen should be a capable woman, of kindly disposition and strong character. Precocity in the child must be checked, and a regular life, and healthy ideas will do much to prevent night terrors.

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Beyrand has shown that these usually occur during the third hour of sleep and considers they are excited by gastro-intestinal irritation, due to improper food or to worms, or from lead poisoning by the paint of toys or pottery, while Soltman found they existed in anaemic children of neuropathic stocks. Should night terrors exist their exciting causes are to be investigated and remedied, but night terrors are quite distinct from

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ordinary night-mares, having been shown by Debacker to be vivid hallucinations of sight and feeling. It is probable that the slight cause of nightmare in the healthy child produce in neuropathic temperaments a morbid physical disturbance.

During the winter months the child may be given tonics, and of these the simplest are the best, as in children there is no question of stimulating worn out tissues but of assisting the natural processes of assimilation and for this purpose malt extract or emulsion of cod liver oil combined with phosphates of malt are sufficient. A drug which ensures the regular functioning of the excretory systems is sulphur. In the Highlands of Scotland it was a routine practice to give children once a week a tablespoonful of a confection of sulphur, soda, and treacle a mixture by no means unpleasant to take, and which undoubtedly acted as a prophylactic against many ailments in the child, being further credited with ensuring clear complexions. The actions of Sulphur are dealt with elsewhere (Chapter XVII) and the writer holds it an excellent practice to give a tablespoonful of this mixture (Formula XX) to young children once a week.

The child should not be sent to School before the age of eight, when hours of study and exercise are to be carefully regulated. Later on, for boys, a public School course will ensure a powerful physique, and the question of a profession, avoiding those for which long hours of indoor study are necessary, demands careful thought. The learned professions, especially Medicine, should be

excluded, and probably the Navy as a calling, offers the healthiest life.

MEASURES AGAINST PHTHISIS.

It is impossible to do more than touch on some of the more important measures which have been taken against the spread, and for the effective treatment of phthisis. We may consider this from the points of view of local authorities and of private enterprise.

Compulsory Notification

By the terms of the Public Health Act 1870, and subsequent Acts relating to infectious diseases, and by the Public Health (Scotland) Act 1897, it is possible for a local authority to include pulmonary tuberculosis as an infectious disease, of which the notification is compulsory.

This means that the patient may be subject to the penalties of the Public Health Act, which are as follows:-

1. The patient may under certain circumstances be compulsorily removed by order of any justice to an isolation hospital.

2. By the provisions of Section 124.

Any person who

(1) While suffering from any dangerous infectious disorder wilfully exposes himself without proper precautions against spreading the said disorder in any street, public place, shop, inn, or public conveyance, or enters any public conveyance without previously notifying to the owner, proprietor, or driver that he is so suffering,

CHAPTER XXIII.

THE WAR AGAINST PHTHISIS.

It is impossible to do more than touch on some of the more important measures which have been taken against the spread, and for the proper treatment of phthisis. We may consider this from the points of view of local authorities and of private enterprise.

Compulsory Notification

By the terms of the Public Health Act 1875, and subsequent Acts relating to infectious diseases, and by the Public Health (Scotland) Act 1897, it is possible for a local authority to include pulmonary tuberculosis as an infectious disease, of which the notification is compulsory.

This means that the patient may be subject to the penalties of the Public Health Act, which are as follows:-

1. The patient may under certain circumstances be compulsorily removed by order of any justice to an isolation hospital.

2. By the provisions of Section 126.

Any person who

(1) While suffering from any dangerous infectious disorder wilfully exposes himself without proper precautions against spreading the said disorder in any street, public place, shop, inn, or public conveyance, or enters any public conveyance without previously notifying to the owner, conductor, or driver that he is so suffering: or

(2) Being in charge of any person so suffering so exposes such sufferer; or

(3) Gives, lends, sells, transmits, or exposes without previous disinfection any bedding, clothing, rags, or other things which have been exposed to infection from any such disorder; - shall be liable to a penalty not exceeding five pounds; and a person who while suffering from any such disorder enters any public conveyance without previously notifying to the owner or driver that he is so suffering shall in addition be ordered by the court to pay such owner or driver the amount of any loss and expense they may incur in carrying into effect the provisions of this Act with respect to disinfection of the conveyance.

By Section 127 it is incumbent upon every owner or driver of a public conveyance to immediately provide for the disinfection of such conveyance after it has to his knowledge conveyed any person suffering from a dangerous infectious disorder, and by the following section (128) a penalty is imposed upon any person - inclusive of an innkeeper - who lets any room in which a person has been suffering from a dangerous infectious disorder without having such room thoroughly disinfected.

In view of this, it has been found necessary to have a sub-section of the local act, where phthisis is placed on the list of notifiable diseases, so that early cases are not liable to prosecution for appearing in public places.

In favour of compulsory notification is the fact that it forms the most efficient method of handling the treatment of the disease, and of preventing those affected engaging in occupations likely to lead to the infection of others.

