Essays on consumption : together with some clinical observations and remarks on pneumonia / by J. Edward Squire.

ESSAYS ON CONSUMPTION

J. EDWARD SQUIRE. M.D.

CONSUMPTION.

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ON

CONSUMPTION

TOGETHER WITH SOME

CLINICAL OBSERVATIONS

AND

REMARKS ON PNEUMONIA.

BY

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WITH AN INTRODUCTION

BY

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AUTHOR'S PREFACE.

In collecting into one Volume some of the Papers and Articles which I have at various times read before the Societies or published in the medical journals, I have to acknowledge my obligation to the Council of the Epidemiological, Royal Medical and Chirurgical, and Pathological Societies, and to the editors of the various journals who have kindly given permission to reprint those which have already appeared in the Press.

In selecting the papers I have endeavoured, without materially altering the original text, to present a fairly connected summary of the ætiological factors of Consumption which serve to guide us in formulating preventive measures, and to indicate these measures.

The relative importance which belongs to the different ætiological factors is, however, not preserved, since undue prominence is naturally here given to those points which I have made the

subject of personal study. Thus the question of heredity takes up an undue proportion of space, as does also the influence of other pulmonary diseases in predisposing to Consumption. The proper perspective, if I may so call it, of the clinical picture is sacrificed to give prominence to the writer's individual investigations and deductions. But if it were not for the writer's personal contribution — small and insignificant though it may be—there would be no reason for the present volume, since treatises on Consumption are numerous and many of them excellent.

Some clinical observations have been added, some of which may have a certain value. The observation on page 238, relating to the seat of secondary infection of one lung from the other, is one of the outcomes of the writer's observation which he has not seen mentioned by others, and the chart of the daily temperature curve of acute Consumption (mixed infection) is also original. This chart appeared in a previous work—*The Hygienic Prevention of Consumption*—published by Chas. Griffin & Co., by whose courtesy I am permitted to reprint it.

Two papers on Pneumonia, and one on a Cardio-respiratory Murmur, though not directly concerned with Consumption, have been included amongst the clinical observations.

There is of necessity some repetition in such a collection as this, since the various papers overlap as it were, each one having been written so as to be reasonably complete in itself.

The chronological sequence of the papers has not been followed, but the date of each is mentioned in the Table of Contents.

There are but few men who, by their personal observations on disease, light upon discoveries which remain as monuments to themselves and constitute landmarks to all succeeding investigators and students.

The majority of the landmarks which guide us in our study of disease are like cairns raised by the single stones which are, from time to time, placed by the many explorers in the realms of Medicine, and which, though each contribution is in itself insignificant, gradually increase in size

until they rival in importance the monuments raised by individuals.

To one such cairn which has lately assumed imposing dimensions — the cairn representing Tuberculosis—I here add a few small stones.

To Sir William Broadbent I am indebted for his kindness in giving the time out of a busy life to look through these essays, and for writing an Introduction to the collection.

J. EDWARD SQUIRE.

5, Harley Street, W.

TABLE OF CONTENTS.

Part II.—THE PREVENTION OF CONSUMPTION.

Chapter VII.

Chapter VIII.

Chapter IX.

Chapter X.

Part III.—THE TREATMENT OF CONSUMPTION.

Chapter XI.

Chapter XII.

Chapter XIII.

Chapter XIV.

Chapter XV.

Chapter XVI.

Part IV.—CLINICAL OBSERVATIONS.

Chapter XVII.

Chapter XVIII.

Chapter XIX.

Chapter XX.

Chapter XXI.

Chapter XXII.

PNEUMONIA.

Chapter XXIII.

Chapter XXIV.

Chapter XXV.

ILLUSTRATIONS.

INTRODUCTION

By Sir William Broadbent, Bart.

INTRODUCTION

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The collective publication of Dr. J. Edward Squire's valuable Essays on Consumption is well timed. The fact that Consumption is preventible, which forms the subject of the earlier chapters, is gradually reaching and impressing the public mind, and more rapidly open air is obtaining recognition as the most important of the remedial influences at our command in the treatment of Tuberculosis. Dr. Squire, however, is not simply an echo of ideas which are becoming generally accepted. The Essays date from 1889, and it is right to point out that the instructions to the Out-patients of the North London Consumption Hospital, of which Dr. Squire is one of the Physicians, for the destruction of sputa and other preventive measures

given in 1892 were, so far as my knowledge goes, the first of the kind to be issued in this country.

All parts of these Essays are worthy of attention. The prevention of Consumption and tuberculous disease generally is fully discussed in all its branches. While favouring notification of Tuberculosis as a contagious disease, both in regard to men and cattle, Dr. Squire recognises the practical difficulty attending its application, but to quote his conclusion: "The question of "preventive legislation directed towards the "diminution of tuberculous diseases is not to "be put aside because of its difficulties, though "they are many and great. Where the national "good is concerned, means have always been "found to grapple with and overcome even "greater difficulties than present themselves "here." It may be added that they are already being resolutely dealt with by earnest and intelligent Medical Officers of Health in various parts of the country.

On the question of Treatment, Dr. Squire has a special right to be heard, since he has given all the more important methods which have been advocated a fair and extended trial, and it is interesting to find that he has arrived independently at substantially the same conclusions as Sir Samuel Wilks. Recoveries take place almost impartially under a great variety of remedies, the underlying element of success in all being mainly attention to the general health.

There are many other points to which attention might have been called, such as the causation and diagnosis of Consumption, but this is not a review but an Introduction. I have considered it my duty to write it as a recognition of Dr. Squire's early efforts in the work of prevention of Tuberculosis, but I can also warmly recommend his book on its merits.

W. H. BROADBENT.

Part I.

THE ÆTIOLOGY OF CONSUMPTION.

CHAPTER I.

DEFINITIONS.

The terms "Consumption" and "Phthisis," originally suggested by the prominent symptom of wasting or emaciation, are employed synonymously to designate Tuberculosis of the lung. They are convenient as being short and in common use, but they are indefinite since they refer to symptoms or results rather than to pathological causes. These terms were applied to any morbid condition which produced certain symptoms, and so included more than one disease. It is in the restricted sense of Tuberculosis that I employ these terms, and the following remarks are equally applicable to both "Consumption" and "Phthisis." We must at the outset have an agreement as to the definition of the terms. The term "Phthisis" is convenient from its shortness, and having been long in use it is more or less familiar outside the medical profession. But having been in use before the bacillary nature of pulmonary Tuberculosis was established, or even dreamed of, it was employed to designate several conditions of the lung now known to be distinct in causation and in pathology. We recognise morbid states of the lung producing partial consolidation, or restricting expansion, or even leading to the formation of cavities, which are not due to a tubercular

origin, and in which the tubercle bacillus is not discoverable; and with these we may have febrile disturbances, wasting and prolonged illness, and occasional hæmorrhage, as well as cough.

Some of these morbid conditions would be included under the comprehensive term of "Phthisis," and lungs so damaged offer a ready soil for the development of the tubercle bacillus if infection is present.

But until tubercular infection is superadded, these diseases are to be distinguished from pulmonary Tuberculosis, to which the term "Phthisis" is now more generally restricted.

It was the discovery of the bacillary cause of tubercular Phthisis which justified the inclusion of Consumption amongst the preventible diseases; and with so widely prevalent and so fatal a disease the possibility of prevention gives hope for the future.

The importance of the tubercular disease lies in the fact that the causative bacillus must gain entrance into the lung *from without*, and the disease is impossible without infection from some pre-existing case of Tuberculosis. Recognising this, we may suggest prophylactic measures based upon a study of the life-history of the micro-organism, and of the modes by which it is given off from the bodies of the sick, and by which it gains entrance into those of the susceptible.

We see, then, that over-crowding does not *produce* pulmonary Tuberculosis, neither do inclement climate, poor food, nor imperfect clothing. But these have their influence—and that a most powerful one—in favouring the spread and severity of the disease when tubercular

infection is introduced into a community. Over-crowding not only aids the spread of Phthisis by tending to concentration of infection, but predisposes to the disease by diminishing the resisting power of individuals living under such unhygienic conditions. Inclemency of climate, by causing lung diseases and bronchitis, may render the respiratory organs specially receptive of bacillary infection, and insufficiency of food and clothing tends to a general diminution of resistance to disease.

Thus the *predisposing causes* of Phthisis are many and powerful, and are not all of them controllable. The existence of immediate predisposing causes, however, accentuates the importance of prophylactic measures directed against the spread of the exciting infection.

Tuberculosis may be defined as a specific inflammatory affection due to the action on the tissues of the *bacillus tuberculosis*. The causative bacillus may, though rarely, be implanted in the tissues during their development *in utero*, and produce disease before or immediately after birth. This may be distinguished as "Congenital Tuberculosis," which may affect any organ or tissue, but is usually generalised—General Tuberculosis. More commonly the causative micro-organism gains entrance into the body from without *after birth*, the mode of entrance depending upon the age, occupation and surroundings of the individual. All who are exposed to infection are not equally susceptible, a predisposition being necessary unless, as may occasionally happen, the infection is very intense. This predisposition may be general, or local, or both; in the latter case the local predisposition may determine the seat of the primary manifestation of the disease.

General predisposition—the most powerful—may be constitutional (inherited), or may be acquired. The primary seat of the disease may be determined partly by the mode of infection, partly by local damage (predisposition) from injury or disease in a particular organ or tissue. In infancy infection is most frequently conveyed in the food—the milk of a consumptive mother, or from a tuberculous animal; the primary seat of disease is then in the alimentary tract and in the absorbent system leading from this. Once started the disease tends to become generalised.

The lungs are less often affected primarily.

In childhood the path of infection may depend upon some local predisposition in individuals with a constitutional tendency. Relaxed throats and tonsillitis offer a nidus for the bacilli in the mucous membrane of the fauces and tonsils, and strumous (tubercular) glands in the neck are more common than abdominal Tuberculosis. Primary pulmonary Tuberculosis, though still less common than in young adults, now begins to occur. The active development of the epiphysal ends of the long bones may account for the arrest of bacilli at these points, and so determine the onset of Tuberculosis at the joints. In later life, when the young adult goes out into the world, he may be exposed to air contaminated by phthisical fellow-workers. The lungs then become the primary seat of the disease, more especially if they are damaged by the inhalation of dusty particles. Whatever be the causes of infection to which an individual may be exposed, his liability to be harmed thereby will depend chiefly upon his constitutional strength or weakness—his resisting power against disease; and this, as we have already mentioned, is often inherited.

CHAPTER II.

THE CAUSATION OF CONSUMPTION.

STANDING almost at the head of the causes of death—as the most fatal disease in this country—Consumption is almost more to be dreaded than was small-pox before the days of vaccination. For Consumption is pre-eminently a disease of young adults; it attacks its victims at an age when they have just commenced a life of usefulness, and when they are just beginning to turn the teaching of youth to account and may expect not only to support themselves, but to repay, to some extent, the care bestowed on their early training. Unlike small-pox and other acute fevers, which though sharp are short in duration, Consumption draws out its painful course for months and years. We need no longer dwell upon this, for the disease is so wide-spread that there are few who cannot recall from personal knowledge some instance of the suffering and distress caused by Consumption. Amongst the well-to-do the prolonged illness of the invalid and the distress of his friends at his suffering are sufficient to leave an indelible impression on those who have seen them; but amongst the poor there is added to all this the heavy tax on the small exchequer in supporting an invalid, made all the more heavy to bear in that the sufferer is the one who was just beginning to be a help to his parents.

No wonder that we should seek to check this disease—

no wonder that, since medical knowledge has as yet found no certain cure for this malady, preventive medicine should welcome any well-established facts which hold out hopes of diminishing its prevalence, if not of preventing it entirely.

One would imagine that it was only necessary to publish the fact that Consumption is now known to depend upon conditions which are controllable, and to notify the measures needed to limit the spread of the disease, in order to have at once the ready co-operation of all. Yet we find that all endeavours to prevent disease meet with opposition from the ignorant and those whose business it is to trade on ignorance, and are received with apathy by those who are not antagonistic. But preventive medicine, strong with the power of knowledge, works against all opposition, and finds its reward in the results which slowly but surely follow its endeavours.

When I read my first paper on the Prevention of Phthisis (before the Epidemiological Society of London) about ten years ago, few in this country believed in the practicability of combatting this disease by sanitary regulations. Now there is hardly a Consumption Hospital in the country which does not issue instructions to its patients informing them how to lessen the risk of distributing their disease, and Medical Officers of Health throughout the kingdom look upon the prevention of Tuberculosis as one of their important duties. Already we can find, in the annual death registers, evidences of a diminution of this disease sufficiently marked to encourage us to further efforts. If we take the number of deaths from Consumption for the past twenty years we find them steadily

decreasing. In 1875, 52,943 persons died from Consumption. In 1885 the number was 48,175, and in 1895 it had fallen to 41,641; and this with an increasing population.

This improvement is even more clearly shown if we take the ratio of deaths from Consumption to the number of the population, which shows a fall from 2,202 per million in 1875 to only about 1,400 per million at present.

It is a very important point to bear in mind that this diminution in the death-rate for Consumption is very largely due to the improvement in the general hygienic and sanitary condition of the people, for the improvement commenced before any special precautions against Consumption could be formulated. May we not expect that, as the knowledge of how Consumption is caused and spread becomes more generally recognised, still more marked diminution of this disease will be apparent?

We cannot, however, attempt to lay down rules for the prevention of any evil until we know something of how that particular evil is brought about. And although some generations ago there were regulations suggested for preventing Consumption—notably in Italy—it is only since Koch discovered that the disease is dependent upon a living germ that systematic and practical prophylactic measures have been possible. Since that important discovery — twenty years ago — numberless workers have investigated the conditions under which the germ can grow and flourish, and from their labours have sprung the preventive rules which are gradually being put into force.

One very important fact has been ascertained, namely,

that Consumption is only one of a number of maladies which all depend upon the same micro-organism, and which are now classed together under the name **Tuberculosis**. Consumption is Tuberculosis of the Lungs; but we meet also with Tuberculosis of Joints, Tubercle of the Intestines (Tabes mesenterica or Consumptive Bowels), Tubercular Meningitis when the disease affects the coverings of the brain, and Strumous or Scrofulous Glands when Tuberculosis attacks those structures.

Thus in the war waged by preventive medicine against Tuberculosis, not only do we hope to diminish the prevalence of Consumption, but to lessen the ravages of all those diseases which depend upon the same germ.

Let us briefly review the present state of our knowledge of the Causation of Consumption. As long as the disease was considered to be dependent upon climatic causes alone there could be no hope of preventing it in a country where damp and variableness are the chief characteristics of the climate. But when the interchange of medical observations between the different parts of the world became more easy, it was soon realised that Consumption was not peculiar to any one country or climate. Further observation showed it to be a disease of thickly-populated countries and crowded cities, and therefore most prevalent in the centres of civilisation.

Over-crowding.

This is well illustrated by some observations made in France, which showed that the prevalence of Consumption in towns increases in proportion to their size, rising from

a death-rate of less than 2 per 1,000 for towns of under 5,000 inhabitants up to 5 per 1,000 in Paris.

The same fact is seen in the difference between the death-rate for Consumption in the extra-Metropolitan portions of the counties of Middlesex, Surrey and Kent (15·75 per 1,000), and that of London, which is 21 per 1,000.

So, too, we note a much higher death-rate from Consumption in the more central portions of London than is found in the outskirts.

Not only was over-crowding found to favour the disease, but amongst the workers in certain trades it was especially prevalent. Even before the true nature of the disease was discovered, something had been done to prevent Consumption by checking over-crowding and by regulating certain dusty occupations. The means taken to prevent the metallic dust from file-making, needle-grinding and similar trades from filling the air of the work-rooms has had a most notable effect in diminishing the incidence of Consumption upon those engaged in these occupations.

But this was not enough. Even before Koch's discovery of the disease germ, several investigators had found that tubercular disease could be communicated from man to animals, and from one animal to another. For many generations there had been a popular feeling that Consumption was "catching"—a view occasionally shared by some physicians, as by Morgagni some two hundred years ago. This idea was now shown to be founded on facts, and is at present accepted by almost all the medical world. We have, then, to deal with Consumption as an

infectious disease, and our efforts must, in great measure, be directed to destroying the infection. We have the analogy of other infectious diseases to guide us in our preventive regulations. We know that, in the case of scarlet fever for example, if we can isolate the sufferer with this fever, and destroy all infectious particles which are given off from his body, we can prevent others from catching the disease. We know also that not everyone who is exposed to the infection of scarlet fever takes the malady—all persons are not equally susceptible, nor is any one person equally susceptible at all times.

Thus in advising as to the prevention of any infectious disease, we require to know how the infection is given off from the sick and how carried to others; and if we can also determine what conditions govern the susceptibility of persons to suffer from infection, we may further guard against the risk of catching the disease by strengthening the resisting power of the individual. So with regard to Consumption. We require to know the nature of the infection, how it is given off from the sick, and how conveyed to others; and, in addition, we must study the conditions which determine susceptibility—in other words, what conditions render our tissues a suitable soil for the development of the infectious particles which may reach them. We have, in short, to study, first, the germ or seed of the disease and the conditions under which it flourishes or the means by which it may be destroyed; and, second, the conditions which favour its development within the body, and which thus produce susceptibility or "predispose" to the disease in the individual.

We shall see that climate cannot be quite left out of

consideration in the causation of Consumption, but, like over-crowding, dusty and sedentary occupations, and certain other conditions, must be considered amongst the predisposing causes—those conditions which render us susceptible to infection.

The Germ.

Let us, then, examine first the micro-organism, which is the essential factor in the production of Tuberculosis.

This is a minute rod-shaped organism or bacillus about one ten-thousandth of an inch in length, which was shown by Koch to be invariably associated with tubercular disease. As it is of the first importance, in view of preventive measures, to be assured that there is no doubt as to the nature of the infection—no mistake about this organism being the essential factor in the causation of the disease—it will be well to examine shortly the proofs of this.

Although the detection of this bacillus in all tubercular lesions furnishes strong presumptive evidence that it has some essential connection with the disease, this alone does not amount to proof. Koch therefore proceeded to grow these micro-organisms outside the body. He prepared a broth which might supply nourishment to the organisms, and placing some of this in a test tube, heated it sufficiently to destroy any living particles which might be present. Then placing a small quantity of tubercular matter in the tube, he found, after a time, that some fungus-like growth had taken place on the broth. Examining this growth it was seen to consist of a mass of the rod-shaped bacilli. Some of these were transplanted on

to broth in a second test tube, and from the growth which appeared in this he inoculated a third tube. There was now no remnant of the original tubercular material—only a "pure culture" of bacilli. Some of this "culture" introduced into the body of a healthy animal, was followed by the development of Tuberculosis. Even when the growth was carried through seven generations outside the body, inoculation of an animal with material from the seventh test tube was able to produce Tuberculosis. Here, then, is proof sufficient that the entrance into the body of the tubercle bacillus will cause the disease. In the body, as in the test tube, the bacilli multiply enormously, so that a person or an animal affected with tubercular disease produces innumerable bacilli which, if they are thrown off from the body and gain entrance into the body of another person or animal, may infect this other with the disease.