Against it is the fact that phthisis runs a long course and is not to be accurately described as a highly infectious disease. In the case of an infectious fever, running a short course, it is no great interference with the liberty of the subject, that isolation be enforced for a few weeks.

It is different with phthisis. If a man is to be deprived of his capacity as a wage earner because he has, through no fault of his own, become phthisical, it must be the duty of the State to provide for his wife and children. As there are over a hundred thousand consumptives in Britain, it were too great a burden to lay on the taxes of the country. The only solution must be a system of national assurance of all wage earners, after which, the State having provided for all contingencies, it is permissible to interfere with the liberty of those becoming phthisical, if this be in the best interests of the community.

At present in England, Sheffield and Bolton alone have compulsory notification. With regard to Sheffield Dr Scurfield, Medical Officer of Health, reports on the working of the system as follows: -

"Two inspectors are engaged in this work. They visit the homes of the patients and leave copies of printed instructions designed for promoting the chances of cure

and diminishing the risks of infection. They advise as to the necessity of plenty of fresh air, distribute pocket spittoons to those too poor to buy them, and disinfect the patients' rooms by spraying with formalin at each visit during the later stages of the illness. It is not to be expected that the full benefit of the new provision will be obtained until something is done in the way of providing hospital accommodation for the badly housed advanced cases and sanatorium treatment for the early and curable cases."

"There has been no opposition on the part of the public or the medical practitioners. I have no doubt there has been some little failure to notify on the part of medical practitioners. This, however, is what one would expect with a new Act." He adds: -

"I think that the Act is now being fairly well complied with. There are a small percentage of cases - about 1.2 per cent - where the medical man does not wish the inspectors to call, in which case the medical attendant is asked to fill in the enclosed form marked "A" and to use a copy of the enclosed leaflet 'Advice with regard to consumption.' (Form and leaflet not reproduced).

"The provisions of the Act are being carried out without friction, and I am of opinion that the work is doing a considerable amount of good."

In the city of St Louis, the following ordinance is in force.

ORDINANCE IN RELATION TO CONSUMPTION IN St. LOUIS

(United States of America)

Office of Health Commissioner.

St. Louis, April 28, 1905.

Ordinance No, 22,024.

An Ordinance Declaring Consumption to be Communicable and Prescribing Regulations to Control the Spread of the Disease.

Be it ordained by the Municipal Assembly of the City of St. Louis, as follows: -

Section 1. Consumption; whether classified as "tuberculosis of the lungs," "phthisis pulmonalis", "tubercular phthisis," "tubercular consumption," "pulmonary consumption," "tubercular broncho-pneumonia," or however nominated, is hereby declared to be a communicable disease, and the Health Commissioner, with the approval of the Board of Health, shall prepare rules prescribing essential measures for preventing the spread of the disease and promoting its arrest or recovery. He shall cause a printed copy of such rules to be distributed in every case occurring within the limits of the City of St Louis of whose existence he may have information.

Section 2. It shall be the duty of each and every physician in the City of St Louis, when called to attend any patient residing in the City of St Louis who has symptoms of tuberculosis, to promptly make or cause to be made a microscopical examination of the sputum of such patient. If this examination or the clinical evidence demonstrate tuberculosis, the physician shall at once report the case to the Health Commissioner, giving name of

patient, occupation, residence, place of employment, age, sex, nationality and social condition, and a record shall be kept of such reports, which shall be accessible only on order of the Health Commissioner.

Section 3. Whenever the Health Commissioner shall have information from the attending physician of the existence of any case of consumption in the city he shall cause the premises to be examined, and have notes taken of the sanitary condition of the same, the number of persons living in the house, and whether the patient occupies a room by himself or herself, or with others; and he is hereby directed to furnish in each case, directly or through the physician, (when one is in charge) a copy of the rules in relation to the management of the sanitary features and surroundings of the case as herein provided for.

Section 4. Houses in which there are cases of consumption shall not be placarded for that disease.

Section 5. When a case of supposed consumption is reported to the Health Commissioner, and no physician is in charge, the Health Commissioner may appoint a city physician to examine and report the same. If the clinical evidence or a microscopical examination of the sputum of such patient demonstrate tuberculosis, the Health Commissioner shall furnish to the patient, or to the patient's nearest relative, friend or caretaker, a printed copy of directions as to the best means of preventing the spread of the disease and promoting recovery. He shall instruct the inspectors of the Health Department to report whether directions and instructions are obeyed.

Section 6. The Health Commissioner shall have the power to cause a microscopical examination to be made of the sputum of persons having symptoms of tuberculosis, whenever it be requested by the attending physician or by the city physician detailed to examine the case, and he shall have the power to order said examination to be made by any competent person in the employ of the Health Department, and such person so employed shall promptly make the examination and a report thereof, free of charge. When such persons shall have completed this examination, the result shall be reported in duplicate, one copy to be forwarded to the physician upon whose application the examination was made and the other to the Health Commissioner. Law following officer, the doctor receiving 2/5

Section 7. Rooms that have been occupied by consumptive patients shall be thoroughly cleaned and disinfected as soon as the case has terminated, or been removed, permanently or temporarily. This may be done under the direction of the attending physician, but such disinfecting and cleaning must be done in accordance with the rules of the Health Commissioner, and the physician must report to the Health Commissioner how and when this cleaning and disinfection were done. Upon application to the Health Commissioner, all apartments that have been recently occupied by cases of consumption will be disinfected free of charge. Premises not disinfected as provided by this section are hereby declared to be a nuisance and detrimental to the public health.

Section 8. Any physician failing to comply with any of the provisions of this ordinance, or any person or

persons resisting the enforcement of any of the provisions of this ordinance, shall be deemed guilty of a misdemeanor, and upon conviction, shall be fined not less than fifty nor more than two hundred and fifty dollars, to be recovered for the use of the City of St. Louis before any court having competent jurisdiction.

Approved April 7, 1905.

Voluntary Notification.

This has now been introduced into a large number of towns, Brighton leading the way in 1899, where it is carried out as follows: -

(1) Every case of phthisis is notified by a doctor or Poor-Law relieving officer, the doctor receiving 2/6 for every private case, and 1/- for every hospital or parish case.

(2) The patient's room is cleansed and disinfected, when required. This is always done when a change of address occurs, or when the patient is admitted to the Sanatorium. Bedding is usually also disinfected by steam, and all these measures are carried out in every instance after the death of a phthisical patient, whether or not the patient's illness has been previously notified.

(3) The patient is instructed as to the precautionary measures required, printed cards being given in addition to exact verbal instructions. If poor, he is supplied with a pocket spittoon for outdoor use, and with Japanese paper handkerchiefs for indoor use.

(4) If the patient is lodged under unfavourable conditions, and especially if he is in danger of infect-

ing others, he is, by arrangement with his medical attendant, admitted for a month into the Borough Sanatorium.

(5) A careful sanitary inspection is made of each house in which the phthisical patient lives, and sanitary defects are remedied.

The following card is given to patients:

Precautions for Consumptive Persons.

Consumption is, to a limited extent, an infectious disease. It is spread chiefly by inhaling the expectoration (spit) of patients which has been allowed to become dry and float about the room as dust, or by directly inhaling the spray which may be produced when a patient coughs.

Do not spit except into receptacles, the contents of which are to be destroyed before they become dry. If this simple precaution is taken, there is practically no danger of infection. The breath of consumptive persons is free from infection, except when coughing.

The following detailed rules will be found useful, both to the consumptive and to his friends.

1. - Expectoration indoors should be received into small paper bags and burnt immediately; or into a receptacle which is emptied down the drain daily and then washed with boiling water.

2. - Expectoration out of doors should be received into a suitable bottle, to be afterwards washed out with boiling water. If a paper handkerchief is used, this must at once be placed in a waterproof bag, the contents subsequently burnt, and the bag washed daily.

3. - Ordinary handkerchiefs, if ever used for expectoration, should be put into boiling water before they have time to become dry; or into a solution of a disinfectant, as directed by the doctor.

4. - Wet cleansing of rooms, particularly of bedrooms occupied by sick persons, should be substituted for "dusting" and sweeping.

5. - Sunlight and fresh air are the greatest enemies of infection. Every patient should sleep with his bedroom window open top and bottom, a screen being arranged, if necessary, to prevent direct draught and the patient should occupy a separate bedroom.

N.B. - The patient himself is the greatest gainer by the above precautions, as his recovery is retarded and frequently prevented by renewed infection derived from his own expectoration.

6. - Persons in good health have little reason to fear the infection of consumption. Over-fatigue, intemperance, bad air, dusty occupations and dirty rooms favour consumption.

The course of notification is indicated in the following table: -

Year	No. of new cases notified	No. of Cases Re-notified		New Cases notified per 100000 of population.	No. of New Cases treated in Sanatorium	No. of Annual deaths from Tuberculosis in Brighton
		During 1904	Prior to 1904			
1899	111	-	-	92	-	215
1900	105	-	-	85	-	232
1901	153	9	--	124	-	237
1902	224	35	17	179	31 (from May)	227
1903	316	45	37	251	98	278
1904	363	49	36	286	130	259

All Day Sanatoria.

These mostly exist in Germany, where they are run in conjunction with the system of national workman's insurance. At the Royal Victoria Hospital, Edinburgh, this treatment has also been introduced in conjunction with the Victoria Dispensary.

In Germany the great point about these Sanatoria is their extraordinary cheapness. In its original simplicity an open-air institution can be arranged, if the barrack is hired, for 4 to 5000 Marks; with 100 to 120 patients it is self-supporting. The selling of milk forms an important source of income. In Berlin for instance, the milk is bought at wholesale prices, while the sick clubs pay retail prices to the Institution; this difference almost covers the running expenses. The idea of open-air Institutions originated by Dr Becher was first realised in Berlin in 1900, thanks to the active co-

operation of Prof. Pannwitz. The rapid development of the open-air Institutions is shown in the rapid and constant increase of the annual number of nursing days in the institutions.