Here, then, is the great danger. Every person or animal suffering from tubercular disease is a possible source of danger to others. Seeing how many persons suffer from Tuberculosis, and knowing also that the disease is not uncommon in animals, how is it that any of us escape? There are some peculiarities in the conditions of life of the bacillus which bear on this question. First of all the micro-organism grows and develops slowly—so that if it gains entrance into the healthy living body, there is time for the cells of the body to destroy the bacilli before they have time to multiply sufficiently to cause disease. We know that the living cells of the body can destroy deleterious germs when these are not present in too great numbers; and we probably owe to this fact our escape

from many infections to which we are exposed. If, however, the vitality of our tissues is diminished from any cause, if our cells are not strong enough to destroy the bacilli, then the disease may develop—thus we explain the influence of Predisposition. Then again, the bacillus only thrives on certain soils and in a warm temperature. It is killed by a heat of about the boiling point of water; and though not necessarily killed by cold, it is unable to develop at the ordinary temperature of the air in this country.

Not only so, but it is killed by sunlight. If these culture tubes, of which I have spoken, be exposed to direct sunlight, all growth is stopped within a few hours and the colony is killed. Even if the tube is kept in ordinary daylight—diffused sunlight—the colony is killed in a few days, whereas kept in the dark the growth of the bacilli goes on. Here is a striking exemplification of the health-value of sunlight, and an explanation of the prevalence of Consumption amongst those who live and work in underground rooms.

We have now arrived at the important proposition that there can be no Consumption without the entrance into the body of a special germ which is derived from some other case of tubercular disease, whether in man or in animals. We have now to examine the ways in which this germ may get into the body. In doing this we shall also discuss the ways in which the sick give off the infection and the manner in which the infection is spread. With regard to the latter we may at once state that the two chief dangers are to be found in the expectoration of consumptive patients and the milk of tubercular animals.

There are four ways in which the germ may get into the body, which I propose to discuss.

1. The germ may be implanted in the body at birth.

Consumption is often spoken of as a typically hereditary disease. How often do we hear it said of some one, "Poor fellow, he is sure to become consumptive; you know, it is in the family"! How often do we come across a person living in constant dread of the disease, which has proved fatal to other members of his house! I think it is most important to break down the wide-spread belief in the futility of fighting against heredity in Consumption. The cases in which the germ of Tuberculosis have been found in the body at birth are very few in number, although most careful investigations have been made. Few now will be found to hold the doctrine of direct heredity as a common cause of Consumption.

Once we realise that Consumption depends upon infection and cannot arise *de novo*, we must recognise the fact that heredity only acts as a predisposing cause; and predisposition, as we shall see, may be combatted.

2. The germ may enter the body through a wound or sore on the surface.

This has occurred from a cut caused by a broken spittoon used by a consumptive patient, and by the process of tattooing performed by a phthisical person.

I had under observation a short time ago a patient who developed a large tubercular sore on the leg, where the infection probably occurred through an eczematous patch or sore.

This mode of infection generally leads to a local Tuberculosis only.

3. The infection may enter by the mucous membrane of the digestive tract.

This mode of infection has been abundantly proved, and has been placed beyond all possible doubt by the investigations undertaken for the Royal Commission on Tuberculosis, whose Report has lately been issued. The point of chief importance which is established in this Report is that the milk of cows which are affected with Tuberculosis may contain the infective bacillus, and will produce the disease in animals which are fed on such milk. When we consider the enormous number of young children who suffer from tubercular disease primarily affecting the abdominal glands, and remember that cow's milk forms a chief part of the diet for the first few years of life, we may well ask ourselves whether there may not be some connection between these two facts. Physicians have long recognised that it is undesirable for consumptive mothers to nurse their infants, and since the tubercle bacillus has been detected in the mother's milk the reason for this caution is evident. But with regard to cow's milk it is more difficult to persuade people of the need for care. Tuberculosis is far from uncommon amongst cattle, and is especially liable to affect cattle which are kept in closed sheds, especially if these sheds be over-crowded and insufficiently ventilated. The prevention of Tuberculosis amongst cattle is occupying the attention of the authorities, and in my opinion is of as much importance as the inspection of cattle kept for food, and of meat or milk.

We have seen that the tubercle bacillus is destroyed by heat, and even the milk from a tubercular cow may be safely used as food if it has been boiled. We have then a simple and effectual method of preventing any possible infection of Tuberculosis from milk, by boiling it immediately before use. The flesh of a tubercular animal has been shown to be a possible source of danger, but the danger is small as compared with that from milk, and is reduced to a minimum by cooking.

4. Dealing, however, with Consumption, or Lung Tuberculosis, as we are here, the most important path of infection is through the air passages.

This is probably not the only mode of infection by which the lungs become primarily attacked, but it is one of the most frequent. But, since also every case of Tuberculosis, whatever part of the body is affected, forms a centre of infection from which the germs of Consumption may be given off, we cannot disregard the questions I have just briefly mentioned. The prevention of Consumption cannot be disassociated from the wider subject of the prevention of Tuberculosis in general.

Infection is carried into the respiratory tract by the air, and we must therefore consider how the infectious particles get into the air. We may first lay down the general proposition that the air becomes infected by the germs given off by consumptive persons or animals. The breath of a phthisical patient seems to contain no bacilli, and though it is not free from risk to occupy the same bed with a consumptive, the danger is not from the breath.

One of the most constant and distressing symptoms of

Consumption is the cough, which is generally accompanied with expectoration of matter brought up from the lung. This expectorated matter may easily be seen with the aid of a microscope to be teeming with the special bacilli. Whilst the matter is moist the bacilli are fixed by the moisture and cannot get into the air. But if the matter is allowed to dry it soon crumbles into fine powder, and this fine dust will blow about in the air. The bacilli are not killed by drying, and if they reach a suitable soil for development will soon start into activity. The dust of rooms in which consumptive patients are living may be found to contain these bacilli. I have found them in the dust allowed to settle on a glass slide moistened with glycerine which was placed in one of the wards of the Consumption Hospital to which I am attached. We may almost certainly affirm that these germs get into the air from dried expectorated matter, and that if all expectorated matter is received into a spitting cup containing fluid and then destroyed by burning before it has dried, there would be little risk of polluting the air. But amongst the class from which hospital patients are drawn, the filthy habit of indiscriminate expectorating is unfortunately common. In this way not only the pavements but the floors of their rooms become sources of danger from consumptive persons of this class; and in hospital most careful supervision and the fear of expulsion from the hospital are insufficient to prevent occasional infringement of the stringent rules on this point.

Some recent investigations with dust from a group of twenty buildings which constitute a large sanatorium for consumptives in America are instructive. The dust from all the buildings except one was found to be innocuous.

The exception was a small cottage always reserved for the most advanced cases, and lately occupied by two men, one of whom had been complained of for his habit of spitting on the floor. Thus we see that consumptives may be a grave source of danger to others, but by attention to a detail which entails no great trouble this danger may be avoided.

The pocket handkerchief should not be used for receiving expectoration, for when this dries some of the infective dust may be shaken into the air. Pieces of linen which can be immediately burned, or handkerchiefs of Japanese paper which can be similarly destroyed, should be used when the spitting cup is not available. Such a handkerchief should be held in front of the face during a violent cough, to catch any small particles of phlegm which may be expelled by the force of the cough.

It is probable that the infection of scrofulous glands in the neck reaches these structures through the mucous membrane of the fauces or back of the throat. I have demonstrated the possibility of this mode of infection in animals.

It is doubtful whether infection can pass through healthy mucous membrane; in all probability an abrasion or scratch, or a diseased condition of this membrane (as from inflammation), is necessary to allow entrance.

This leads us to consider predisposition.

Predisposition.

We have seen that the power of the bacillus for harm depends much upon it falling upon soil suitable to its development.

The susceptibility of the individual to suffer from (or to "take") infection has therefore an important influence on the production of Consumption. We know from experimental research that healthy living tissues do not furnish a favourable soil for the growth of micro-organisms, but on the contrary destroy them before they can develop and cause harm. When, however, the vitality of the tissues is impaired, these same germs will develop, multiply and produce disease. Such impairment of vitality may be due to a general delicacy of the individual, or to some special impairment of a single organ, or of some particular portion of the body.

In the course of investigations which I have made on this subject, I was led to the conclusion that general ill-health or debility of the individual is a more potent predisposing cause of Consumption than impairment of the lung tissues in an otherwise healthy person.

The chief conditions which have been shown to influence predisposition to Consumption, are Heredity, Locality and Habitations, Occupation, and previous illnesses.

CHAPTER III.

THE CAUSATION OF CONSUMPTION (*continued*).

STATE medicine fulfils its highest object when it successfully attacks the diseases which produce the largest death-rate; and diminishes, where it cannot prevent, the most prevalent maladies, by controlling their determining causes. A glance at the annual report of the Registrar-General shows the tremendous mortality in this country from diseases which affect the respiratory system; and, so far as the peculiarities of our climate account for the prevalence of these ailments, preventive medicine has no scope for action. But where they can be shown to be influenced by causes which are to any extent under control, it is to the interest of the community to direct attention to the removal or amelioration of these conditions.

Amongst affections of the respiratory system two stand out prominently as most frequent causes of death, namely, Bronchitis and Phthisis. Bronchitis kills most largely at the two extremes of life– in infancy and in old age—and is greatly influenced by climatic conditions. Phthisis, on the other hand, independent of season, claims its victims at that time of life when the individual should be in full vigour, and shows its greatest death-rate between the ages of fifteen and sixty-five, or during the working

period of life. The prevalence and intensity of Phthisis are influenced by many conditions which result from the aggregation of persons in a crowded population, or from the industries which mark an advancing civilisation. A more intimate knowledge, in later years, of its causation allows us to place it in close relation with the zymotic or infectious diseases, in the diminution of which preventive medicine has already so successfully exerted its influence. Already the more careful attention to sanitary and hygienic conditions of life has had an appreciable effect in diminishing the mortality from Phthisis. But such improvement has resulted from measures directed in great part against other diseases—especially those commonly classed as infectious,—or from sanitary regulations adopted on general principles.

The State, which has elaborated regulations to prevent the spread of small-pox or scarlet fever, has not yet made any systematic attack against a disease which annually kills more than a fourth of all the males who die during the working period of life. It is true that the Sanitary Authorities are not unmindful of the fearful mortality from tubercular diseases, and that the importance of diminishing the *preventable* deaths from this disease has not escaped the attention of the medical advisers of the Government. This is evident from the reports of the Medical Officer of the Local Government Board, especially of late years, where most valuable inquiries into the causes and transmission of tubercle, and of the effects of sanitary improvements on the prevalence of Phthisis in certain localities, are recorded. But surely, so wide-spread and fatal a disease requires that regulations should be specially

directed against those conditions which can be shown to increase its prevalence or its mortality, and that such measures should be enforced with a completeness commensurate with the national importance of the subject.

I propose here to bring together some of the facts which prove the dependence of Phthisis on preventable causes, or, at least, on causes under human control, and to indicate the lines on which preventive measures should be conducted.

Since Koch, in 1881, discovered the Bacillus tuberculosis (thus proving the correctness of the views of Chauveau (*a*), Baumgarten (*b*), and others, some of whom had even got so far as to find bacteria and micrococci in tubercular deposits (*c*)), and since the repeated corroboration of his discovery, Phthisis must be looked upon—unless we reject the bacillary theory of tubercle altogether—as an infectious disease, in the sense that it is produced by the reception into the body of a specific particle or micro-organism from without, *i.e.*, not originating *de novo* in the body. That the disease does not affect all who are exposed to the infecting cause results from the necessity of the bacillus finding a suitable habitat in the tissues before it can produce spores. With lowered vitality of the tissues the bacillus more easily finds such favourable conditions. This impaired vitality constitutes in the individual a predisposition to tubercle, and may depend upon hereditary constitution, or an acquired debility either of the whole

(*a*) *Recueil de Méd. Vét.*, 1872.

(*b*) *Berliner Klin. Wochenschrift*, 1880.

(*c*) Zurn, Buhl, Klebs, Schüller, Reinstadler, Toussaint, Aufrecht, Deutschmann.

system or a particular organ. We have therefore to consider the conditions under which the specific poison or bacillus may be generated and be admitted into the body, as well as the causes which predispose the individual to suffer from its attacks; and to inquire how far both of these may be controlled. Although I do not propose to exclude the non-preventable causes in the following review of these conditions, I shall devote a larger amount of consideration to those which seem to be within the controlling power of hygiene and sanitation.

To commence with the microbe.

The Bacillus tuberculosis has three peculiarities which it is most important to bear in mind.

1. It only thrives within a limited range of temperatures, viz., from about 82° to 107° Fahr.
2. It does not form spores in the air.
3. It grows slowly, requiring for its development as many days as the anthrax bacillus requires hours.

In artificial cultivation media the bacillus takes from seven to ten days to develop a colony (*d*), and from fourteen to twenty-one days are required to produce a crop of tubercles (*e*).

These peculiarities in inoculation experiments (*f*) serve to account for the rarity with which healthy persons become infected with Phthisis. For, as the bacillus does not reproduce itself in the air, it must gain entrance into the body, and be retained there for an appreciable time in

(*d*) Koch. (*e*) Salmonsen.

(*f*) Cohnheim, *Die Tuberkulose vom standpunkte der Infectionslehre*, Leipsig, 1880.

a suitable habitat, before it can develop. Even if taken into the body it may be expelled before its development is completed; this probably occurs with all of us daily.

The spores, however, appear to resist destruction much more than the bacillus itself.

There are several modes by which the bacillus may gain an entrance into the body.

1. By a cut or scratch on the skin.

This is a true inoculation. Experiments on animals have proved that the introduction of tubercular matters under the skin or into one of the cavities of the body almost invariably produces tubercle, whilst the introduction of non-tubercular irritating substances has no such effect.

Such a means of infection is necessarily a rare accident. It has occurred through a cut on the finger during the performance of an autopsy on a tubercular patient (*g*); from a cut with a spittoon used by a phthisical patient (*h*); from a bite (*i*); during the Jewish ceremony of circumcision (*j*), and in other ways (*k*). It is seen also in the production of "butchers' tubercles" on the hands, from the handling of tubercular meat (*l*). This generally produces a local Tuberculosis only.

(*g*) Verneuil, *Le Progrès Médical*, Jan. 26th, 1884.
(*h*) Tscherning. See *Med. Record*, Aug. 1886.
(*i*) Schmidt, *Arbeiten aus der Chirurg. Poliklinik*, Leip., 1888.
(*j*) Lindmann, *Deutsch. Med. Woch.*, 1883.
(*k*) Lesser, *Deutsch. Med. Woch.*, No. 29, July 19th, 1888.
(*l*) Lingard, *Evidence before Committee on Pleuropneumonia and Tuberculosis*, 1888, Q. 8025, p. 256.

2. By means of the genito-urinary mucous membrane.

The possibility of this means of infection seems to be shown by the detection of the tubercle bacilli in vaginal discharges of women (*m*), and in the testis and vesiculæ seminales (*n*) of males, as well as amongst active spermatozoa in the semen (*o*). This mode of infection may possibly play some part in cases of transmission of the disease between husband and wife, although the close contact occasioned by occupying the same bed renders the breathing of air expired by the affected person a factor which cannot be overlooked. The occurrence of such infection is well exemplified by two cases, in one of which a tubercular husband infects successively four wives, all previously healthy (*p*), and in the other a phthisical woman infects three husbands in succession (*q*).

3. By the product of conception, and direct hereditary transmission.

The proofs of these modes of infection are not easily obtained. That tubercle may occur in the fœtus has been demonstrated by several observers, both in man (*r*) and amongst animals (*s*). Cases of tubercle in infants from a

(*m*) Cornil and Babès.

(*n*) Jani, *Med. Rec.*, 1887, Aug. 15th, p. 341. See also Landouzy and Martin.

(*o*) *Report on Pleuropneumonia and Tuberculosis*, 1888, p. xx.

(*p*) Herman Weber, *Trans. Clin. Soc.*, vol. vii, p. 144.

(*q*) Zasetzky, *Vratch*, No. 47, 1884, p. 797.

(*r*) Chabrin, Alison, Demme, Scanzoni.

(*s*) Johne, *Wiener Med. Blätter*, No. 15, 1885; Koubasoff, *Comp. Rendu*, 1885, No. 8.

few days old are more frequently recorded (*t*), but, if the child has breathed, still more so if it has been suckled, other sources of infection become possible.

Landouzy and Martin found that animals inoculated with the blood or portions of the internal organs of a fœtus, in which no appearance of tubercle was detected although the mother died of Phthisis, became affected with general Tuberculosis (*u*).

4. By the mucous membrane of the alimentary canal.

Numerous experiments have been made which prove the possibility of tubercle being communicated to animals when tubercular matters are mixed with their food (*x*). As Tuberculosis is common in cattle, and affects pigs, rabbits, and fowls, and probably sheep and goats, it becomes an important question as to how far Tuberculosis may be communicated to man by means of milk or flesh used as food. Communication by the milk of tubercular cows has been experimentally effected in animals (*y*); and whether this is only possible when the udders are affected, or may occur wherever the seat of the tubercle may be, an additional reason is here found for the inspection of the cows kept for dairy purposes. In the *British Medical Journal* of Jan. 5, 1889, a case is mentioned in which some pigs fed on the milk of tubercular cows became

(*t*) Epstein, Thomson, Landouzy and Martin, Angel Money, Huguenin.

(*u*) L. Landouzy and H. Martin, *Rev. de Méd.*, 1883, iii.

(*x*) Villemin, Chauveau, Tappeiner, Klein and Heneage Gibbes, Gerlach.

(*y*) Bollinger, Klebs, Foot, Leube, Kommereil and Lochmann, Chauveau, H. Martin.

affected with the disease. Galtier (*z*) concluded that cheese-whey prepared from tubercular milk might be infective. Cows more readily become tubercular when kept in close stalls and over-crowded, and over-lactation is a predisposing cause (*a*). The milk of tubercular cows should probably never be allowed to be sold, and tubercular meat might with advantage be seized and destroyed. The infective power of milk is destroyed by boiling (*b*), but cooking meat may not render it innocuous, as the centre of a joint may not get sufficiently heated (*c*). Opinions differ at present as to whether or not human and bovine Tuberculosis are identical. Minute differences are described between the bacilli found in man and in cattle (*d*), and some animals appear not to be equally susceptible to inoculation with human and with bovine tubercular matters (*e*). But the weight of evidence at present points to the conclusion that Tuberculosis should be included amongst the infectious diseases in the Contagious Diseases (Animals) Act. The late Congress on Tuberculosis, in Paris, expressed a strong opinion on the subject in their conclusions, drawn up at the close of the meeting (*f*).