1900	12,011
1901	28,914
1902	67,626
1903	92,231
1904	132,936.

The following cities have erected open-air Institutions after the pattern of Berlin: Leipsic, Frankfurt-on-the-Main, Posen, Cassel, Karlsruhe, Stettin, Halle, Dusseldorf, Dessau and Munich, in Germany; Mons, in Belgium, and Vienna in Austria.

For the construction of an open-air Institution, a wooded tract of land $\frac{3}{4}$ of a hectare large is enclosed in a railing 1.50 m. high. A Döcker barrack is first put up, containing office, examination-room, kitchen, pantry and three small compartments; then an Abyssinian well is bored, and a filter dug for the waste-water. Then there must be put up on the grounds: (1) a hall for the accommodation of the patients in bad weather, 24 m. long, 5.80 m. wide, 3 m. high in back, and 4.10 m. high in front: (2) a wash-and china-pantry 2.70 m. long, 1.80 m. wide, 2.20 m. high in front, 2.50 m. high in back: (3) a wash-room for the patients, 3.45 m. long, 1.85 m. wide, and 2.35 m. high: (4) a bath-room of about the same proportions: (5) a water-closet 4.10 m. long, 2.55 m. wide, and 2.90 m. high. Then there are shelves, tables and gymnastic apparatus. Each patient is given a reclining-

chair and a cover. Lastly, there is the fitting up of the kitchen, the providing of dishes and drinking vessels, knives, forks, spoons, spit-bottles, and table linen. The management of the institution is in the hands of a nurse; under her are a cook and two kitchen maids, and a night watchman.

The work is superintended by a patroness and a physician. The patients come as early as possible to the institution and remain till evening. The female patients are allowed to bring one to two children too young to attend school. The permission to bring infants has turned out very well. In the Berlin institutions the patients receive milk, dinner, bread and butter and sandwiches, under some circumstances, cocoa, etc. (In other institutions, for instance in Frankfurt and Düsseldorf a certain sum per day is paid for board). Dinner, consisting of $\frac{1}{4}$ of a pound of meat, and soup or vegetables, costs 30 Pfennig (about 3d.) a litre of milk (the patient receives according to the orders of the physician, one to two litres or more a day) costs 20 Pfennig, bread and butter 5 Pfennig, and sandwiches 10 Pfennig. All this only refers to open air institutions for adults. The board in the childrens' open-air institutions is arranged differently: here the children receive full board for 50 Pfennig a day: the childrens' institutions also differ from the institutions for adults in respect to medical treatment. The patients of the institution for adults remain, during their stay at the institution under the care of their physician in town; the physician of the institution only acts in case of necessity. In the institutions for

children, on the contrary the patients receive full medical treatment from the directing physician.

The Home Treatment of Phthisis.

It is essential in the treatment of phthisis among the working classes, that there be some form of home supervision. This idea has been elaborated and carried out in Edinburgh at the Royal Victoria Dispensary by Dr R.W. Philip, who has described the working of the Dispensary as follows: 380

"Up to 1887, there was in Edinburgh no central or concerted action in relation to the treatment of pulmonary tuberculosis. Patients were received and excellently treated in the Royal Infirmary and other Hospitals so long as it was possible to keep them. The general dispensaries of the city received and prescribed medically for those consumptive patients who presented themselves for treatment. Such treatment necessarily consisted largely in the prescription of some form of cough mixture. The duration of the patient's treatment depended on the continuance of the more aggressive symptoms, and his faith in the prescriber or prescription. Consumptive patients, when too ill to come to the dispensary, were commonly relegated to the list of chronic or troublesome patients, visited occasionally by a frequently changing series of medical students, whose conceptions of treatment, doubtless excellent so far as they went, did not extend very far. As an officer of the Royal Infirmary and a large public dispensary, one felt frequently heart-sick at the evident ineffectiveness of the assault made on so tremendous an

evil. Such considerations led naturally to the practical question whether some more definite and organised effort might not be made. Accordingly, in the autumn of that year, having satisfied myself that a well-directed movement towards the end in view would have the approval of those who might take the trouble to think of the matter, I succeeded, with the help of a few kind friends, in establishing the Victoria Dispensary for Consumption in the heart of Edinburgh. Its object was to afford a central institution toward which all poor persons affected with consumption might be directed. The scope of the institution was a large one, and included: -

(a) The reception and examination of patients at the Dispensary, and the keeping of a record of every one thus received, with an account of his illness, history, surroundings, and present condition, the record being added to on each subsequent visit.

(b) The instruction of patients how to treat themselves, and how to prevent or minimise the risk of infection to others.

(c) The dispensing of necessary medicines, disinfectants, and sputum-bottles, and, where the family conditions seemed to warrant it, of food stuffs and the like.