(*z*) *Comp. Rendu*, t. civ.

(*a*) Lafosse, *Traité de Pathologie Vét.*, vol. i, p. 432. Also Flemming and Jaccoud.

(*b*) H. Martin, *Rev. de Méd.*, 1884, p. 151.

(*c*) Gerlach; *Report on Pleuropneumonia and Tuberculosis*, 1888, p. xxii; and Lamallerée.

(*d*) Klein, *Rep. of Med. Officer to Local Govt. Board*, 1883-4.

(*e*) Klein, Appendix B., No. 2, *Rep. of Med. Officer to Local Govt. Board*, vol. xv, 1886, p. 149.

(*f*) See *Revue d'Hygiène*, Aug. 20th, 1888, p. 723. For Discussion, see *Gaz. des Hôp.*, 1888, lxi, Nos. 87, 88.

5. By means of the mucous membrane of the respiratory tract, and by the air-cells of the lungs.

This is probably the most frequent mode of infection. Experimental proofs of the efficacy of this mode of infection are numerous (*g*), and it is found that the inhalation of tubercular particles has more power to produce the disease than their ingestion (*h*).

The tubercle bacillus has been found in the air of rooms in which phthisical patients are living (*i*), and the dust from such rooms has been proved to be infective (*k*).

The above being the modes by which it is possible for the infecting bacillus to gain an entrance into the body, let us see what reasons there are for believing that Phthisis may be communicated from person to person.

The contagiousness of Phthisis was a matter of popular belief long before experiment proved this point. Many of the older physicians, including Morgagni (*l*), believed in it; and Villemin (*m*) and Budd (*n*) give reasons for this belief. Evidence of its introduction into previously healthy localities is furnished by the immunity of savage races before their contact with Europeans; and facts are

(*g*) Giboux, Schottelius, Corning, Tappeiner, Klein.
(*h*) Tappeiner, Klein.
(*i*) Williams, *Lancet*, March, 1883; Ransome, *Proc. Roy. Soc.*, 1882.
(*k*) Cornet, *Internat. Klinische Rundschau.*
(*l*) Flemming, *Brit. and For. Med.-Chir. Rev.*, Oct. 1874, p. 461.
(*m*) *Etudes sur la Tuberculose*, Paris, 1865.
(*n*) *Lancet*, vol. ii, 1867, p. 452.

recorded by Webb (*o*), Flindt (*p*), and Alison (*q*) in support of this view. The latter found that Phthisis introduced into previously healthy districts by an affected individual spreads to those in close contact with the invalid, and, having attacked the susceptible, disappears in that locality until reimported.

As the intensity of all infection is necessarily increased by close contact and the crowding together of sick and healthy, the necessity for attention to ventilation and the prevention of over-crowding is apparent, and becomes more so when the conditions which produce a predisposition are considered.

Predisposition.

Next, as to the conditions which favour the development of the bacillus.

In the air of towns and all places where tubercular persons or animals are congregated there must be large numbers of tubercle bacilli. We must, therefore, continually take in these microbes with each inspiration, and yet only a certain proportion of individuals become affected with Phthisis. In health it is probable that the expiratory act, aided by the ciliary action of the bronchial mucous membrane, may expel the bacilli before they have been able to develop; or that, having escaped expulsion, they do not find the tissues in a condition to afford a suitable *nidus*. But should the expiratory effort be deficient, or

(*o*) *The Contagiousness of Phthisis*, 1885.

(*p*) *Vide* Jaccoud, *Curabilité et Traitement de la Phthisie Pulmonaire*, Lubbock's translation, p. 76.

(*q*) *Archives de Méd.*, Sept. 1885.

the vitality of the tissues be impaired, the microbes may not only effect an entrance into the lungs, but easily find there favourable conditions for their development. Both these effects may result from disease of the respiratory organs, or from constitutional enfeeblement; and we may therefore affirm generally that a predisposition to Phthisis is produced by diseases of the respiratory apparatus, and by any causes which depress the health—such as insufficiency of food, air, and exercise; fatigue, mental depression, dissipation, and exhausting illness.

We will now examine the more important predisposing causes in detail.

1. Hereditary Predisposition.—Apart from the possible direct transmission of tubercle from parent to offspring, mentioned previously (pp. 14, 25), the children of unhealthy parents, such as those affected with Phthisis, are often of weak constitution, and thus inherit a general predisposition to tubercle. Increased care is then requisite, especially during the time of growth and development, not only to remove or diminish this susceptibility by physical training, but to keep them from surroundings and occupations which in themselves produce or increase the predisposition. To prevent the marriage of phthisical persons may be theoretically desirable, but is far removed rom the range of practical hygiene.

2. Influence of the Air and Sun.—Sunlight and fresh air are essential to healthy life, and to ensure an adequate supply of both is the basis of all hygiene. As it is in

dwellings and workshops that deficiencies of both are most commonly met with, the influence of this cause may be remembered in discussing habitations and occupations.

3. Age and Sex.—Pulmonary Phthisis is essentially a disease of adolescence and adult life. Though tubercle attacks children from infancy, it is then more generalised, and affects other parts—such as the intestinal tract, the glands, and the membranes of the brain—with greater frequency than it affects the lungs. Even if tubercular Meningitis may be a result of direct inheritance, the affection of the mesenteric glands suggests infection through the alimentary canal. In adults the lungs are most commonly the starting-point of the infection, which may spread later to other organs.

The two sexes seem to be affected by pulmonary Phthisis in almost equal proportions, but the disease appears to commence at an earlier age and to run a more rapid course in females. Exhausting discharges—such as menorrhagia, debility from rapid child-bearing, and over-lactation, produce in women a predisposition which must not be overlooked.

4. Occupation.—The influence of occupation as a predisposing cause of Phthisis has long been recognised, and is of the utmost importance, not only because it operates so widely, but because it is one of the causes which it may be found possible to control and diminish. The large incidence of Phthisis upon those who follow dusty employments has attracted much attention from observers. Dr.

Greenhow (*r*) calculated that forty-five thousand deaths occurred annually from this cause in England and Wales, and he believed that the whole of this mortality was preventable by the introduction of better methods of ventilation and working. Dr. Pollock (*s*) also gives good evidence of the predisposing influence of certain indoor and dusty occupations. In these cases the susceptibility to Phthisis evidently arises from the inhalation of fine, irritating particles into the lungs, and the consequent impairment of these organs, rendering them more susceptible to the invasion of the bacillus, which readily finds a suitable *nidus* in the damaged tissue. Amongst dusty occupations, stone-masons, coal-miners, knife-grinders and needle-polishers, chaff-cutters, and those employed in hackling flax, carding cotton, grinding steel, porcelain and pearl for buttons, mattress-making and cleaning, &c., afford instances of the effect of dust in producing a large mortality amongst those who follow these callings. Amongst the potters in Staffordshire, Phthisis caused 18·1 per cent. of all deaths for males, and 11·5 per cent. for females (*t*); and the effect of the occupation is well shown by comparing the proportion of cases of Phthisis amongst the potters who were admitted to the North Staffordshire Infirmary with that amongst other classes.

The potters showed 20·9 per cent. for males and 16·96

(*r*) E. Headlam Greenhow, M.D., *On the Prevalence of Certain Diseases in different Districts in England and Wales.* Papers relating to the Sanitary State of England to the General Board of Health, 1858.

(*s*) *The Elements of Prognosis in Consumption*, 1865.

(*t*) *Report on the Staffordshire Potteries to the Med. Dept. of the Local Govt. Board*, 1872, Appendix B., p. 44.

for females suffering from Phthisis, whilst the males who were not potters gave a proportion of 13 per cent., and the females 11 per cent. (*u*). In cotton factories, few men who enter certain rooms ever live to attain thirty-eight years of age (*x*). Dr. Pollock (*y*) suggests that a veil should be worn over the nostrils and mouth, and that the men should grow beards. But he adds: "A perfect ventilation and extreme cleanliness are, however, the best modes of prevention. The metallic dust in mines, the floating particles of flax, cotton, and straw, are all possible to remove, rather let us say impossible to continue, in the presence of a good system of ventilation." This strikes the key-note of all regulations for the prevention of Phthisis. In certain well-ventilated mines the mortality is one-third less than in others less well-managed, and the simple use of the fan in workshops materially lessened the quantity of disease among the grinders of Sheffield (*z*).

Apart from the irritating effects of dust, the influence of occupation is found in the large amount of Phthisis amongst those who follow indoor and sedentary occupations of whatever kind. Here we have to take into account insufficient exercise and the want of fresh air, not only from the large amount of time spent indoors, but from the frequent over-crowding of work-rooms.

Amongst tailors, Dr. Edward Smith (*a*) found that

(*u*) Dr. Arlidge, *Social Science Congress*, Leeds, 1871.
(*x*) Pollock, *The Elements of Prognosis in Consumption*, 1865.
(*y*) *Op. cit.*, p. 370.
(*z*) Pollock, *loc. cit.*
(*a*) *Report of Medical Officer to Local Govt. Board*, vol. vi, 1863, p. 429.

Consumption and other forms of chest disease constitute two-thirds of all the causes of death; but this computation would appear to be too high, for in the ten years from 1871 to 1880 the mortality from Phthisis and diseases of respiratory and of the circulating system amongst tailors does not constitute two-thirds of their total mortality (*b*). The proportion is more nearly one-third. Dr. Ord (*c*) noticed the prevalence of Phthisis amongst dressmakers and milliners, and attributed it to the long hours, want of exercise, and deficient ventilation of work and bed-rooms. Although the Factory Act allows of the inspection of work-rooms where numerous persons are employed (*d*), over-crowding is probably still rather the rule than the exception. Each work-room should be licensed to hold a certain number of persons for so many hours a day, the number being determined in each case not only by the cubical contents of the room, but by taking into consideration the means of ventilation; and some penalty should be enforced if the number of workers is exceeded. But perhaps the most serious class of cases, and certainly the most difficult to bring under control, is that large body of persons who take the work to their own homes. Amongst the home-workers it is not uncommon to find a single room serving as living-room for a whole family, and work-room for the older members. A typical example from my

(*b*) *Supplement to the Forty-Fifth Annual Report of the Registrar-General*, 1871-80, p. xxxix.

(*c*) *Report of Med. Officer to Local Govt. Board*, vol. vi, 1863, p. 362, Appendix 10.

(*d*) See Factory Acts, 1864-67; Work-places Regulation Act of 1867; and section 19 of Sanitary Act of 1867.

own observation is the following:—In a room, half of which seems to be taken up by a bedstead, on which lies a woman dying of Consumption, the husband is at work before a fire (although it is summer) pressing a coat, the steam from which, as he presses the hot iron over the damp cloth, renders the close air humid; beside him his son sits stitching at some other garment, and near the bed is the eldest daughter with her sewing-machine, probably waistcoat-making, whilst two or three younger children play about on the floor; the single window is shut and cannot be opened, either because the frame is too rotten, or because from long disuse it has stuck too tight to move. Where could anyone imagine a better field for the operations of the tubercle bacillus?

Mr. Lakeman, Inspector under the Factory Act for the northern district of the metropolis, in his evidence before the Select Committee of the House of Lords in 1889 to inquire into the sweating system, is reported to have suggested that all places in which work was done should be registered; even if a father, mother, and two or three daughters were working in their own cottage in the country for someone else, he would have the cottage registered. Such a plan, however desirable for some reasons, would tend to strike at the privacy of home-life, and could not be tolerated. But the evil might be diminished—in the same way that public wash-houses have removed the necessity of filling a room with freshly washed linen hung up to dry—by the establishment by local authorities of public work-rooms, where for a small sum a man might take his work and find conveniences and appliances for his occupation, besides companionship. A woman's room

would probably attract a large number of poor seamstresses and waistcoat-makers, especially if a *crêche* in the neighbourhood relieved them of the care of the younger children whilst the others were at school. Then there might be some pleasure in the family reunion in a fresh well-ventilated room at home after the day's work was over, and the head of the family would have less excuse for spending the evening at the public-house.

The influence of dust is added to the want of ventilation in the predisposition to Phthisis which is so marked amongst printers. Dr. Edward Smith (*e*) found that amongst the printers of London, Phthisis, in proportion to other diseases, is twice as prevalent as even among the general male population of London, and furnishes 46 per cent. of all the causes of death in the men who follow this employment. He gives some suggestions for remedying the unhealthiness of the printer's occupation. The want of ventilation is most marked where unsuitable buildings have been adapted to suit the trade, and less where the premises have been specially built for the business. In the compositors' rooms, where the window is the chief provision for the entrance of air, a difficulty arises from the type-frames being placed immediately under these, so as to be in a good light. The men then find the draught too great when the window is open, and therefore keep it closed. Such rooms should be provided with some ventilating inlet which diffuses the incoming current of air (*e.g.*, Harding's), and so prevent draughts.

Amongst unhealthy occupations we may also include

(*e*) *Report of Med. Officer to the Local Govt. Board*, vol. vi, 1863.

that of bakers, who suffer from the effects of inhaling flour-dust, of bad hours, and of exposure to extremes of temperature; and carpenters who inhale wood-dust. Actors, also, and persons employed behind the scenes in theatres, suffer from dust, bad hours, and draughts. Innkeepers were found by Alison (*f*) to present a high proportion of Phthisis cases, which he attributed partly to alcoholic excess; and billiard-markers suffer from long hours in a bad atmosphere in addition.

With regard to indoor occupations generally, it is found that "in proportion as the male and female populations are severally attracted to indoor branches of industry, in such proportion, other things being equal, their respective death-rates by lung-disease (Phthisis) is increased" (*g*).

Preventive measures against Phthisis should commence with an efficient inspection of factories, workshops, and mines; over-crowding should be prevented, and ventilation enforced. Power is now given to enforce ventilation in work-places (*h*), but the importance of the subject to the health of the community requires that such power should be far more used than it is at present. If anything were wanting to emphasize the influence of indoor occupations on the amount of Consumption, it might be found by contrasting the comparative immunity from this disease enjoyed by nomadic races or by the ranche men of Australia. Even log-rolling in Canada has been effectual in removing a tendency to pulmonary Phthisis.

(*f*) *Op. cit.*

(*g*) *Third Report of Med. Officer to the Privy Council*, 1860, p. 30.

(*h*) *Fourth Report of Med. Officer to the Privy Council*, 1861, p. 29.

5. Locality and Habitation.—Under this heading we may include the influence of localities with regard to climate and temperature, altitude, nature of the soil, and density of the population; and finally, the effect of insanitary conditions in dwelling-houses.

(a) *Locality and Climate.*—Popular opinion is still too apt to consider Phthisis as depending upon the nature of the climate, and therefore to look upon a high death-rate from Consumption as an inevitable necessity in this country. But tubercular Phthisis is found in almost every variety of climate, from the heat of India to the cold of Canada. The variableness of our climate exerts some influence by producing frequent catarrh and inflammation of the respiratory organs, and thus predisposing them to receive and retain the infective bacilli, and in this way cannot be disregarded. But I believe the influence of climate is infinitesimal when compared with the effect of the density of population in this country.

(b) *Altitude.*—There seems to be no doubt that in elevated regions—especially if over 5,000 feet above the sea-level—the tendency to Phthisis is diminished. This is probably due not only to the purity of the mountain air, but also to the diminution of atmospheric pressure, which allows greater chest-expansion. But, unless hygienic conditions are attended to, the good effects of altitude may be counterbalanced. In the factories of the Jura mountains, with a large working population, at an altitude of 3,500 feet, Tuberculosis is common (*i*).

(*i*) Jacobi, *Archiv. de Pediatr.*, vol. v, No. 10.

(c) *Nature of the Soil.*—Buchanan's observations on the influence of moisture of the soil upon the prevalence of Phthisis are well known. He noticed that the drying of the ground by the drainage works of certain towns was followed by a marked decrease of Phthisis. The improvement noticed in fifteen towns, was from 49 per cent. in Salisbury, and 47 per cent. in Ely, to 11 per cent. at Merthyr (*k*). Following up these observations, a further inquiry was instituted into the dependence of Phthisis upon the dryness of the soil, and he was able to show that, in a number of districts, the prevalence of Phthisis had a marked relation to the proportion of inhabitants who lived on pervious or on retentive soil. In the group of districts where Phthisis was least prevalent, 909 per thousand of the inhabitants lived on pervious soil, and only 91 per thousand on retentive soil, and in the most phthisical districts the proportion was 642 on pervious, and 358 on retentive soils in every thousand inhabitants (*l*).

A similar dependence on the dampness of the soil was noticed by Bowditch (*m*) of Boston, U.S., in 1862, and these observations were afterwards confirmed by the Registrar-General for Scotland (*n*).

Here again we have a predisposing cause of Consumption, which can be remedied by drainage and should not be neglected.

(d) *Density of Population.*—The prevalence of Phthisis

(*k*) *Report of Med. Officer to the Privy Council*, vol. ix, 1866.

(*l*) *Report of Med. Officer to the Privy Council*, vol. x, 1867.

(*m*) *Consumption in New England and elsewhere; or, Soil-Moisture one of its Chief Causes*, Boston, 1868.

(*n*) *Seventh Annual Report of Registrar-General for Scotland.*

wherever large numbers of persons are congregated together, as in towns, is partly due to the increased opportunities for direct infection, as well as to the predisposing effect of the unhygienic accompaniments of town-life. Dr. Farr (*o*) shows that the increase in deaths from Consumption in cities over those in country districts is as much as 39 per cent., and also that for every person who dies of Consumption in the counties, 1·24 die in the cities. He says: "The tendency to Consumption was increased 24 per cent. to typhus 55 per cent. in the town districts; but as the absolute mortality from Consumption is three times as great as from typhus in towns, and nearly four times (3·73) as great in the country, the excess of deaths from Consumption caused by the insalubrity of towns is greater than the excess of deaths by typhus" (*p*).

The effect of town-life is also shown by the fact that, whereas the death-rate for Phthisis for London was 1,858 for every million persons living (*q*), that for the extra-metropolitan portions of the counties of Middlesex, Surrey, and Kent was only 1,393 per million persons living—or only three-quarters of the London death-rate.

(e) *Dwelling-houses.*—The injurious effects of inattention to hygienic details in dwelling-houses may depend upon faulty construction or on over-crowding. Dampness may be prevented by a concrete foundation, and by the insertion of a damp-proof course in the brick-work, just above the level of the ground. But what can be said for the under-

(*o*) *Vital Statistics*, 1885, p. 167.
(*p*) *Ibid.*, p. 172.
(*q*) *Registrar-General's Report*, vol. xvii, 1887.

ground rooms in faultily constructed houses in which whole families live in our towns? Healthiness, under such circumstances, is too much to expect. Although it may be too late to remedy essential structural defects in existing houses, precautions to exclude damp ought to be insisted upon in all newly constructed houses. So, too, efficient means of ventilation—whether by windows or otherwise—should be insisted upon by inspectors of new buildings before they are certified as fit for habitation. We have, fortunately, now no window-tax, which unintentionally placed a premium on light and fresh air in dwellings; the minimum area of windows to each room should be regulated by law, and some such regulations as are suggested in the Model Bye-laws of the Local Government Board (*r*) should be in force for the whole country. So, too, the minimum height of rooms should be regulated according to their floor-area.

But no provisions of this kind will ensure efficient ventilation where houses are crowded together without sufficient open space round them. Dr. Ransome (*s*), in a paper read before the Epidemiological Society in 1886, has collected some valuable evidence, showing the marked prevalence and spread of Phthisis where houses are densely crowded together. In London, although an open space is required in rear of each house, this may be built over to the height of the ceiling of the ground floor; and even this small provision for thorough ventilation only applies to houses built upon land not occupied before 1882. Where houses are

(*r*) Knight's *Annotated Model Bye-laws of the Local Govt. Board*, 1885. IV. New Streets and Buildings, Bye-laws 57, 58, p. 112.

(*s*) *Trans. Epidemiological Soc.*, N. S., vol. v, 1886-7, p. 124.

built back-to-back, through-ventilation is impossible, and the progressive Phthisis death-rate in direct ratio with an increased percentage of houses unfurnished with means of through-ventilation is very suggestive (*t*). If back-to-back houses must be permitted at all, they should be built in blocks of four houses, with an open space around each block, and windows on all four sides.

The regulation of the width of streets is also of importance. No court or street under a certain breadth should be allowed to be blocked at either end: there should always be an opening at each end of a narrow street or court, one, at least, being of the full width of the court and open from the ground upwards; the height of the houses should be regulated by the width of the street or court, and the length of narrow streets should be limited. Such regulations are contained in the Local Government Board's Model Bye-laws; and if section 157 of the Public Health Act of 1875 were made compulsory, instead of permissive, they could easily be enforced throughout the country.

But, even if we get houses properly built and healthily situated, we cannot expect diminution of Phthisis unless the number of inhabitants is regulated to the size of the dwelling. Over-crowding in dwelling-houses is as potent a cause in breeding Consumption as the insanitary condition of the houses. Nothing could demonstrate this more forcibly than the experience of the army. In 1839 the average mortality throughout the army from diseases of the lungs was about eight per thousand annually, of

(*t*) *Report on Back-to-Back Houses, Local Govt. Board*, 1888, p. 41.

which four-fifths arose from Consumption, whereas the highest estimates in civil life were only half as much. Amongst the troops, the highest death-rate from these causes was in the Foot Guards, where it was 13·8 per thousand. That the Guards were not more susceptible to diseases of the lungs than the other troops, was shown by the fact that when they were serving in Canada they were less affected by these diseases than the infantry of the line who were in that country, whilst the infantry of the line who took over the barracks in London which were vacated by the Guards, showed a greater amount of sickness than the usual amount for the same period amongst the Guards under similar conditions. This at once points to some local causes, which were thus summarised (*u*) :—Defective barrack accommodation ; the great amount of night duty ; the deteriorating influence of residence in a large town ; the greater facilities for, and temptation to, dissipation.

It is a curious commentary on the slowness with which matters relating to health receive official attention, to find the report of a Commission to inquire into the causes of this excessive mortality (*x*) dated nearly twenty years later than the report from which the above facts are taken. But when the barrack accommodation was improved, although the other conditions mentioned above remained, the mortality from chest and tubercular diseases amongst the troops at home stations fell from 10·1 to 4·2 per

(*u*) *Statistical Reports on the Sickness, Mortality, and Invaliding among the Troops in the United Kingdom, the Mediterranean, and British America*, 1839.

(*x*) *Report of the Commission to Inquire into the Excessive Mortality in the Army*, 1858.

thousand (*y*). Attention to ventilation and over-crowding in prisons also has considerably diminished the death-rate from Phthisis in those institutions.

Over-crowding is still a marked characteristic of the habitations of the poor (*z*), and its evil effects are increased by the dread of allowing air to enter their rooms direct from outside. Thus ventilation is unknown and dirt is rampant. Some improvement could be effected by enforcing throughout the country the bye-laws as to houses let in lodgings (*a*) (Public Health Act, 1875); by placing tenement-houses under the regulations drawn up for common lodging-houses (*b*); or by some such measures as are in force in Glasgow for the "ticketed houses" in that city. But only personal influence can prevent the over-crowding which is to be found in the houses of the better classes, where servants are made to sleep in rooms little better than cupboards, or in a cellar under the front door-steps.

6. Influence of certain Diseases—Local and General.—Several maladies produce a predisposition to Tuberculosis by their local impairment of vitality of the lungs—such as bronchitis, pneumonia, pleurisy, and repeated catarrhs; others by their general lowering of the resisting power of

(*y*) *Our Homes, and How to make them Healthy*, 1883, p. 502. See also Lawson, "Phthisis in the Army," *Journ. of Statistical Society*, Jan. 1887.

(*z*) Vide *Report of Royal Commission on the Housing of the Working Classes*, vol. i, p. 7.

(*a*) Public Health Act, 1875, sect. 90.

(*b*) *Idem*, sect. 80.

the tissues—such as typhoid fever and diabetes; and insanity may be placed amongst these predisposing maladies. Measles and whooping-cough possibly act both generally and locally. Every severe epidemic of measles in London serves, in all probability, as the starting point of numerous cases of Tuberculosis, which might have been prevented if means had been taken to stop the spread of the epidemic by the timely closing of schools. Deformities of the chest, such as occur in rickets, act locally by diminishing expiratory power. Alcoholism seems to act as a predisposing cause by its general debilitating effect; and in considering the large incidence of Phthisis on tailors and printers, the tendency to heavy drinking in these trades must not be overlooked. Injury to the lung may also be the starting point of Phthisis. Thus, successful sanitary measures directed against other diseases may affect the amount of Phthisis; and philanthropists can give material assistance in diminishing the prevalence of this disease by their fight against dirt, drink, and dissipation.

It appears, then, that many of the conditions on which Phthisis depends come well within the reach of preventive medicine. The fundamental principles which must form the basis of any successful attempts to diminish the prevalence of this disease are—(1) to provide a sufficiency of fresh air in and around dwellings and work-places, and (2) to endeavour to improve the resisting power of the individual by hygienic and physical training during the period of growth and development, and, in later life, by exercise and recreation to relieve the strain of continuous application in the struggle for existence. Gymnastics and

games form an important part of the details of school-life, as well as mental training; and work is better done at all times if healthy relaxation alternates with labour. Anything which provides for healthy recreation for the working-classes after their day's work is over must tend to improve their physical condition, and consequently their resisting power against disease.

We owe it to succeeding generations, as well as to those of our own day, to allow no time to be lost in commencing a systematic attack against a disease which annually destroys so many useful lives, and which is handed down in so many families as a fatal inheritance.

If any apology is needed for such a paper as this, it may be found in the words of Sir John Simon—that the mortality of Phthisis ought to be very jealously criticised, for "the tendency to tubercular disease is one which transmits itself from parent to child; and thus, if in any one generation the disease be artificially engendered or increased, that misfortune does not confine its consequences to the generation which first suffers them. Whatever tends to increase tubercular disease among the adult members of a population must be regarded as assuredly tending to produce a progressive degeneration of race" (*c*).

(*c*) *Introductory Report of Med. Officer to the General Board of Health.* Papers relating to the Sanitary State of the People of England, 1858, p. xxii.

CHAPTER IV.

PULMONARY AFFECTIONS WHICH MAY LEAD TO PHTHISIS.

By the term "Phthisis" I wish to be understood to refer to pulmonary Tuberculosis.

There are of course other conditions besides tubercular disease which may lead to consolidation of the lung, destruction of tissue, and cavities, thus giving physical signs similar to those found in tubercular Phthisis. But the presence of the tubercle bacillus as the exciting cause of the lung mischief marks the tubercular disease with such important characteristics that it should be separated clearly and distinctly from other lung affections.

It is the fact of Phthisis being an *infective* inflammatory disease which constitutes its chief danger to the patient, and which suggests precautions on the patient's part for the benefit of others. The limitation of the disease is rendered difficult, because the micro-organisms may wander from the centre of the mischief and be carried by the lymphatics, and perhaps also by the blood stream, to distant parts, and there induce fresh inflammatory changes which in turn become new foci of infection.

As, then, the presence of the bacilli constitutes the essential factor in Phthisis, and as spontaneous generation of micro-organisms is not proved to be possible, no disease or injury of the lung will produce Phthisis without tuber-

cular infection; and the most seriously crippled lung will not become the seat of Phthisis if the invalid is in a place where tubercle is entirely absent. In this country, and in most places where human beings congregate, the infective bacillus is constantly present; and especially so in the over-crowded dwellings of the poorer classes, which furnish so large a proportion of our hospital patients.

Though healthy organs offer no suitable nidus for the development of such bacilli as may reach them from without, these become a real source of danger when they attack an organ weakened or damaged by disease. The spores of the tubercle bacillus, carried about in the air, more easily reach the lungs when the respiratory movements—especially expiration—are deficient, and are more readily retained when the epithelium of the air-passages is damaged, or when sticky mucus coats the walls of the tubes or plugs the air-cells; and being retained under such circumstances, they find a suitable soil for development in the morbid products in which they rest.

At first sight it would seem as though all pulmonary diseases would be likely to lead to Phthisis, but we find a great difference between them in this respect; some, like pleurisy, being frequent antecedents of Phthisis, others, such as general emphysema, being only infrequently associated with Tuberculosis.

We may expect to find that infection from tubercle is favoured by diseases of the respiratory system which interfere with the movements of respiration, either as a whole or in some portion of the chest, or which lead to inflammatory exudation in the air-passages or in the air-cells; and more especially by those affections which are

chronic in their course, or which leave behind them a more or less permanently damaged condition of the tissues.

We may thus make a rough classification of those conditions of the respiratory system which directly predispose to pulmonary Phthisis. First, those which act by interfering with respiratory movement; secondly, changes occurring in the tissues, by which a suitable nidus for the bacillus is formed.

Some morbid conditions interfere with the respiratory movements as a whole, and although morbid changes in the chest walls are not pulmonary diseases, they cannot be ignored as direct causes of the latter. It is sufficient here to notice the important influence on the respiratory movements of such conditions as deformities of the chest—as from rickets and spinal curvature—ossification of the costal cartilages, and paralysis of the muscles used in respiration.

But respiratory movements are also affected by alterations in the lungs themselves, such as emphysema and fibrosis of the lung; whilst pleural adhesions and collections of fluid or of air within the pleural cavity are classed with pulmonary affections. Some of these conditions limit the movements of portions of the lung, and may thus determine the *seat* of the primary deposit of tubercle.

It would seem, from my observation of cases, as though causes which lead to unequal expansion of different parts of the lung had more effect in favouring Phthisis than those which affect the movements of all parts of the lung equally. This may be noted in deformities of the chest, as well as in emphysema, pleurisy, &c.

Finally, as causes of Phthisis, we have diseases of the

air-passages and lungs producing local changes which may favour the development of the tubercle bacillus, such as bronchitis, pneumonia, and pleurisy.

Though perhaps not the most important as causes of Phthisis, we may first consider interference with respiratory movements. It is impossible entirely to separate interference with respiratory movements from tissue changes in the lungs as predisposing causes of Phthisis, for in most of the affections we are now considering both these causes are combined.

Fibrosis of the Lung.—This occurs frequently as a result of chronic tubercle in the lung, but it is found under many other conditions. Thus it may be part of a generalised fibrosis of many organs of the body, either resulting from the degeneration of advancing age, or being induced by some special condition such as chronic alcoholism or syphilis. It may also occur in the lung alone from the local irritation of inhaled particles—as in miners and stone masons, and in others engaged in dusty occupations.

Fibroid overgrowth in the lung, therefore, often exists apart from Tuberculosis; and though it cripples the lung and gives rise to dyspnœa and cough, and may even produce cavities in the lung (chiefly by dilatation of the bronchi from the contraction of the fibrous tissue), it is not then properly called "Phthisis." But it is a common clinical experience that persons with fibroid lungs are the subjects of Tuberculosis; and we may consider that a fibroid lung is particularly prone to become tuberculous when the person is exposed to tubercular infection. This is largely due to the loss of elasticity of the lungs, which

prevents their rebound after distension, with a consequent impairment of expiratory force.

We may generally say, when we recognise the physical signs of fibrosis of the lung, that if the patient is not already tuberculous he will speedily become so. Fortunately the spread of Tuberculosis in a fibroid lung is slow, and breaking down of tissue with consequent excavations is not the rule; so that the *chronic* nature of the fibroid lung affection is not altered by the addition of the tubercle, and the detection of bacilli in the expectoration is of less prognostic importance than in some of the more acute diseases.

It is difficult to select cases in illustration of tubercle following fibroid disease; for fibrosis, both before and after tuberculisation, runs a very chronic course, and it is impossible to say exactly when tuberculisation first occurs. Fibroid disease in its early stages produces but slight symptoms, and patients rarely seek advice until the fibrosis in the lung has made considerable progress. Moreover, a negative result of examination of the sputa for bacilli is no proof that there is no tubercle.

Emphysema.—When general throughout the whole of the lung, emphysema has a similar *mechanical* effect on respiration to fibrosis, with which it is frequently associated. We might, therefore, expect that general pulmonary emphysema would be a favourable condition for the development of tubercle, especially as, with the dilated alveoli, there is a general diminished vascularity of the lung. Yet we know that Phthisis following generalised (lobar) emphysema is uncommon; though it does

occasionally occur, and this condition is not preventive against Phthisis as has been supposed.

Localised emphysema is very commonly found co-existent with tubercular deposit, but here it is usually a result of the Phthisis—*e.g.*, from coughing—rather than a cause.

Pleurisy.—We must not forget that many pleurisies are themselves tubercular in origin—a result, not a cause, of Tuberculosis. Pleurisy in a delicate person is always a matter of anxiety, and if repeated, or when slow in resolving, should suggest tubercle. In many of my cases of Phthisis in which pleurisy is mentioned as an antecedent illness I am disposed to think that tubercle was present in the lungs before the date of the pleurisy, and was the exciting cause of the latter. That pleurisy is, however, a not infrequent antecedent of Phthisis becomes abundantly evident in looking through a series of hospital reports. Pleurisy may act in several different ways in predisposing to Phthisis. The lung may be compressed and the movements of the chest interfered with by fluid in the pleural cavity, and this may favour Tuberculosis of the apex; or the pleuritis is complicated with pneumonia—a common occurrence—and the inflamed portion of the lung is the susceptible part; or adhesions left between the two layers of the pleura lead to unequal expansion of the lung, and perhaps localised emphysema.

In looking through a number of my hospital cases of Phthisis, I was struck by the fact that, in the majority of instances in which pleurisy was mentioned as an antecedent illness, the tubercular deposit apparently commenced at some distance from the seat of the pleuritis—

in the apex of the same or of the opposite lung. Where there has been much effusion we can understand how the apex of the lung on the affected side might be rendered more prone to Tuberculosis; but how are we to explain the connection between pleurisy at the base of one lung and tubercle later in the apex of the opposite lung? A similar condition is to be noticed also in relation to pneumonia, and it was probably observation of this fact which led Louis ("Recherches sur la Phthisie") to disbelieve in the predisposing influence of pleurisy and pneumonia. What the exact connection between a pleurisy on one side and Tuberculosis of the opposite lung may be I am not prepared to say; but given an attack of pleurisy in a delicate person, I should prefer to get him well away from the neighbourhood of consumptives for some time. We know that any febrile illness may act as a predisposing cause to Phthisis from the general debility produced. We may therefore say that pleurisy and pneumonia act as predisposing causes of Phthisis by their debilitating effects on the individual, and that in some cases these diseases may in addition determine the seat of tuberculisation by their local effects on the lung.

The following cases will exemplify the direct effect of pleurisy in favouring Tuberculosis:—

Case 1. Charles B., æt. 25, packer. No Phthisis in the family. Admitted January, 1892. With the exception of scarlet fever when six years of age, the patient was quite well until June, 1891, when he attended St. Bartholomew's Hospital with pleurisy in the left side. I have ascertained from the house physician at St. Bartholomew's that he had no sign of tubercle at that time. On admission into my wards in January he had breaking down tubercular deposit

in the left apex, and a cavity with surrounding deposit at the left base. Tubercle bacilli were numerous in the sputa. He improved considerably during his stay in hospital.

It is not often that one is fortunate enough to find so clear a case as this one, for here the patient was attended at a large hospital for his pleurisy, and on examination of his lungs there he apparently was at that time free from tubercle. Yet six months later there was undoubted Tuberculosis.

Case 2. William G., seaman, æt. 26, was in Plymouth Hospital for five months in 1885 with pleurisy on the right side. On leaving hospital he caught cold, and was again laid up for three months. He had a third attack about 18 months later, and was an in-patient at Brompton in 1887. He came under my care in April, 1888, and there was then softening tubercular deposit throughout the whole upper lobe of the right lung. There was no history of Phthisis in the family.

Here we may fairly assume that the first attack of pleurisy was not tubercular, for the man came of a healthy family and was following an outdoor occupation. The condition of his lung when I saw him did not suggest a Tuberculosis of as long duration as three years. The first attack of pleurisy evidently weakened the lung, for he soon got a second attack, and later a third. It is possible that he had become tubercular before the last pleurisy.

Case 3. William B., æt. 33, baker. Mother died of Phthisis. Three years before admission into the North London Consumption Hospital under my care he had pleurisy of the right side which was tapped. The side filled again and was tapped in all six times, the fluid on each occasion being clear. He had, when I examined him, tubercular deposit in the right apex. Twelve months later he was again in my wards, the disease had increased at the right apex

where excavation had occurred, and there was some deposit in the left apex also.

Case 4. Fanny W., parlour-maid, æt. 22. Good family history. Admitted April, 1890. Diphtheria in 1887. Pleurisy on the left side in February, 1889, and again on the opposite side (right) in July, 1889 and in January, 1890. She had felt pains in the chest off and on since the first attack of pleurisy, and on admission to hospital had extensive deposit throughout the left lung.

This is a good example of the difficulty met with in determining what relation the pleurisy may bear to the Phthisis. With a bad family history, one would have felt certain that the pleurisy followed rather than caused the Phthisis.