(d) The visitation of patients at their own homes, more especially of patients confined to the house or to bed, and this for the double purpose of treatment and of investigation into the state of the dwelling, the general conditions of life, and the risk of infection to others in the neighbourhood.

(e) The selection of more likely patients for hospital treatment, either of early cases for sanatoria, or of late cases for some incurable institution.

(f) The guidance generally of patients, and friends of patients, and other inquirers on questions related to consumption."

Dr Philip ³⁸¹ also considers that a completely organised warfare against phthisis should have the following base: -

^{of} (1) A Home for dying patients, chiefly in the interests of other persons.

(2) A Sanatorium or Sanatoria for selected patients who, with a view to the cure of the disease, require a special régime not procurable in their own homes.

(3) Colonies for the after life and supervision of patients in whom the disease has been sufficiently arrested to make selected open-air employment feasible and desirable.

(4) A Tuberculosis Dispensary, with the functions and relations I have attempted to outline. ^{or}

A Tuberculosis Dispensary, unless carried out on lines such as these, is of very little value. Especially is this the case in those where medicines are sold at reduced prices, and there is no selection of patients. These are nothing better than charitable organisations for the distribution of cod liver oil, and in the experience of the writer constitute one of the ^{cravata} ~~general~~ forms of Hospital abuse.

CHAPTER XXIV.

THE STATE AND PHTHISIS.

The duty of the State with regard to phthisis lies in the abolition of those predisposing causes of the disease which are under its control, and in legislation to check the disease at its source.

Overcrowding and slum property has a direct relation to the disease, as seen from the following tables from the Health Reports of the County of London, and the London County Council.

TABLE I.

Proportion of total population living more than two in a room (in tenements of less than five rooms)	Death rate per 1000 living				
	1894	1895	1896	1897	1898
Districts with under 10 %...	1.07	1.18	1.07	1.14	1.10
" " 10 to 15 %...	1.38	1.49	1.46	1.42	1.43
" " 15 to 20 %...	1.57	1.64	1.61	1.63	1.61
" " 20 to 25 %...	1.81	1.83	1.67	1.75	1.80
" " 25 to 30 %...	2.11	2.09	2.06	2.10	2.07
" " 30 to 35 %...	2.26	2.42	2.13	2.32	2.42
" " over 35 %...	2.46	2.66	2.55	2.64	2.65

TABLE II

LONDON, 1901-5

Phthisis death-rates in relation to "overcrowding" (1901) Census.

Proportion of total population living more than two in a room (in tenements of less than five rooms).	1901-5 crude phthisis death rate per 1000 persons living	Standard death-rate	Fraction for age and sex correction	Corrected death-rate per 1000 persons living.	Corrected death-rate (London 1000)
Under 7.5 per cent	1.109	1.718	1.00991	1.120	717
7.5 to 12.5 " "	1.376	1.705	1.01761	1.400	896
12.5 to 20.0 " "	1.495	1.771	.97969	1.465	937
20.0 to 27.5 " "	2.075	1.805	.96124	1.995	1276
Over 27.5 " "	2.068	1.651	1.05090	2.175	1390
London	1.563	1.735	1.00000	1.563	1000

It is thus obvious that a measure for the better housing of the working classes would do much to diminish their predisposition to phthisis.

Legislation against the Sources of Infection

To secure the cessation of the infection of man by the products of tuberculous bovines, it were necessary to make it illegal to keep alive or to sell for any purpose, a tuberculous animal, and to insist that all bovines, whether used for dairy purposes or for "killing" must pass the tuberculin test. The enforcement of such an Act would mean large sums in compensation during the earlier years of its operation, which would be well repaid by a diminished death rate from phthisis.

The State of Columbia has enforced the following special regulations against the infection of infants.

1. That the milk which is supplied to infants under the age of 3 years in the District of Columbia should be certified by the health officer. Milk should contain not more than 5,000 bacteria per cubic centimeter, should not be more than twelve hours old, and should be delivered in artificially cooled packages, the term "artificially cooled packages" not to be interpreted to require the constant presence of ice, but merely the keeping of the contents at the prescribed temperature.

2. That the health officer of the District be authorized to advertise for dairies which will be willing to so modify their barns, stables, etc., if necessary, as to secure a license from him for the production of certified milk under the most modern improved sanitary conditions.

3. That each cow furnishing the milk in the dairy be tested, under the supervision of the health officer, for tuberculosis or other contagious or infectious diseases, and that any animal so suffering be excluded from the herd.

4. That a daily sample or samples drawn from the supply of each certified milk furnished to the city be secured for the purpose of making the bacterial count and determining the chemical composition, etc.

5. That parents and guardians be urged to use only certified milk, at least for infants' food, in the District of Columbia for all infants under the age of 3 years.

6. That if private dairies cannot be induced to furnish certified milk, the Commissioners of the District of Columbia, as a health measure affecting in the highest degree the welfare of the District of Columbia, be asked, if it can be legally done, to establish a municipal dairy for the purpose of furnishing certified milk for the use of infants under 3 years of age in the District of Columbia.