When the effusion remains unabsorbed and becomes purulent, we have a general as well as a local cause for susceptibility. For the debility common in empyema reduces the defensive power of the system generally, and this intensifies the risk to the damaged lung. Thus we find that empyema is not uncommonly followed by Phthisis. Nor is the risk in this direction much lessened by free opening, for the partially collapsed lung is liable to suffer from Tuberculosis.

Case 5. *Empyema as an antecedent to Phthisis.*—Richard W., traveller, æt. 29. One sister died of Phthisis, and the patient himself had always been delicate and had a piece of bone removed from the ankle when he was twelve years old. In August, 1890, he caught cold, which was followed by pleurisy on the left side. Later paracentesis was performed, the fluid being thick (purulent). He came under my care in July, 1891, and then had a cavity about the level of the left scapula, with deposit and softening extending to the apex.

Pneumothorax.—Air in the pleural cavity is a result of Phthisis. In the rare cases in which it is antecedent to

Phthisis, it is rather the accident which caused the pneumothorax which leads to Phthisis, than the occurrence of air in the pleural cavity.

The second group in our classification includes those diseases of the air-passages and lungs which predispose to Phthisis mainly by producing a local nidus for the bacilli, such as catarrh, bronchitis, bronchiectasis, and pneumonia.

Catarrh.—Many patients date the first symptoms of what we discover to be Phthisis from a common cold, which instead of disappearing in the usual way remains and imperceptibly developes into the more serious illness. In many of these cases, we may assume that the "cold" which we are told preceded the Phthisis was in fact part of the disease; in other words, the patient mistook the early symptoms due to tubercle in the lung for a simple cold. But there is little doubt that neglected catarrh may predispose to subsequent Tuberculosis, and by some authorities this is considered one of the most potent predisposing causes. Tuberculosis of the lung induced by catarrh is exemplified in the following cases.

Case 6. John W., æt. 20, labourer. Good family history. Always healthy and well until present illness. About twelve months before admission he got wet and caught cold, but took no particular notice of it. Has had a cough ever since, and night sweats for six months. On admission (May, 1890) there is deposit in the left lung, with excavation at the apex.

Case 7. James M., æt. 40, gardener. One brother died of Phthisis. Quite well until the present illness, with the exception of enteric fever when twelve years of age. Got wet nine months

before admission and caught cold. Has had a cough ever since. On admission (January, 1890) there was deposit and softening throughout the left lung, with cavity at the apex.

Case 8. Robert M., æt. 33, gardener. One sister died from hæmoptysis, aged 34. He attributes his illness to catching cold two or three months before admission in January, 1892. After this cold he had "influenza," and has had a persistent cough since. On admission there was deposit (early) at the right apex; bacilli were found in the sputa.

The three preceding cases improved considerably in hospital, and as far as my observation goes Phthisis following catarrh may be expected to do well unless there is a bad family history, probably because the lung was little damaged before becoming tubercular.

Bronchitis.—*Acute Bronchitis* is less likely than the chronic ailment to form the starting point of tubercular disease, for the illness is comparatively short, and the patient usually is kept at home during the attack and away from tubercular infection.

In the following case, however, Phthisis appears to have been started by an attack of acute bronchitis, during the latter part of which there was exposure to intense tubercular infection.

Case 9. L. E. D., æt. 27, a nurse at a provincial hospital for consumption, was laid up with an attack of bronchitis. Examination of the chest at this time gave no suspicion of tubercular deposit in the lungs. Before she was quite recovered from the bronchial attack, a bad case of Phthisis in the wards required special nursing, and she was put on duty to attend to this patient, whom she nursed until his death soon after. Three months later, after she had left this hospital, I examined her and found tubercular deposit in the left apex, and in seven months more she died from acute Phthisis. There was no family history of Phthisis.

It may be that there was tubercle in the lung before the bronchial attack, although, as the chest was examined by one of the physicians to a Consumption hospital who failed to detect any signs, this is not likely.

Chronic Bronchitis or winter cough is sometimes antecedent to chronic Phthisis. A person may suffer for several years from winter cough, and then the cough becomes persistent throughout the year, and tubercle bacilli are found in the expectoration. In such cases, although the winter cough may have been the starting point of the Tuberculosis, the emphysema and fibroid degeneration in the lungs due to the chronic bronchitis will usually prevent the rapid destruction of the lung from the tubercular process. For, as we have seen, general emphysema, though not commonly associated with Phthisis, is not incompatible with tubercle, and fibrous tissue does not readily break down. The large *râles* of bronchitis occurring in the course of Phthisis often mask the signs of tubercular mischief, which may then be overlooked if reliance is placed on a single examination of the chest.

Bronchiectasis as a starting point of Phthisis is well illustrated by the following case, which was shown at one of the clinical meetings of the Harveian Society before there were any signs of Tuberculosis.

Case 10. George T. L., æt. 20, seaman. No family history of Phthisis. Patient was quite well until three years before his admission into the hospital in December, 1888. At that time he caught cold, which was followed by cough and pain in the right side which "caught his breath." He was found on admission to have a greatly dilated bronchus forming a cavity at the right base,

but no tubercle. About twelve months later the patient was again under my care, and had then tubercular softening in the right lower lobe, and deposit in the upper lobe on the same side.

Pneumonia.—Acute idiopathic lobar pneumonia, terminating in complete resolution, is not a likely cause of Tuberculosis. But when the inflammatory products are long in being absorbed there is more risk from tubercular infection. In cases of Phthisis in which there is a history of antecedent lobar pneumonia we often find the Tuberculosis starting in some distant part of the lung, as I mentioned when speaking of pleurisy.

Case 11. Clement C., æt. 27, carpenter, good family history. Strong and well until eleven weeks before admission, when he had an attack of Pneumonia at the right base, and was confined to his bed almost up to the time when he came to the hospital. He was found to have early Tuberculosis of the right lung, which later attacked the left apex. He died ten weeks after leaving the hospital, where he had been for six weeks.

Case 12. Some time ago I was asked to see in consultation at Clapham a young man who had acute Phthisis, and whose lung disease dated from an attack of acute pneumonia a few weeks previously. Here there was no question of the pneumonia, which had followed upon his getting wet and over-fatigued during a long bicycle ride.

Sometimes an attack of "inflammation of the lung" mentioned by a patient, in giving the previous history, really refers to the acute febrile stage of Tuberculosis. This was evidently not so in the above case, but it may have been so in the following.

Case 13. Annie B., dressmaker, æt. 37, dates her illness from an attack of inflammation of the lung four weeks before admission. She gives a history of four previous attacks all on the left side, and has had a cough for several years. One sister died of Phthisis.

This patient had softening tubercle at the left apex. Several similar cases might be quoted.

When the upper lobe of the lung is the seat of pneumonia we are often led to expect the presence of tubercular deposit as a cause of the inflammation. But apex pneumonia occurs without tubercle. The two following cases are interesting in this connection.

Case 14. *Apex Pneumonia without Tubercle.*—Mrs. M., the wife of an army medical officer, was taken ill four years ago with fever, cough, and great prostration. Examination showed pneumonia involving the apex of one lung. For some days she was dangerously ill, but eventually the lung cleared completely, and has remained healthy up to the present (1892).

Here there was apparently no tubercular deposit, for I have had several opportunities of examining the chest since, and have found no evidence of mischief in the lung. There was a history of a similar attack of pneumonia, also at the apex, some years before this one.

Case 15. *Apex Pneumonia with Tubercle.*—In the spring of 1891 I saw, with Dr. Watson, of West Hampstead, a young married lady (Mrs. R.) who had clear signs of pneumonia at one apex without any marked illness, but with a good deal of cough. There was no history of a sudden onset, but the patient had been ailing a week or two before she sought advice. I expressed the opinion that the pneumonia was probably started by a patch of tubercle in the apex of the lung, which would be detected when the surrounding pneumonia cleared up; and as the only certain way of deciding the presence of tubercle I suggested the microscopical examination of the sputa. Bacilli were found in the expectoration, and as the apex cleared large crackling râles became audible. The disease rapidly increased, and the patient went abroad to avoid the English winter.

When possible the sputa should always be examined for

bacilli in pneumonia of the apex, as this frequently is caused by tubercular deposit.

There is a subacute variety of pneumonia which may take a long time to resolve, and which is specially liable to be associated with subsequent tubercular disease. This variety may follow whooping-cough, measles, or influenza.

The following is a case of this kind of pneumonia, which fortunately has not as yet been followed by Tuberculosis.

Case 16. Hilda W., æt. 12, had whooping-cough in August, 1891, at the same time as the other members of the family. She had for years suffered from spasmodic asthma, and was longer in getting rid of her cough than the other children, cough attended with whooping persisting until November. By Christmas she seemed quite well again, but early in January she was listless and feverish, and then was found to have dulness of the left base as high as the angle of the scapula, with tubular breathing. The temperature reached 103° F.; respirations 44. There was no rigor or marked sign at the outset. The dulness and other signs gradually disappeared, but the lung was not clear until the end of February.

Here we have a pneumonia which takes a long time to resolve, and which but for the previous whooping-cough might have suggested a tuberculous origin, until its complete resolution negatived such a supposition. This case, following whooping-cough, is mentioned as an example of a condition which is very likely to favour subsequent Tuberculosis of the lung, although fortunately there appears now to be no tubercle.

Catarrhal pneumonia is more frequently a cause of Phthisis than lobar pneumonia, and it is this variety of lung inflammation which often follows whooping-cough, measles, or influenza. These three diseases are frequent antecedents of Phthisis, and here we have the marked

general debility which lessens the resisting power of the individual, as well as the special predisposition due to the morbid condition of the lung. The case last mentioned illustrates the predisposing lung mischief after whooping-cough, whilst those which follow show Phthisis after measles and influenza respectively.

Case 17. *Phthisis following Measles.*—Lydia W., æt. 14, had measles four years before she came under my care, and had suffered from shortness of breath and cough ever since. She began to cough up blood soon after the measles, and was in Brompton Hospital twice before she was admitted to the North London Consumption Hospital in August, 1888. She then had extensive tubercular softening in the right lung, and deposit in the left apex.

Phthisis following Influenza.—During the last few years a large number of patients have come under my notice with Phthisis, who attribute their illness to influenza. And when we consider the extreme debility produced by this ailment—often lasting a long time—there can be little doubt of its predisposing to Tuberculosis. The pneumonia which occurs as a complication or sequela of influenza may be long in clearing away, and then a local as well as a general predisposition is established. In both the cases I have selected to illustrate Phthisis following influenza there was some lung complication with the attack.

Case 18. John S., æt. 41, gardener, had influenza with pleuro-pneumonia in February, 1890, since which he has had cough, shortness of breath and pain in the chest. He was admitted to my wards in February, 1891, with tubercular deposit in the left lung.

Case 19. Emily H., æt. 21, ladies maid, had influenza with pneumonia in January, 1890, and was admitted under my care in the following July with slight deposit at both apices.

Hæmorrhage in the Lungs is often an indication of Tuberculosis, even when no physical signs are detected. The sputa, if any, should be examined for bacilli. Pulmonary hæmorrhage from heart disease or congestion from any cause, may be the starting point of Tuberculosis in the lung.

It has been stated that mitral disease is antagonistic to Phthisis, but I have records of many examples of the co-existence of these two morbid conditions, which I hope to bring forward on some other occasion.

Injuries to the Lung.—Injuries to the lung may induce Phthisis, either by the hæmorrhage, or later by the adhesions of the pleura.

In the following cases an injury to the lung may have had some effect in determining the subsequent Phthisis, or at least the seat of primary deposit.

Case 19A. Richard H., æt. 43, labourer. Fracture of left clavicle twelve years ago. Dates his present illness from a cold caught five months before admission. Tubercular deposit at left apex.

Case 20. John E., æt. 43, formerly hawker. Fell down a cellar about three years ago and broke his ribs, bringing up blood as a result of the accident. Nine months later he commenced to spit blood, and has since been a constant inmate of hospitals. He has softening at right apex, and slight deposit on the left side. He has an alcoholic history, but gives no family history of Consumption.

Case 21. Arthur C., æt. 29, coachman. Good family history. Quite well until five years before admission, when he was thrown from a horse and struck the left side of his head and chest. Cough began three or four months after the accident, and has persisted. Tuberculosis of left lung with cavities in upper lobe.

Case 22. W. B., æt. 23, painter. Good family history. Quite

well till four years before admission, when a blow on the left side caused him to cough up blood immediately afterwards. The bleeding soon ceased, but has recurred several times since. On admission, in 1888, there was tubercular deposit at the left apex. Two years later he again came under my care, the whole left lung was then involved, and there was deposit in the lower lobe of the opposite lung.

Case 23. George B., æt. 35, painter. No Phthisis in family. Admitted November, 1890. Fracture of left clavicle seventeen years ago; was stabbed in the upper part of the chest on the right side about twenty years ago. Fifteen years ago fell off a scaffolding and had bleeding from nose and mouth. In 1888 brought up a pint of dark blood, and has not been well since, having cough, expectoration, pain in chest, and sweating. On admission, softening at right apex. Here possibly adhesions where he was stabbed determined the seat of the deposit of tubercle many years later.

The cases in which the above-mentioned pulmonary affections appear to have been the starting point of tubercular mischief are few as compared with the numerous examples where one of these diseases occurs in the course of a pre-existing Phthisis. It is important, however, that we should recognise them as possible causes, as well as frequent complications, of pulmonary Tuberculosis.

In conclusion, what are the practical deductions to be drawn from the recognition that non-tubercular affections of the lungs predispose to the deposit of tubercle? Perhaps the most important is that we must exercise a careful supervision of such patients as have become thus rendered liable to Consumption, and make careful examination of the lungs when any persistent cough, raised temperature, or continued depression of health point to the possibility of tubercular infection. Tubercular disease may be curable in its early stages, but becomes more and more difficult to check as the deposit increases. Thus the importance of

the early recognition of Tuberculosis is essential; and as the early signs are not easy to detect, it is necessary that a careful examination of the chest should be made whenever there is any suspicion of ill-health. It is not enough to listen through clothing, or to wait until large deposit or softening renders the nature of the disease obvious to the most casual observer. The chest must be bared, and examination made in such a way that slight divergencies from the normal can be detected; a clear knowledge of the indications noted is also necessary in weighing the evidence obtained by careful investigation in order that the early presence of tubercle may be recognised. Microscopic examinations of the sputa should be made when there is cough with expectoration. It would be well to recommend a periodical and systematic examination of the chest at intervals for a year or two in all cases where bronchitis, pleurisy, or pneumonia have occurred in delicate persons; especially where there is any tubercular tendency, and more particularly in young persons during the period of development. The same advice may well be given where the health has been lowered by whooping-cough, measles, or influenza.

CHAPTER V.

THE INFLUENCE OF HEREDITY IN PHTHISIS.

The question of heredity in Phthisis is one of great importance and of some difficulty. The importance of the subject cannot be doubted, for there is hardly a family in the country in which Phthisis has not claimed a victim in one or more generations. If heredity be a potent cause of the disease, how few are the individuals who could hope to escape! But is the disease hereditary? The difficulties of deciding the question are numerous. Not only is the collection of the necessary *facts* far from easy, from the want of knowledge or the want of accuracy on the part of patients in giving their family histories, but the *deductions* from these facts when obtained are by no means clear.

It is difficult also to approach the question with an unbiassed mind, in the face of the almost universal acceptance of the hereditary nature of Consumption handed down from the past. The universality of the belief in the hereditary nature of Consumption is easily explained when we know how frequently Phthisis is found attacking several members of the same family in successive generations; and anyone who wished to uphold the doctrine of heredity in Phthisis would have no difficulty in amassing facts in support of his thesis. It is the apparent self-evidence of the matter which makes it

difficult to conceive that there is any room for discussion. As Wilson Fox remarks, "the influence of this cause (heredity) in the production of Phthisis is unquestioned." That Phthisis is more common in some families than in others is not open to question; but the same is true of almost any disease, or indeed of any occurrence. If we were to investigate the statistics of scarlet fever or of malarial fever, we should find apparent support for a contention that these are hereditary; for undoubtedly we should find these diseases occurring repeatedly in some families, and never recorded in others.

Most physicians have met with families the members of which, if exposed to the infection of scarlet fever, are almost certain to catch the disease, and always suffer from it severely. They do not "inherit" scarlet fever, but those who belong to such families should most carefully avoid exposure to infection. Alcoholism, like other forms of neurosis, may often depend upon an inherited predisposition; but we all recognise that careful training and complete abstinence from alcoholic beverages from childhood—*i.e.*, avoidance of the determining cause—will prevent the constitutional weakness from developing in this particular form. Some other form of neurosis may develop, but not alcoholism with its degradation and disgrace. So with the constitutional weakness which predisposes to Consumption, we cannot always ensure that the individual shall become robust and enjoy a long life; but we may guard him from becoming consumptive, and, by so doing, not only benefit him, but avoid a centre of infection to others. Every individual saved from Consumption is a direct benefit to the public health.

A family in which for generations the sons have served in the army and navy would possibly show malarial disease in some of its members for several generations; but this would not prove any hereditary transmission of malarial taint. The occurrence of ague for generations in families living in the Fens is no proof of the hereditary nature of malaria; nor is the frequency with which pediculi attack the children of successive generations of some poor families any evidence of heredity in this occurrence. In the above instances the mode of life, place of residence, and surrounding conditions are easily seen to exert so powerful an influence in determining the morbid condition as to furnish a complete explanation of its causation; and we at once agree that the greater incidence of these conditions on certain families is due to the members of them following the same profession, or living amidst similar surroundings. With Phthisis, however, we are less able to see clearly the relative value of hereditary influence on the one hand, and of occupation and surroundings on the other. It is only within the present generation that the nature of pulmonary Consumption has been determined, and the influence of the surroundings and occupation of the individual on its causation more accurately studied.

The belief in the influence of heredity in Consumption rests largely on the fact that, in a large proportion of cases in which the disease occurs, it is found that other members of the patient's family—it may be one or both parents—have suffered from the same complaint. This is almost to be expected, apart from any theory of heredity, with such a widely prevalent disease as Consumption.

Formerly, the belief in heredity was such that this was looked upon as the most important factor in causation, and members of a family in which Consumption had already claimed victims almost considered themselves as hopelessly doomed to an early death from this disease. It was not until about the middle of the present century that the influence of heredity was shown to be less than had till then been supposed, and Walshe pointed out that in only one-half of the cases of Consumption could any evidence of hereditary predisposition be established. Now, we find the proportion of cases in which some hereditary predisposition may be assumed is stated to be about 30 per cent., or less than one-third of the cases.

Beliefs which have been held for ages are not easily relinquished, even when the contentions upon which they were founded have been disproved. It requires a more ample array of facts and more accurate reasoning to disprove an old theory than to successfully launch a new one. In the present case my inquiry has led me to consider the old theory of heredity in Phthisis to be fallacious. I did not propose to myself, in instituting this inquiry, the task of trying to disprove the theory that Phthisis is an hereditary disease. I set myself to analyse, as far as possible, the notes of 1,000 cases, chiefly drawn from my hospital records, and to see how the facts, as I find them given in these ward notes, fit in with the theory of heredity.

In the foregoing preliminary remarks I have merely endeavoured—with the object of clearing the ground and counteracting prejudice in favour of the heredity theory—to urge that the question of hereditary influence is still

open. I may at once state that, though I think my figures warrant a conclusion that direct heredity plays a less important part in the causation of Phthisis than has sometimes been supposed, there appears to be no doubt that *a predisposition to become phthisical*—if exposed to tubercular infection—is often dependent upon inherited constitutional weakness.

Before proceeding further it will be well to define more exactly what we understand by heredity as a factor in disease causation.

(A) There is first **direct hereditary transmission** of a disease, the development in the offspring before birth of a morbid condition derived from the parent. The child is born suffering from the disease. This is true hereditary transmission, and is exemplified in congenital syphilis; it also appears in some inherited deformities, such as supernumerary fingers or toes. The disease is then properly called "congenital." This kind of heredity is seen in those cases, comparatively few in number, in which Tuberculosis has been found in the young of animals *in utero*, or in the infant at birth.

(B) Then, secondly, the disease may be implanted in the body before birth, but does not manifest itself until some time after, *e.g.* infantile syphilis.

It is almost impossible to prove the occurrence of this mode of transmission for Tuberculosis; for there are so many ways in which the causative bacillus may get access to the body of the infant after birth. It is, therefore, difficult to say whether a Tuberculosis, showing itself some weeks after birth, is inherited (congenital) or

acquired; in the majority of cases I should suspect the latter mode of origin.

In both the above categories a definite germ of disease is implanted; they differ thus essentially from the next two.

(c) Of these, the most common way in which heredity acts in disease causation is that there is transmitted a peculiar liability or susceptibility to develop a certain disease if the individual be exposed to the exciting cause of that particular disease; there is a diminished resisting power with regard to some special ailment. Here a predisposition is inherited, not the disease itself, and we may call this "direct hereditary predisposition." Examples of this form are numerous, as in gout and insanity. This susceptibility may be transmitted by parents who, though themselves inheriting the predisposition, have escaped the disease—"atavism." It may be noted that this escape of the susceptible person shows that the inherited tendency may be combated and the disease itself avoided by avoiding the determining or exciting cause.

(d) In the last form the inherited susceptibility to disease may be general, not special, *i.e.* a liability to take any of a wide range of diseases to the exciting cause of which the person may subsequently be exposed. There is a diminution of resisting power against disease in general. We may term this general inherited predisposition to disease, or delicacy of constitution.

Direct hereditary predisposition is what is now generally implied when Consumption is said to be inherited.

As we are not now discussing Tuberculosis in general,

we may dismiss, for the purposes of this inquiry, the two first-mentioned forms of heredity, *i.e.* congenital diseases. Phthisis, or primary pulmonary Tuberculosis, is essentially a disease of adult life, and it is difficult, with our present knowledge of the causative micro-organism of Tuberculosis, to imagine the bacillus remaining latent in the body from birth to maturity (*a*).

We have, then, to consider—

(1) Direct hereditary predisposition to Phthisis.

(2) General inherited delicacy of constitution in relation to Phthisis.

The first, being a definite tendency to a particular disease transmitted from parent to offspring, would be a true heredity, and as such is a matter of considerable importance. It behoves us, therefore, carefully to inquire

(*a*) Cases frequently occur which tend to show that the bacillus of Tuberculosis may remain potentially active in the body for a long time without causing symptoms, and may yet eventually light up fatal disease. Encapsuled masses of tubercular disease, containing bacilli, may remain for considerable time in the lung without any apparent effect on the health, until in some way the bacilli escape and infect other parts of the lung, the disease then rapidly spreading. An example of the escape of bacilli from such an encapsuled mass was shown by me at the Pathological Society in 1893. Those cases in which the individuals in early life suffered from strumous glands—Tuberculosis of the superficial cervical lymphatic glands—and then in later life develop pulmonary Tuberculosis, may be examples of a similar event; though it is impossible to exclude the influence of a fresh infection of the lungs from without. The comparative immunity of the sufferers with strumous glands in childhood from subsequent Phthisis is against the theory of auto-infection in these cases.

whether it can be proved to exist for Phthisis, and if so to what extent.

The second, though having an important bearing on the future health of the individual, hardly comes properly under the designation of an hereditary predisposition to Phthisis, since it might equally plausibly be considered an hereditary tendency to scarlet fever or to yellow fever, or to almost any disease to which the individual might become exposed. This is in fact the essential point to which I wish to draw attention, and which I have here set myself to examine. Is the inherited predisposition, which is at present considered so important and so potent a factor in determining the incidence of Consumption, a true heredity, or is it merely an illustration of the fact that those of delicate constitution, from whatever cause, are more liable to contract diseases to which they become exposed than those who are constitutionally robust?

Again we find it necessary to define terms somewhat more exactly. What do we understand by "delicate" and "robust" constitutions?

The breeders of animals know well how much of the usefulness and capacity for work depends upon the stock from which the animal springs. They recognise that only selected animals should be utilised for breeding purposes, and that these stud animals must be kept under certain conditions as to food, air, and exercise, in order that their offspring may turn out valuable. They endeavour to ensure that whatever peculiarities of shape, colour, or disposition the young animals may inherit from

their parents, they shall have no blemish and no inherited liability to illness. The offspring of healthy parents who have been living in accordance with hygienic requirements will probably be themselves healthy, and if kept under similar hygienic conditions will in all probability grow up strong and capable of a full measure of work. Such animals, if exposed to unfavourable (unhygienic) conditions, such as extra strain of work or unhealthy surroundings, are less likely to break down, or will withstand the evil effects of these conditions for longer than animals less carefully bred. They have "robust" constitutions — the strength which lies in healthy tissues and healthy organs performing their functions naturally and harmoniously. Divergence from this desirable condition, if habitual, constitutes "delicacy," the ever-present liability to break down under a strain, or to become *invalid*.

Suppose the constitutionally robust animal to be afterwards placed under conditions unfavourable to health, his inherent strength may prevent a break-down. But although not suffering from any definite disease, such an animal, whose tissues and organs are now unable perfectly to perform their several functions, would, in all probability, produce offspring similarly deficient in functional vigour—*i. e.* constitutionally delicate. A stage further in the downward course, and the animal would be incapable of producing offspring at all. This, then, illustrates the well-known fact that unhealthy offspring may come from parents who have no actual disease.

The same is true for human beings: if the parents are

not strong and healthy, the children will be delicate in constitution, less able to bear the strain of work and anxiety, and more easy victims to disease than the children of perfectly healthy parents. The debility of the parents is not necessarily due to disease; it may be dependent upon unhygienic surroundings, yet the result is similar in the children—they have less power of resistance against disease. The Londoner of the fourth generation is said not to exist; and even if this statement oversteps the bound of literal accuracy, it is undoubtedly true that the conditions of life in the crowded slums of a large city tend to diminution of vitality in the individual. The children of parents thus debilitated, and themselves brought up in the same unhygienic surroundings, are weakly and delicate, and have small resisting power against disease whether it be Tuberculosis or some other malady. The particular disease which attacks them depends upon the liability to exposure to one or another of them. The debility of the parents may, on the other hand, be due to their being consumptive; and if this cause a greater liability to the children becoming phthisical—if there be a special or specific hereditary predisposition—we should find the children of such parents phthisical in greater proportion than the children of parents whose debility arises from other causes. Let us then carefully examine our figures to see how far this is the case. Out of 1,000 patients suffering with Consumption, 325 gave a history of Phthisis in one or both parents, and 675 gave no such history. In 474, out of the 1,000, I was able to obtain a complete history of the family as shown below (Table I).

Table I.

Showing the incidence of Phthisis in the Children of non-phthisical and of phthisical Parents respectively.

474 Cases.	Children.		Died in Infancy.		Phthisical.	
	Total No.	Proportion to each family.	Total No.	Per cent. of whole.	Total No.	Per cent. of children, omitting infantile deaths.
A. 275 families. With *no Phthisis* in parents	1,745	6·34	193	11·06	386	24·87
B. 84 families. *Fathers phthisical*	511	6·08	67	13·11	138	31·08
C. 82 families. *Mothers phthisical*	506	6·17	56	11·06	155	34·44
D. 33 families. *Both parents phthisical*	165	5·00	18	10·90	58	39·45
199 families. Phthisis in one or both parents (Summary of B, C, D)	1,182	5·93	141	11·92	351	33·71

We find here that out of 1,000 cases of Phthisis, one or both parents were consumptive in 325, or 32·5 per cent. In 474 of these cases I have obtained a fuller account of the family, and of these 199 (nearly 42 per cent.) give a history of Phthisis in the parents. In the families of the non-phthisical parents, 24·87 per cent. of the children became phthisical, against 33·71 per cent. of the children of the phthisical parents (*b*). This shows a

(*b*) It is remarkable how closely the figures obtained by two

difference of only about 9 per cent.—a very small influence in favour of hereditary predisposition.

This additional 9 per cent. of phthisical children in those families in which the parents are consumptive might be explained by the increased exposure to the exciting cause, to which those living with consumptive parents are necessarily liable. Dr. Philip, of Edinburgh, in an analysis of 1,000 cases of Phthisis, put the percentage of cases in which the disease seemed to him to be due to direct infection at seven, or very nearly the whole proportion which, in the figures given above, represents the possible influence of hereditary predisposition.

It is argued that the parents may transmit this specific predisposition from their parents, without being themselves consumptive; so that to exclude the possibility of hereditary transmission in any case we should know the history of earlier generations. We might grant this, and yet take a very hopeful view of the effect of hereditary influence in Phthisis. It seems probable that inherited peculiarities become weakened in each successive generation, and tend to die out unless strengthened by intermarriage with a family in which the same peculiarity is strongly marked. If, therefore, the offspring of phthisical parents escape the disease, they transmit the predisposition with a diminished potency, and their children should be even more able to escape. I would lay stress upon this point, that however frequent we may find

different modes of inquiry approach to one another. The proportion of phthisical patients who give a history of Phthisis in their parents is 32·5 per cent., whilst the proportion of the children of phthisical parents who became consumptive is 33·71 per cent.

the hereditary influence to appear in statistics, it is not so strong as to cause alarm; though it may be strong enough to serve as a caution, and to indicate the necessity for extra care in avoiding the determining cause of the disease.

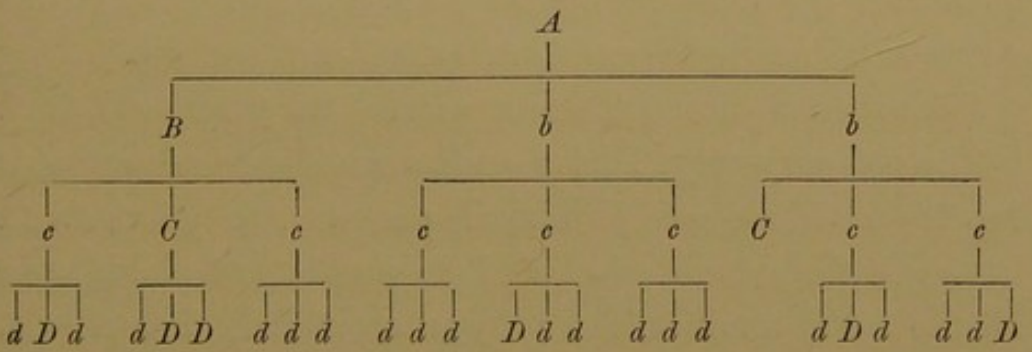
We will now proceed to a fuller examination of our figures, and see what we can glean from them. In the 1,000 cases a history of Phthisis in one or both parents was obtained in 325 cases, giving a proportion of 32·5 per cent. of the total number of cases; 604 were males, and of these 175 had a history of Phthisis in the parents, *i.e.* 28·97 per cent. The 396 females gave a history of Phthisis in the parents in 150 cases, or 37 87 per cent.

So far we see that in 1,000 families in which one or more members of one generation became consumptive, one or both parents also had Phthisis in about a third of the whole number. If we were to include uncles and aunts and grand-parents, the proportion of families in which Phthisis was present in previous generations would be increased to about 35 to 40 per cent. This would seem to show a fairly strong hereditary influence, but on the other side we must put the not inconsiderable number of families in which phthisical individuals have had children and grandchildren free from the disease, and which necessarily do not appear in statistics taken from the records of hospitals for Consumption. We are at once face to face with one great difficulty in attempting to decide the influence of heredity in Consumption. To obtain reliable results we should commence with phthisical individuals, and obtain the history of their children and grandchildren, or their offspring for several generations;

and compare the result as regards the incidence of Phthisis with that in the descendants of a similar number of individuals who were not consumptive. This would require much laborious searching of family records, or the cumulative results of observations continued through several generations. All statistics which commence with the phthisical patient and trace the family history back through one or more generations, give results as regards heredity which are fallacious, and which place the proportion of hereditary cases too high.

In illustration of this statement let us take a supposititious family with a very bad phthisical history. We have seen that of the children of phthisical parents, only about one-third become consumptive. In the following scheme capital letters indicate phthisical individuals, the small letters those who remain free.

SCHEME *of supposititious Family with bad Phthisical History. Capital Letters indicate Phthisical Individuals.*



To illustrate how the influence of heredity appears unduly great if we trace *back* from phthisical patients *D*, instead of *forward* from *A*.

Here we have Phthisis in the parent *A* attacking one in three of the children, and showing in two out of nine

individuals in the third generation, and six out of twenty-four in the fourth generation. If we start with the last generation we have six cases of Phthisis (*D*), of which two show Consumption in the parents, a proportionate heredity of 33·3 per cent.

If we trace back to grand-parents the proportion showing heredity is 50 per cent., and going back one more generation we find it to be 100 per cent. Even if we only go back one generation and take the collateral branches (uncles and aunts) the hereditary influence appears as high as 83·3 per cent., that is five out of six consumptives (*D*) show Phthisis in parents or uncles and aunts (*C*). If, however, we commence with the phthisical patient *A*, we find the influence of heredity represented by 33·3 per cent. in the children, by only 22·2 per cent. in two generations, and by 25 per cent. in three generations, in spite of the large number of consumptives in the last generation.

We might, under unfavourable conditions of life, find quite as large a proportion of phthisical persons in three generations derived from a perfectly healthy stock.

Seeing, then, how fallacious will be the results obtained in the ordinary way, I have endeavoured to find out how far the children of phthisical parents are affected. I have not the details of the whole number of children in all the 1,000 families, but only in about half of these, viz. 474. In these 474 families there were 275 in which neither of the parents was phthisical. These families comprised 1,745 children (an average of 6·34 to each family); 935 were males and 787 females, and 23 who died in infancy, of whom the sex is not mentioned. If

we divide these 23 proportionally between the sexes we should have 947 males and 798 females (see Table II).

TABLE II.

Apparent Heredity in 474 Cases.	Sex of Children.	Children.		Died in Infancy.		Phthisical.	
		Total No.	Proportion to each family.	Total No.	Percentage of whole.	Total No.	Percentage of children, omitting infantile deaths.
A. Nil. 275 families	M.	947	3·44	111	11·72	244	29·06
	F.	798	2·90	82	10·41	142	19·83
B. Paternal. 84 families	M.	267	3·17	39	14·60	62	27·19
	F.	244	2·90	28	11·47	76	35·18
C. Maternal. 82 families	M.	246	3·00	33	13·41	84	39·43
	F.	260	3·17	23	8·84	71	29·95
D. Double. 33 families	M.	84	2·54	12	14·28	28	38·88
	F.	81	2·45	6	7·40	30	40·00
Summary of B, C, D. 199 families	M.	597	3·00	84	14·07	174	33·91
	F.	585	2·93	57	9·74	177	33·52

Of the *male children* 111 died in infancy (11·72 per cent.), and of the remaining 836, 244 became phthisical, or 29·06 per cent.

Of the *female children* 82 died in infancy (10·41 per cent.), and of the remaining 716, 142 became phthisical, or 19·83 per cent. The proportion of phthisical invalids of both sexes is 24·87 per cent.

The above figures tend to show the influence of occupation in determining Phthisis where there is no special predisposing cause in the family; for the males suffer out of all proportion to the females.

In 204 out of the above 275 cases I have noted the

grand-parents and uncles and aunts of the patient, and in 179 there was no Phthisis at all known in the family, whilst in 25 cases there was Phthisis in grand-parents or collaterals. I have endeavoured to find whether the proportion of individuals affected with Phthisis is modified by this presence of Consumption in the remote members of the family. The numbers are too small to be of much value, but my figures show that in the families where Phthisis has been previously known, though the total incidence of Phthisis was less than where no Phthisis has been present (19·39 per cent. against 20·48 per cent.), the proportion of females attacked by Phthisis is much greater in those families where previous examples of the disease are noted (males 16·25 per cent., females 22·35 per cent. as against males 27·78 per cent., females 18·69 per cent.) (Table III).

Table III.

Showing a further Analysis of 204 (*from Cases* A *in the previous Table*) *in which the History of Grand-parents, Uncles, and Aunts could be determined.*

204 Cases with no Phthisis in the Parents.	Sex of Children.	Children.		Died in Infancy.		Phthisical.	
		Total No.	Proportion to each family.	Total No.	Percentage of whole.	Total No.	Percentage of whole, omitting infantile deaths.
179 cases with no Phthisis previously known in the family	M.	581	3·24	70	12·04	142	27·78
	F.	522	2·91	62	11·87	86	18·69
25 cases, Phthisis in grand-parents or collaterals . .	M.	84	3·36	4	4·76	13	16·25
	F.	89	3·56	4	4·49	19	22·35

In 84 families (out of the 474) the father was phthisical. These families comprised 511 children (an average of 6·08 to each family), of whom 267 were male and 244 female; 67 (or 13·11 per cent.) died in infancy, and of the remaining 444, 138 became phthisical, or 31·08 per cent.

In these families the incidence of Phthisis is greater on the female children than on the males (35·18 per cent. as against 27·19 per cent.).

Delicacy of constitution is as potent a cause as the effect of occupation.

We see, also, the result of weakliness in the father shown in the greater proportion of infantile deaths, especially amongst the male infants. The weakly sons having died off, the proportion of Phthisis amongst the surviving male children is less than in the previous class, but the girls are more prone to Phthisis than in families where there is no hereditary predisposition. We must remember, however, that the girls stay more at home than the sons, and are in closer contact with the phthisical parent, especially when the disease causes him to give up work and lie up.