7. That, pending the time which must necessarily elapse for the inauguration of a service of certified milk for infants' use, the health officer be authorised, as a result of his inspections, to publish a list of dairies from which the milk supply is drawn, giving the average rating of each dairy, the chemical composition and bacterial count of the samples.

8. That for the purpose of securing modified milk for infants in ill health, according to the prescription of physicians, the milk commission of the District Medical Society be requested to secure the establishment of a laboratory under its supervision in which only certified milk shall be used, and in which, by the use of a separator or otherwise, milk of a definite chemical composition may be prepared from certified milk, in harmony with the physicians' prescriptions relating thereto.

9. That a complete chemical and bacteriological laboratory be established in connection with the health office of sufficient size to do all necessary work.

10. That the term "certified milk" as used herein is to be applied to milk secured at dairies subject to a periodic inspection, and the products of which are subjected to constant analysis, The cows providing the milk

must be properly fed, free from tuberculosis or other contagious diseases, and housed in clean stables, properly ventilated, and they must be supplied with wholesome water and feed, and kept clean. The milk must be drawn under precautions to avoid infection, immediately strained and cooled, and packed in sterilised bottles which are kept at a temperature of not to exceed 50° F., until delivered to the consumer.

CHAPTER XXV.

BACTERIOLOGICAL TECHNIQUE

Ziehl-Neelsen Stain. This is made by adding 1 c.c. of a saturated alcoholic solution of fuchsin to 9 c.c. of a 5% solution of phenol.

A small piece of sputum is spread in a thin layer on a slide, and dried by rapidly passing over a Bunsen flame. The above solution is heated to boiling in a test tube, and a drop allowed to fall on the slide. Stain for five minutes.

Decolourise in 20% nitric acid until the red is no longer visible.

Counterstain in a 1% solution of Methylene blue.

Ehrlich-Weigert Stain. Eleven centimetres of a saturated solution of fuchsin in absolute alcohol is added to 100 c.c. of the saturated solution of commercial aniline oil (made by shaking up the oil in water and then filtering). This should be made fresh every third or fourth day. A small bit of the sputum is picked out on a needle or platinum wire and spread thin on the top-cover so as to make a uniformly thin layer. The top-cover is slowly dried about a foot above a Bunsen burner. Sufficient of the staining fluid is then dropped upon the top-cover, which is held at a little distance above the flame until the fluid boils. The staining fluid is

then washed off in distilled water or put under the tap, decolorised in 30 per cent nitric-acid fluid, again washed off in water, and mounted on the slide. In doubtful cases the long process is used, the cover-slips remaining twenty-four hours in the stain. The bacilli are seen as elongated, slightly curved, red rods, sometimes presenting a beaded appearance. They are frequently in groups of three or four, but the number varies considerably. Only one or two may be found in a preparation or, in some instances, they are so abundant that the entire field is occupied. Repeated examinations may be necessary.

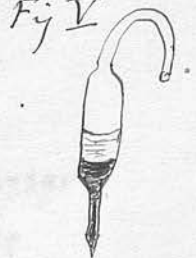
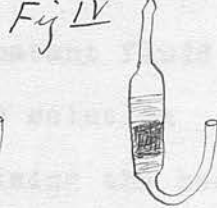
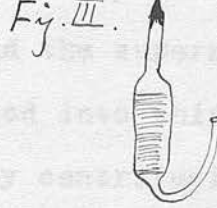
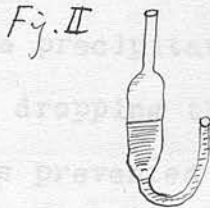
OPSONIC INDEX

This most delicate method of estimating the resistance of the body to any bacteria, discovered by the now classic researches of Wright and Douglas, has proved itself of extreme value in the diagnosis and prognosis of phthisis, and has further been used in the treatment of infective diseases by the injection of vaccines, or dead cultures of the causal bacillus. As frequent reference will be made to it in the course of this work, a short account of its nature and technique will not be out of place. The method described here is that of Wright and Douglas, modified by the writer.

The three essentials of the Opsonic Index are (1) blood serum, (2) living phagocytes, and (3) an emulsion of the bacteria, against which it is desired to find the resistance of the patient.

1. Serum. This is obtained in the following manner.

The thumb is washed with alcohol, then with normal saline (.85%) after which an elastic ligature is wound round the base in order to cause congestion. Bend the thumb, prick it at the base of the nail with a clean beaver pointed needle, and allow the blood to run into a Wrights capsule (Fig. I.