In 82 families the mother was phthisical. These comprised 506 children (6·17 to each family), of whom 246 were male and 260 female. Fifty-six (or 11·06 per cent.) died in infancy, and of the remaining 450, 155 became phthisical, or 34·4 per cent.

Here, as in families where neither parent was phthisical, the male children suffer proportionally more than the female (39·43 per cent. against 29·95 per cent.). A greater proportion also of male children die in infancy (13·41 per cent. male, female 8·84 per cent.). The proportion

of females affected is actually smaller than in the families where the father was phthisical. It might be urged that the male children inherit the mother's characteristics, whilst the female children are most influenced by the male parent. This is the reverse of what Dr. Reginald Thompson gathered from his figures. When the mother is delicate the proportion of male children is deficient, whilst those that are born are wanting in stamina, as shown by their liability to Phthisis.

In 33 families both parents were phthisical; and in these there were 165 children (5·0 to each family). Eighteen died in infancy (10·9 per cent.), and of the remaining 147, 58 became phthisical, or 39·45 per cent.

The female children were only slightly more affected with Phthisis than the males (38·8 per cent. of males to 40·0 per cent. of females). The infantile deaths are numerous amongst the male children, but few amongst the girls. In the survivors Phthisis is common.

In the above families where Phthisis is noted as having attacked one or both parents, it cannot be stated whether the disease was present before the birth of the patient. In the majority of cases it is probable that at the patient's birth the parent was non-phthisical.

To sum up the preceding remarks, we see that if we take 1,000 cases of Consumption, we get a history of Phthisis in one or both parents in 325 cases, or 32·5 per cent.

We see also that in families of which one or both parents are phthisical, 33·71 per cent. of the children suffer from the disease; whereas in families with no Consumption in the parents, 24·87 per cent. of the children become phthisical. Phthisis in the mother results in a higher

percentage of Phthisis in the children than when the father alone is phthisical. This might be expected, without conceding any hereditary influence, from the more intimate relation between mother and children. When both parents are phthisical 39·45 per cent. of the children become consumptive.

It seems, then, that the influence of heredity may be represented by about 9 per cent.—the difference between the percentage of Phthisis in the children of non-phthisical parents and those whose parents were consumptive. Even when both parents were phthisical the influence of heredity cannot be seen to result in more than fifteen children in every hundred becoming phthisical.

I have collected the following figures from various sources, so as to get the percentage in which Phthisis occurred in the parents of consumptive patients from a sufficiently large number of cases.

	Cases.	Percentage heredity.	Number of cases in which the parents were phthisical.
Brompton (1st report) ...	1010	24	242
,, (2nd report) ...	4482	28	1255
Edward Smith	1000	21	210
Wilson Fox	384	33	127
Theodore Williams ...	1000	12	120
Philip	1000	23	230
Müller	988	21·8	215
Koranyi	900	20	180
Wahl	745	26·4	197
My own cases...	1000	32·5	325
Total	12,509	24·79	3101

We have here 12,509 cases, of which there was Phthisis in one or both parents in 3,101 instances, or 24·79 per cent. We may take it from the above figures that

25 per cent. is a fair average for the extent of apparent heredity from parents.

Naturally when phthisical grand-parents and collaterals are included we have a larger percentage of apparent heredity :

	Cases.	Percentage heredity.	Number of cases in which the antecedents were phthisical.
Brompton (2nd report) ...	4482	87	3899
Edward Smith	1000	56·2	562
Theodore Williams ...	1000	48	480
Wilson Fox	384	48	184
Cotton	1000	36	360
Müller	988	50·4	498
Bochdalek	3292	48	1580
Total	12,146	62·34	7563

The only comment I have to make on this second list is that the figures seem to me quite unreliable (especially as regards the Brompton report), for all who have much to do with London out-patients know how wide a term "Consumption" is when employed by them in their family histories; it includes, amongst other diseases, bronchitis, asthma, anæmia, and alcoholism.

I have previously pointed out that we have only to carry the inquiry sufficiently far back in order to bring up the apparent proportion of heredity to 100 per cent.

We may note in the first of the above lists how closely the percentage of apparent heredity in Dr. Wilson Fox's cases coincides with my own; in both of these series the patients were drawn from the same class of people, chiefly London poor living under unhygienic conditions (*c*).

(*c*) Having been house physician to Dr. Fox at University College Hospital, I am able to make this statement with confidence.—J. E. S.

It is also worthy of note that in the cases gathered from the London hospitals the percentage of apparent heredity is in excess of that for the total number of cases here shown. This might be taken to indicate that where the people are living under conditions favourable to the development of Phthisis the apparent influence of heredity is greater than elsewhere. To my mind it tends rather to show that mode of life and unhygienic surroundings, not inheritance, caused the disease in the children as in the parents.

The percentage of Phthisis in the children of *non*-phthisical parents amongst my patients is as great as that representing the hereditary influence in the first list; this, again, tends towards the conclusion that the value of the influence of occupation and surroundings in the causation of Phthisis is very much greater than that of any inherited tendency.

The relative value, as a cause of Phthisis, between heredity and the influence of occupation and surroundings is well shown in the difference between the percentage of cases where the parents were phthisical in Dr. Theodore Williams' 1,000 cases from private practice and that of 1,000 cases of hospital patients. Dr. Williams' cases give a proportion of apparent heredity of only 12 per cent. My hospital cases show a percentage of 32·5. Is not this almost sufficient in itself to negative the theory of direct heredity? For if hereditary influence were a potent factor in the causation of Phthisis we should expect to find it showing nearly equally amongst the rich and the poor.

It has been stated that each sex has a tendency to

transmit directly, fathers to sons, and mothers to daughters.

In the majority of cases, as far as I can gather, by which this statement has been supported, the figures are obtained by taking a single case in a family and finding out if either parent was consumptive. For example, a male patient presents himself with Phthisis and states that his father was phthisical; without further inquiry this might lead to error, for he might have two sisters phthisical, and be the only consumptive son out of several in the family. To show that this way of taking the manner of transmission would lead to such an error, I have worked out this point in the way I have above alluded to for some of my cases. In 500 male patients the father was phthisical in forty-eight cases, the mother in forty-six cases, and both parents in eighteen cases—showing transmission from father to son to be slightly in excess of cross-transmission, instead of as seen below.

By getting the history of all the children of a phthisical parent, as I have done in the present series of cases, we get a far more reliable result. It will be seen (Table II), that my figures are directly opposed to the theory of "direct" transmission.

The figures are—

Father to sons . . .	27·19 per cent.
Father to daughters . .	35·18 per cent.
Mother to sons . . .	39·43 per cent.
Mother to daughters . .	29·95 per cent.

This shows a cross-transmission, as do also the figures arrived at by Dr. Wilson Fox from his 384 cases.

In order to see that my figures might not be misleading from dealing with an undue proportion of one sex amongst my cases, I have compared the proportion of males to females in my 1,000 cases with the known proportion between the sexes amongst phthisical patients. I find my 1,000 cases to consist of 604 males and 396 females. In 1,000 cases recorded by Dr. Theodore Williams there were 625 males and 375 females.

The first Brompton report gives a proportion of 61 males and 39 females per cent.

The mortality from Phthisis in London in 1880 gives for every 100 deaths, 57 males and 43 females.

Finally, amongst the 737 children in my tables who were phthisical, I find 418 males and 319 females, giving a proportion of 56·72 males and 43·28 females per cent. The proportion between the sexes in the 1,000 cases dealt with in this series is, therefore, about the relative proportion in which the two sexes are attacked with Phthisis.

Of the 474 cases from which my tables as to children are drawn up, 309 were male patients and 165 female, a proportion of 65·19 males and 34·81 females per cent., or, again, not a long way from the proper relative proportion between the sexes.

CONCLUSION.

There are, then, two important considerations to be examined with regard to the hereditary tendency to Consumption.

First, the nature of the heredity influence.—Is the inherited taint specific? Most probably it is not. Weakly parents, whatever the cause of their weakliness, are likely

to produce delicate offspring. The special danger of Consumption in the parents depends upon the fact that their weakly children are constantly exposed to infection from their parents. There is thus the double danger—an individual with small resisting power, and constant exposure to infection. If the delicate child is early removed from close association with the consumptive parents, half the danger is removed.

The *second* point is to determine as far as possible the extent of the influence of heredity. Here we have to rely upon statistics, a mode of inquiry which is universally acknowledged to be full of fallacies. When we examine critically into the matter, we find that the evidences of heredity, even in the 30 per cent. of cases in which it is said to exist, are not so clear as would appear to a casual observer; for in some of these cases the parents did not become consumptive until some years after the birth of the patient under notice. Occasionally, also, we may find that a mother becomes consumptive after nursing a consumptive daughter; yet, if some years later the daughter were to mention to a medical adviser, unacquainted with the family, that her mother was consumptive, he might include this case amongst the instances of heredity. It is often difficult or impossible to obtain reliable information as to the date at which other members of a patient's family became consumptive, or of any special causes—occupation, &c.—which may have determined the onset of pulmonary Tuberculosis in certain members of a family. It may be that a man's occupation has been the cause of his becoming consumptive, and one of his sons following him in the business also becomes phthisical, whilst the remainder of the family escape. Here again we might be misled into

believing that we had an instance of heredity. So, too, in tracing heredity, we generally start from phthisical patients and trace their family history backwards. To get a more correct result, we should start with the consumptive and follow the history of succeeding generations. The figures obtained by the first method probably give far too high a proportion in favour of heredity influence. The influence of heredity, if over-estimated, is a source of great anxiety to many who have lost relatives from this disease. It is well that we should do our utmost to teach such persons that the knowledge of the family weakness may serve as a timely warning to prevent them suffering as their relatives have done, and is by no means an indication of impending doom.

Consumption is a preventible disease, and those who know that they have inherited a weakly constitution are most in need of attending to the means of prevention.

The analysis of the above cases warrants the opinion that the hereditary predisposition in the case of Phthisis is not a direct predisposition to this malady, but merely the general predisposition to disease which belongs to the offspring of weakly parents; a predisposition which the children of phthisical parents have in common with those of parents whose health is lowered by other conditions. The families of consumptives have, however, the disadvantage, not shared by the delicate children of non-phthisical parents, of living in constant association with a source of infection. They have a general predisposition to take any disease to the infection of which they may be exposed, and being constantly exposed to the infection of Tuberculosis they are prone to become consumptive. This circumstance may well account for these families showing an

incidence of Phthisis only 9 per cent. in excess of the incidence of the disease amongst the children of non-phthisical but weakly parents.

I believe that the present criticism of the influence of heredity by the examination of the total number of children of one generation in a phthisical family deals with a larger number of cases than has as yet been published.

The results of the examination of the figures may not go very far in deciding any of the various questions which surround or are included in the theory of inheritance in Phthisis. The figures themselves may, however, be of some interest and value even to those who cannot agree with the deductions I have drawn from them.

STATISTICS.

Of 1,000 phthisical patients, 325 gave a history of Phthisis in one or both parents. Apparent heredity = 32·5 per cent. Male patients 604; apparent heredity 28·97. Female patients 396; apparent heredity 37·87.

Table showing the proportion of phthisical children to 100 *non-phthisical in* 80 *cases from Dr. R. Thompson's table of heredity* (*p.* 45 *of his book*), *compared with a similar proportion in* 199 *cases of my own with Phthisis in one or both parents.* (*See totals in the first two Tables.*)

		Phthisical.	Non-phthisical.
Dr. R. Thompson.	Males	116·1	100
	Females	137·14	100
My own cases ...	Males	51·32	100
	Including all infantile deaths	76·10	100
	Females	50·42	100
	Including all infantile deaths	66·6	100

Table showing the number of males and females who developed Phthisis out of 474 *families, in* 199 *of which heredity was suspected* (*d*).

Heredity.			Phthisical.	Died in childhood.	Non-phthisical.
Nil 275	Males	947	244	111	592
	Females...	798	142	82	574
Paternal, 84	Males	267	62	39	166
	Females...	244	76	28	140
Maternal, 82	Males	246	84	33	129
	Females...	260	71	23	166
Double, 33.....	Males	84	28	12	44
	Females...	81	30	6	45
Total of	Males	597	174	84	339
199 hereditary cases	Females...	585	177	57	351

Table showing the number of males and females who developed Phthisis out of 80 *families in whom heredity was suspected* (*from Dr. R. Thompson*) (*e*).

Heredity.			Phthisical.	Died in childhood.	Non-phthisical.
Paternal, 24	Males	39	20	2	17
	Females...	54	30	5	19
Maternal, 30	Males	102	38	9	55
	Females ..	78	34	4	40
Double, 14	Males	34	17	9	8
	Females...	30	17	5	8
Atavism, 12	Males	28	23	1	4
	Females...	20	15	2	3
Total	Males	203	98	21	84
(80 hereditary cases)	Females...	182	96	16	70

(*d*) For comparison with Dr. R. Thompson's Table III, p. 45 of his work.

(*e*) Table III of Dr. R. E. Thompson (p. 45).

CHAPTER VI.

HEREDITY IN PHTHISIS (*continued*).

On December 11th, 1894, I read a paper before the Royal Medical and Chirurgical Society of London on "The Influence of Heredity in Phthisis," in which I compared the incidence of this disease on the children of phthisical parents with that on the children of non-phthisical parents living under similar conditions. In that paper (*a*) I showed that in 275 families, in which there was no Phthisis in the parents, comprising a total of 1,745 children, 24·87 per cent. of these became phthisical; whilst in 199 families of phthisical parents, with a total of 1,182 children, 33·71 per cent. of the children became phthisical. This gives a difference of only about 9 per cent.

I pointed out that this comparatively small excess in the incidence of Phthisis on the offspring of phthisical parents might well be accounted for by the greater exposure to infection of those whose parents were phthisical, since these were living with infective persons.

The general argument was, briefly stated, as follows:—The children of parents who are in poor health—whether from Phthisis, or because of unhygienic surroundings, or from any other cause—are likely to be themselves weakly and thus predisposed to contract any disease to the in-

(*a*) See preceding Chapter.

fection of which they may be exposed. If the ill-health of the parents is due to Phthisis their delicate children, being constantly exposed to the infection of Tuberculosis, are liable to become tubercular. They do not *inherit* Tuberculosis, but are more exposed to infection of this disease than those who do not live with consumptives.

If this view be correct, its importance from the point of view of prophylaxis is considerable.

I afterwards completed the statistical examination of 1,000 families,—a sufficient number to give some value to the result, since these families comprise over 6,400 children. In this Chapter I propose to analyse the figures obtained from the 526 cases tabulated since my first paper was published, and the results of the total of 1,000 families. For the sake of comparison, and to give completeness, I have drawn out Tables (VI. and VII.) showing the results given in the Medico-chirurgical paper side by side with the figures of this second series, and the total for the two series combined. The results obtained from this larger number of cases will be seen to differ but slightly from the figures originally published in 1894.

The influence of heredity in the causation of Tuberculosis, and especially in the causation of Consumption, has been held to be very great. So powerful has this influence been considered that it has been used as an argument against the proposals for prophylactic measures which are advocated by sanitary officers and others. The members of consumptive families also have felt that escape from the disease is well-nigh impossible, and meet suggestions for prevention with the reply, "It is no good my taking precautionary measures, the disease is in my

family." Nevertheless, it seems to me that the facts about heredity in Consumption, properly considered and understood, furnish a strong argument in favour of prophylactic measures against Tuberculosis. I have on previous occasions expressed the belief that the influence of heredity in Tuberculosis has been over-estimated, and that amongst the general public the nature as well as the extent of this influence is misunderstood. It is clearly the duty of the medical profession to educate the public on matters affecting disease-causation, so that preventive means may be accorded the approving help of the public, instead of being met with the obstruction or apathetic indifference with which ignorance receives all prophylactic suggestions and regulations. In Tuberculosis especially is it essential that attempts at prophylaxis should be seconded by the public. Without such co-operation the medical profession can do little or nothing; with the intelligent and willing help of all concerned, Tuberculosis may be practically stamped out, and Consumption become as rare as Small-pox is now. One great obstacle to the general acquiescence in measures directed against the spread of Tuberculosis is the wide-spread belief in the hereditary nature of Consumption. Statistics are proverbially unreliable, and statistical evidence has been used to illustrate the influence of heredity in Consumption. I shall make use of this same uncertain weapon to combat the inferences generally deduced from the statistics of heredity; and if I am told that my figures *prove* nothing I am content to accept this criticism, for my object is in part obtained by showing that statistical evidence—whatever it may be worth—is not without exception in favour

of the strong influence of heredity in Phthisis. With regard to my figures, it may be argued with justice that the information obtained from patients as to the medical history of other members of their families is unreliable. Yet the same sources of fallacy exist in the cases where the parents were consumptive and in those in which the parents were non-phthisical, so that a comparison between the two classes may still be a fair one. It is because this element of uncertainty in the correctness of the medical history makes any pretension to scientific accuracy impossible in such an inquiry as this, that I have preferred to use such indefinite terms as "Consumption" and "Phthisis," rather than the more exact expression "Pulmonary Tuberculosis." Before reviewing the figures which I have to submit let us briefly examine the nature of some of the evidence of hereditary influence. The chief argument in favour of the powerful influence of heredity is that in so many cases of Tuberculosis one or other of the patient's parents suffered from the same disease, or that the disease affected the grand-parents, uncles, aunts, or cousins. In many of the cases where the parents were phthisical it would be found that they did not become so until after the birth of the patient; and in these cases, as in those where the parents were never affected, Atavism is brought forward to explain the hereditary transmission of the taint. Something more, however, than the occurrence of a particular disease in several members of successive generations of a family is required to establish heredity, or we might consider measles an hereditary disease. Congenital Tuberculosis—Tuberculosis in the fœtus or newly-born child—is so rare that the hereditary influence cannot be considered

to take the form of the direct transmission of the disease. To bring the later onset of the disease into line with the theory of heredity, we require to imagine an exceptional period of latency in Tuberculosis, which is still only a supposition. If the disease were implanted in the fœtus, remaining latent until called into activity by some accident, we should not expect it to remain inactive through the period of teething and the various ailments of childhood, only to declare itself in early adult life—the period when Consumption most commonly commences. Evidently then it must be a *tendency* to the disease which is transmitted, not the disease itself; and if we can show—as has frequently been shown—that this tendency may be counteracted, we have a powerful argument in favour of the employment of prophylactic measures. Granting that the members of a consumptive family have a special tendency to Tuberculosis, which tendency can be counteracted and removed by certain means, how can we hesitate to employ these means when the disease to be prevented is so fatal?