The blood is allowed to run down the bent limb of the tube until the latter is half full. (Fig. II.) The straight limb is then heated, and sealed by a bunsen flame or spirit lamp. The contraction of the heated air in the upper part of the tube now draws the blood out of the bent limb into the body of the capsule. (Fig. III.) Care must be taken not to let the flame touch that part of the tube containing the blood, which is avoided by holding the body of the tube between the finger and thumb. The capsule is now placed upright in a box of sand until the blood coagulates. (Fig. IV.) Lastly it is turned upside down and centrifuged, whereby the serum is separated from the clot. (Fig. V.) Above is the serum, below is the clot. Make a nick with a glass file on the outside of the capsule a little above the level of the serum, break open the capsule with the fingers, and place the portion containing the serum upright in the box of sand. Besides the capsule of the patients blood another should be prepared from

several healthy persons to serve as a control.

2. The living Phagocytes. These may be taken from the patient or from a healthy person. Prepare the thumb as before, and allow ten drops of blood to drop into an ordinary urine centrifuge tube filled with a solution of sodium citrate 1.5% and sodium chloride .75%. Shake up well, then centrifuge until the red and white corpuscles are precipitated and the supernatant fluid is clear. By dropping the blood into this solution coagulation is prevented and by centrifugising the heavier red cells fall first, while the whites form a thin buff layer above them. With a pipette draw off the supernatant fluid containing the blood plasma, taking care not to disturb the red and white corpuscles at the foot of the tube. Next fill up the tube with a .85% solution of sodium chloride. Shake up well and again centrifuge until the white cells are precipitated above the red. Repeat this procedure. The phagocytes, numbering 75% of the white cells, are now quite washed and form a thin layer above the red corpuscles at the foot of the tube. Draw off the supernatant saline solution with great care until an imperceptible amount of fluid remains over the washed corpuscles, and place the tube upright in the sand box.

3. The Bacterial Emulsion. Take a piece of tubercle culture about the size of a large pin's head, and grind it down without water for five minutes in an agate mortar. Now add very gradually about 1 c.c. of

a .1 solution of sodium chloride, stirring all the time. Pour into a centrifuge tube and centrifuge for five minutes to get rid of any clumps which may be left. A pale opalescent emulsion should result. After a little experience one can usually tell the correct colour of a well-made emulsion, but if there be any doubt a drop should be examined on a slide under a high power of the microscope, and if any clumps are present it must be centrifuged again.

The phagocytes, bacteria and serum being prepared they must now be mixed in a Wright's Pipette. This is a glass tube drawn out into a long fine pipette, on which a mark is made with a grease pencil about .5.c.m. from the end. On the large end fit a rubber teat.

(Fig VI.)

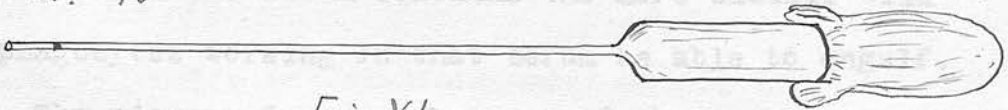


Fig. VI.

By means of the rubber teat draw into the pipette three equal quantities of washed corpuscles. The method of measuring three quantities is to draw the fluid into the pipette as far as the grease pencil mark, then let in a little bubble of air, which is followed by another quantity of fluid, and so on. In this way take up three quantities of washed phagocytes, one quantity of bacterial emulsion, and three of the patient's serum. It is essential to take up the quantities in this order to avoid contamination of the phagocytes and emulsion by the serum. Expel the contents of the

pipette on to a clean slide, then draw up again in order to mix well the three solutions. Repeat this five times. Now having drawn the mixture into the pipette seal off the end in the flame, and remove the rubber teat.

In a similar manner prepare a pipette of three quantities of washed phagocytes, one quantity of bacterial emulsion, and three of normal serum.

Put a distinguishing mark on each pipette and place in an incubator at 37° C. for 15 minutes. This incubation causes phagocytosis, as the serum acts on the bacilli and prepares them for ingestion by the phagocytes. Those bodies in the serum which are antagonistic to the bacilli and render them an easy prey to the phagocytes are the opsonins. It is obvious that the more opsonins the serum contains the more bacilli will the phagocytes working in that serum be able to engulf.

The pipette is now taken out of the incubator, the sealed end broken with the fingers, and the contents expelled several times on to a glass slide by blowing with the mouth through the wide end of the tube. This ensures even distribution of the phagocytes.

A drop of the mixture is now placed on a clean glass slide, whose surface has been well roughed by rubbing with .000 emery paper. Lay the slide flat on the table and make a blood film either by spreading out the drop with the edge of another slide, held at an angle of 45° drawn over the surface of the first slide, or by spreading it out with a piece of cigarette paper. The film should be rapidly dried over a flame, after which it is:

1. Fixed in saturated corrosive sublimate solution for 5 minutes.
2. Washed with water.
3. Stained for five minutes in with hot Carbol-Fuchsin solution.
4. Washed with water.
5. Decolourised for five minutes in 5% $H_2S O_4$ solution.
6. Decolourised for 5 minutes in 2% acetic acid solution.
7. Washed for 5 minutes in water.
8. Stained for 10 seconds in the following:

Medicinal Methylene Blue5 grm.
Sodium Carbonate2 grm.
Aqua dist.	100 cc
9. Wash off excess of stain, blot between filter paper and dry.

The films are now examined under an oil emersion lens and a low eye-piece. At the free edge of the film phagocytes will be seen with nucleus and protoplasm stained blue, while the engulfed tubercle bacilli show up a bright red. If the emulsion of tubercle bacilli be of about the proper strength the preparation made from normal serum should have one T.B. or so in each phagocyte. In each film count the number of bacilli in 50 phagocytes.

Let us suppose the patients film has 30 T.B's in 50 phagocytes and the control film has 50 T.B's in 50 phagocytes, then the Opsonic Index is $\frac{30}{50}$ or .60. The Opsonic Index is the number of bacilli in the phagocytes

of the patients blood film, divided by the number of bacilli found in the phagocytes of the control film. As the control is normal if the patient's serum is rich in opsonins the Index will be high, if poor in opsonins, low.

F O R M U L A E.

I. Febrile Mixture.R_x

Liquor Ammon acetat 3iv
 Spir. aether nitrosi 3 i
 Tinct. aconiti M.i
 Aqua camphor ad 3 i

Sig. To be taken at bedtime.

II. Expectorant Cough Mixture.R_x

Ammon. carbonalis 3 iii
 Tinct. scillae 3 i
 Spir. aether nitrosi 3 i
 Tinct. Strophanthi M xxxii
 Infus. Senegae ad 3 iv.

Sig. A Tablespoonful thrice daily.

III. Astringent Bismuth Mixture.R_x

Bismuth Carbon 3 i
 Sod. bicarb. 3 vi
 Liq. Morphinae hydrochlor. 3 iii
 Mucilag. Acaciae 3 iv.
 Aqua Menth. Pip. ad 3 viiii.

Sig. A tablespoonful three times a day.

IV. Astringent Chalk Mixture.R_x

Liq, Morphinae hydrochlor. 3 iii
 Tinct. Catechu 3 i
 Mistura Cratae as 3 viiii

Sig. A tablespoonful three times a day.

V. Lead and Opium PillR_x

Pil Plumbi cum opii (B.P.)

gr II - IV

Sig. One at bedtime.

VI. Starch and Opium Enemata.

R_x
 Liq. morphinae M xxv.
 Mucilage of Starch ad 3 xx.

VII. Paint for Thrush.

R_x
 Boroglyceride 3 i
 Glycerine 3 iii

Sig. Paint the patches with a camel hair brush.

VIII. Gastric Tonia.

R_x
 Tinct. Rhei 3 ii
 Sod. Bicarb. 3 i
 Ammon. bicarb. gr. ii
 Ol. Menthol Mi
 Aqua chloroformi ad 3 viii.

Sig. A Tablespoonful in a quarter of a tumbler of

cold water fifteen minutes before food.

IX. Mild Pulmonary Sedative.

R_x
 Syrup tolu, 3 iii
 Ol. Menthol Mv.
 Oxymellis Scillae 3 i
 Spir. Chloroformi 3 iv
 Glycerine 3 iv
 Aqua ad 3 viii

Sig, A tablespoonful four times a day.

X. Inhalation.

R_x
 Ac. carbolic pur. 3 ii
 Cressoti 3 ii
 Ol. Pini (Scotch) 3 ii
 Alcohol absol. 3 i
 Spir. Vini rect. ad. 3 i

Sig. 10 to 20 drops to be inhaled twice daily.

XI. Formalin Inhalation

Formalin c.c. 6

Water cc 94.

XII. Hydrocyanic Mixture.*To be sprinkled on an inhaler,
used for ½ hr. twice daily.*

R.

Acid Hydrocyan dil. 3 i
 Acid Hydrochlor. dil. 3 i
 Acid Nitric dil. 3 ii
 Glycerini 3 i
 Infus. Quassia ad 3 iv.

Sig. A teaspoonful four times a day.

XIII. Brompton Mixture.

R.

Liq. Morphinae hydrochlor. 3 ii
 Acid. hydrocyan. dil. 3ss
 Syrup. Tolu 3 i
 Acid Infus. Rosae, ad 3 iv.

Sig. A teaspoonful four times a day.

XIV. Heroin Mixture.

R.

Heroin hydrochlor. gr. 1¼-2
 Glycerine 3 i
 Aqua Lauroceresi 3 v
 Spir. Pruni Virg. 3 i
 Aqua Camphor ad 3 iv

Sig. A teaspoonful twice to four times a day.

XV. Pill for Night Sweats.

R.

Quinine sulph. gr. i
 Pulv. digitalis gr. ½
 Pulv. opii gr. ¼

Sig. One an hour before sleep, or one every
 six hours.

XVI. Tonic.

R/

Tinct. Ferri perchlor. 3 v.
 Liq. Arsenicalis hydrochlor. 3 i
 Aquam ad 3 iv.

Sig. A teaspoonful in water sucked through a glass
 tube thrice daily after food.

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