But as I attempted to show in the preceding Chapter, it is not certain that any *specific* tendency to Tuberculosis is transmitted. It is possible—and, as I think, probable—that what is transmitted by consumptive parents is a want of strength to resist illness of any kind; a general delicacy of constitution which renders the individual a ready prey to any disease. Such a person living with a consumptive (it may be a parent) is likely to become consumptive, not because of any specific tendency, but because, being constitutionally non-resistant, he contracts the disease to the infection of which he is exposed,—and in this case it is Tuberculosis. Some support for

this contention may be found in comparing the proportion of cases in which heredity is claimed amongst 1,000 hospital patients and amongst 1,000 private patients. In the first Report of the Brompton Consumption Hospital, out of 1,010 patients of the poorer classes, one or both of the parents were phthisical in 242 cases, or 24 per cent. Amongst the private patients (well-to-do persons) of Dr. Theodore Williams—one of the physicians to the Brompton Hospital—1,000 cases gave a history of Phthisis in the parents in 120 instances, or 12 per cent.—only one-half of the proportion found amongst the poorer patients. Either heredity is not a potent influence—otherwise it would affect both classes equally—or the hereditary influence is counteracted by healthy living. Either inference gives hope for preventive medicine.

But even the limited influence of an inherited tendency does not appear to me to be so great as would appear from the statistical expression of heredity in Consumption which is usually employed. It is commonly stated that an hereditary influence may be found in from 25 to 30 per cent. of all cases of Consumption, if inheritance from parents alone is taken into consideration, and in about 60 per cent. when phthisical grand-parents, &c. are considered.

In the paper already referred to, I collected from various sources the following figures:—Out of 12,509 cases of Consumption the parents were also phthisical in 3,101 cases, giving a "percentage heredity" of 24·79. In 12,146 consumptives there was a history of Phthisis in the family (parents, grand-parents, uncles, or aunts) in

7,563, or a "percentage heredity" of 62·34. But in all these cases the figures were arrived at by getting from the patient the history of the preceding generations of the family, and wherever Consumption had occurred in even one member of the family the case was classed as an instance of heredity. We only require to carry the family history back another generation or two to find some "hereditary influence" in almost every case. That this method of inquiry gives a misleading idea of the influence of heredity I endeavoured to demonstrate, and set myself to work out the hereditary influence in a different manner. Starting with the consumptive individual, I traced his offspring instead of his ancestors, and noted how many of his children subsequently became phthisical, and how many remained free from Tuberculosis. This led to a somewhat unexpected result; for the figures by which the percentage heredity were now expressed—viz. 33·71—were closely similar to those obtained by the other method of inquiry (viz. 32·5). We may conclude, therefore, that amongst the class from which these patients are derived—the poorer classes, who make use of hospital assistance—about one-third of the offspring of consumptive parents will subsequently become tubercular, and that in about one-third of the cases of Consumption amongst such classes we shall find a history of Phthisis in the parents. This, however, is far in excess of what Dr. Theodore Williams found amongst his private patients. I then checked my results by an inquiry into the proportion of the offspring of non-phthisical parents, in the same condition of life as my other cases (*i.e.* hospital patients), who eventually became consumptive. In this way I

obtained a numerical expression of the influence of occupation and mode of life, for in these cases the influence of heredity was absent. Clearly, also, the influence of the consumptive parent in the causation of Phthisis in his offspring (whether this influence be hereditarily transmitted or not) would be expressed by the increased incidence of Consumption on the offspring of phthisical parents over that on the offspring of non-phthisical parents, all the cases being drawn from families in the same station of life. This is shown in the following Table :—

TABLE IV.

Showing the incidence of Phthisis in the Children of non-phthisical and of phthisical Parents respectively.

1,000 Families.	Children.		Died in Infancy.		Phthisical.	
	Total No.	Proportion to each family.	Total No.	Per cent. of whole.	Total No.	Per cent. of children, omitting infantile deaths.
A. 673 families. With *no* Phthisis in the parents.	4,488	6·66	522	11·63	938	23·65
B. 143 families. Father phthisical	925	6·25	126	13·62	249	31·16
C. 135 families. Mother phthisical	826	6·11	78	9·44	245	32·75
D. 44 families. Both parents phthisical	218	4·95	28	12·84	82	43 15
Summary of B, C, D. 327 families, one or both parents phthisical	1,969	6·02	232	11·78	576	33·16

As will be seen from these results of inquiries embracing 1,000 families, this Table shows—

Percentage of the offspring of *phthisical* parents subsequently becoming tubercular	33·16
Percentage of the offspring of *non-phthisical* parents subsequently becoming tubercular	23·65
Percentage influence due to parents, or "percentage heredity" . . .	9·51

We may then take it as demonstrated that the effect of consumptive parentage affects less than 10 per cent. of the offspring (*a*).

But how much of this comparatively small excess of Phthisis in the offspring of consumptives may properly be put down to heredity is still open to question. We know that Tuberculosis depends upon the presence within the body of the Bacillus tuberculosis; and whilst the direct transmission of the micro-organism from the parent to the *fœtus in utero* is almost unknown, there is abundant evidence that the bacillus may gain entrance into the body from without, in the air and in the food as well as in other ways. Probably the great majority of persons now hold the opinion that infection is the chief factor in the causation of Tuberculosis. I would express the matter by stating that infection is the only essential factor in the causation of Tuberculosis: but though the most robust may become tubercular if exposed for some time to intense infection, there are certain conditions which render a person liable to become infected from even a slight

(*a*) It is interesting to note how nearly this agrees with the "percentage heredity" given by Dr. Theodore Williams for his private patients, who were free from the adverse influences of occupation and surroundings which affect the hospital patients.

degree of infection. Of these predisposing causes, inherited weakliness of constitution is one. That such inherited predisposition may be counteracted is shown by many well-known cases—such as the family recorded by Dr. Hermann Weber, in the Croonian Lectures for 1885—and is also evidenced by the fact that two-thirds of the offspring of consumptives escape the disease. But when an individual with inherited delicacy is living in close and intimate relation with a consumptive, there is so great a risk of infection that the extra 10 per cent. of

TABLE V.

Showing the incidence of Phthisis on the two Sexes in the Children of non-phthisical and of phthisical Parents respectively.

1,000 FAMILIES.	CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
	Total No.	Proportion to each family.	Total No.	Per cent. of whole.	Total No.	Per cent. of children, omitting infantile deaths.
A. 673 families. With *no* Phthisis in the parents	M. 2,448	3·63	310	12·66	565	26·24
	F. 2,040	3·03	212	10·39	373	20·40
B. 148 families. Father phthisical	M. 475	3·20	69	14·52	110	27·09
	F. 450	3·04	57	12·66	139	35·36
C. 135 families. Mother phthisical	M. 399*	(2·95)	41*	(10·27)	126	35·19
	F. 420*	(3·11)	30*	(7·14)	119	30·51
D. 44 families. Both parents phthisical	M. 108	2·45	15	13·88	40	43·01
	F. 110	2·50	13	11·81	42	43·29
Summary of B, C, D. 327 families, one or both parents phthisical.	M. 982*	(3·00)	125*	(12·72)	276	32·20
	F. 980*	(2·99)	100*	(10·20)	300	34·09

* In the other seven, the sex was not stated.

consumptives in the offspring of tubercular parents—as shown in my figures—may easily be thus accounted for.

When we examine the incidence of the disease on the two sexes under the various circumstances (Table V.), we see further reason to attribute the excess of Consumption in the offspring of phthisical parents not to heredity but to infection. Amongst the offspring of the non-phthisical parents it is the males who become consumptive in greater proportion. In this we may see the influence of occupation, and the greater risk of infection to those who go out into the world over those who remain at home. When, however, there is Phthisis in one or both of the parents, we see the extra risk to those who remain at home shut up, so to speak, with a consumptive. It is amongst the female offspring of phthisical parents that the disease shows itself in greater proportion, the exposure to infection at home outweighing the risks of occupation and exposure at work. We note that the incidence of Phthisis amongst the male offspring of phthisical parents (32·2 per cent.) is about 6 per cent. more than that amongst the male offspring of non-phthisical parents (26·4 per cent.); whilst amongst the female offspring nearly 14 per cent. more become consumptive amongst those belonging to phthisical families (34·09 per cent.) than amongst the children of non-phthisical parents (20·4 per cent.). Does not this furnish a strong argument in favour of infection, rather than heredity, as the means of transmission from parent to child? The consumptive father goes out to his work during the earlier part of his illness, and is, therefore, not so much in contact with the other members of the family.

The mother is always at home, and so becomes a constant source of infection to the family from beginning to end of her illness. The effect of this is seen in the increased incidence of Consumption when the mother is phthisical. When both parents are consumptive the risk of infection is materially increased, and the proportion of the offspring who become phthisical is large. Not only is infection intense, but the non-resistance of the offspring is greater when both parents were weakly.

There are many other interesting deductions which might be made from these figures, but I am unwilling to obscure my main argument by following up side issues. There is, too, a danger of utilising figures to prove preconceived theories. I commenced this inquiry without any special theory of the hereditary influence in Phthisis, and was led to question the potency of direct heredity by my figures. I am therefore the more anxious to avoid the attempt to make my figures prove too much.

I am content to claim that—as shown by the small difference between the incidence of Consumption on the offspring of phthisical and non-phthisical parents respectively—the direct influence of heredity is comparatively small, and is, at any rate, considerably less than has been generally believed. If this contention is accepted, I shall have the satisfaction of having done something towards removing one great argument against the practical utility and probability of success in prophylactic measures directed against Tuberculosis. Once let a parent recognise that it is by *infection*, and not by *inheritance*, that he may be a danger to his children, and it will not be difficult to persuade him to take precautions against spreading the

disease. Thus prophylactic measures will be easily introduced into the household, where sanitary inspectors cannot penetrate.

The following tables permit of comparison between the results obtained in the two series of observations, which form the basis of the comments in the two preceding Chapters.

TABLE VI.

1,000 Families.		Children.		Died in Infancy.		Phthisical.	
		Total No.	Proportion to each family.	Total No.	Per cent. of whole.	Total No.	Per cent. of children, omitting infantile deaths.
A. Parents non-phthisical.							
Med. Chir.....	275	1,745	6·34	193	11·06	386	24·87
Second series	398	2,743	6·89	329	11·99	552	22·86
Total.....	673	4,488	6·66	522	11·63	938	23·65
B. Father phthisical.							
Med. Chir.	84	511	6·08	67	13·11	138	31·08
Second series	64	414	6·46	59	14·25	111	31·26
Total.....	148	925	6·25	126	13·62	249	31·15
C. Mother phthisical.							
Med. Chir.....	82	506	6·17	56	11·06	155	34·44
Second series	53	320	6·03	22	6·87	90	30·20
Total.....	135	826	6·11	78	9·44	245	32·75
D. Both parents phthisical.							
Med. Chir.....	33	165	5·00	18	10·90	58	39·45
Second series	11	53	4·81	10	18·86	24	55·81
Total.....	44	218	4·95	28	12·84	82	43·15
Summary of B, C, D.							
Med. Chir.....	199	1,182	5·93	141	11·92	351	33·71
Second series	128	787	6·14	91	11·56	225	32·32
Total.....	327	1,969	6·02	232	11·78	576	33·16

Table VII.

1,000 Families.		Children.		Died in Infancy.		Phthisical.	
		Total No.	Proportion to each family.	Total No.	Per cent. of whole.	Total No.	Per cent. of children, omitting infantile deaths.
A. Parents non-phthisical.							
Med. Chir.	275	M. 947	3·44	111	11·72	244	29·06
		F. 798	2·90	82	10·41	142	19·83
Second series ..	398	M. 1,501	3·77	199	13·25	321	24·65
		F. 1,242	3·13	130	10·46	231	20·77
Total.....	673	M. 2,448	3·63	310	12·66	565	26·42
		F. 2,040	3·03	212	10·39	373	20·40
B. Father phthisical.							
Med. Chir.	84	M. 267	3·17	39	14·60	62	27·19
		F. 244	2·90	28	11·47	76	35·18
Second series ..	64	M. 208	3·25	30	14·42	48	26·96
		F. 206	3·21	29	14·07	63	35·59
Total.....	148	M. 475	3·20	69	14·52	110	27·09
		F. 450	3·04	57	12·66	139	35·36
C. Mother phthisical.							
Med. Chir.	82	M. 246	3·00	33	13·41	84	39·43
		F. 260	3·17	23	8·84	71	29 95
Second series ..	53	M. 153*	(2·88)	8*	(5·22)	42	28·96
		F. 160*	(3·01)	7*	(4·37)	48	31·37
Total.....	135	M. 399*	(2·95)	41*	(10·27)	126	35·19
		F. 420*	(3·11)	30*	(7·14)	119	30·51
D. Both parents phthisical.							
Med. Chir.	33	M. 84	2·54	12	14·28	28	38·88
		F. 81	2·45	6	7·40	30	40·00
Second series ..	11	M. 24	2·18	3	12·50	12	57·14
		F. 29	2·63	7	24·13	12	54·54
Total.....	44	M. 108	2·45	15	13·88	40	43·01
		F. 110	2·50	13	11·81	42	43·29

* In the other seven, the sex was not stated.

TABLE VII.—*continued.*

1,000 FAMILIES.		CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
		Total No.	Proportion to each family.	Total No.	Per cent. of whole.	Total No.	Per cent. of children, omitting infantile deaths.
Summary of B, C, and D.							
Med. Chir. 199	M.	597	3·00	84	14·07	174	33·91
	F.	585	2·93	57	9·74	177	33·52
Second series .. 128	M.	385*	(3·00)	41*	(10·64)	102	29·65
	F.	395*	(3·08)	43*	(10·88)	123	34·94
Total..... 327	M.	982*	(3·00)	125*	(12·72)	276	32·20
	F.	980*	(2·99)	100*	(10·20)	300	34·09

* In the other seven, the sex was not stated.

Part II.

THE PREVENTION OF CONSUMPTION.

CHAPTER VII.

THE PREVENTION OF TUBERCULOSIS.

The main object of our Profession and practice is to avert the evils which accompany or result from disease. When illness has attacked the individual our efforts in this direction can be but partially successful, and they too often fail entirely.

Only by *preventing* disease can we achieve complete success. The schedule of preventible diseases increases with our fuller study of disease-causation, and amongst the more recent triumphs of medical science is the accumulation of evidence showing that Tuberculosis depends upon causes which are controllable, and that one of the most fatal of all diseases may be prevented. But in Tuberculosis we have a long-continued illness, and we cannot rely upon those measures—especially isolation of the sick—which are more easily and successfully applied to the prevention of infectious disease of short duration, such as the exanthemata. Tuberculosis attacks animals and may be transmitted from them to the human subject; preventive measures must therefore be of wider application than would be necessary for a disease which is chiefly or entirely conveyed from man to man. The co-operation of all branches of our profession is, in fact, needed in combatting Tuberculosis. Speaking as one whose interests are mainly clinical, I shall here give chief attention to

the part that the practitioner of medicine may take in the prevention of Tuberculosis, rather than to that which is especially the province of the Medical Officer of Health. It is the prevention of disease in the individual, rather than in the community, which comes within the sphere of the work of the medical practitioner. Since, however, I am about to discuss the *General* Preventive Treatment, I feel that I must indicate the provisions which are necessary from the Public Health point of view as well as the precautions which it is our duty to advise in the family; and with so wide a range of subject I can merely sketch a bare outline, leaving the detail to be filled in from other sources.

For all communicable diseases, of which Tuberculosis is one, prophylactic measures to be successful must be based upon our knowledge of the following points:—

1. The nature of the infective agent:
2. The ways in which the infective material is given off from the sick:
3. The modes by which this is conveyed to others:
4. The paths of entry into the body:
5. The conditions within the body which favour or prevent development of the disease.

Let us briefly review our knowledge on these points with regard to Tuberculosis, and thus see in what way prevention may be possible.

1. The Nature of the Infective Agent.—For our immediate purpose there are two facts of chief importance:

A. The infective agent is *particulate*, and will therefore tend to settle from the air like dust.

B. It is *living*, and can therefore be killed; but if conditions be favourable to its development it is capable of multiplication and increase.

There is still room for observation as to the conditions under which the Bacillus tuberculosis may grow *outside* the body, but there are facts which tend to show that the bacillus may thrive in the dust of dark corners, though exposure to sunlight will prevent its development.

The observations of Dr. Ransome as to tuberculous houses and rooms are of great value, and we cannot ignore the importance of cleansing or disinfecting rooms which have been long occupied by a consumptive individual, especially amongst those classes of the community whose habits are somewhat primitive. Some such precaution would seem to be equally desirable in the lodging-houses and hotels of certain health-resorts. It is—as I have on more than one occasion insisted—in the destruction of the tubercle bacilli outside the body that we shall find the chief use of disinfectants (germicides) in the control of Tuberculosis.

Whatever the facts may be as to the development of the bacilli outside the body, there is no room for doubt that the bacillus multiplies within the body of the tubercular man or animal. Every case of Tuberculosis thus furnishes a further supply of infective material, and the longer the disease is actively progressing the greater will be the increase in the fresh brood of infective bacilli. Thus early treatment, to check if possible the disease at its outset, must be ranked amongst our prophylactic measures. I would here incidentally remark that treatment at the outset of the disease—when alone a cure is

to be anticipated—presupposes early identification of the disease; and that failure to detect pulmonary Tuberculosis sufficiently early is, in my experience, more often due to neglect of full examination of the chest, than to absence of clear physical signs of disease. I have in several cases seen pulmonary Tuberculosis undetected because a *bruit de diable* in the neck seemed to confirm the opinion that anæmia was the cause of the patient's ill-health, and further examination was consequently omitted.

But whilst early diagnosis is to be followed by attempts to cure in human beings, in animals—and especially the domestic animals—early detection of Tuberculosis should be followed by timely destruction of the diseased animal. Professor Sims Woodhead tells us that, though cats are not particularly susceptible to tubercular infection, yet when they become affected they develop immense numbers of bacilli in the lesions.

We cannot afford to foster a tubercle bacillus factory in the household.

2. The ways in which the Infective Material is given off from the affected.—In different cases of Tuberculosis bacilli have been found in nearly all the secretions and fluids of the body, and may therefore leave the body by any of the excretory organs. Bacilli have been found in the seminal fluid and in vaginal secretions; they may be present in pleural exudations and in the pus of tubercular abscesses; they are to be found in the lymphatic glands, serous membranes, and occasionally in the flesh of tubercular animals. All these may, even if only rarely, convey infection.

There are, however, two chief ways in which the bacilli are given off from the body :

A. In the expectoration when the lungs are affected.
B. In the milk when the disease is more generalised.

A. It is by the sputa of consumptives that pulmonary Tuberculosis is most generally disseminated, and it is in advising the phthisical patient as to the disposal of the expectoration that the medical practitioner can do much towards the prevention of the spread of Tuberculosis. The sputum should not be expectorated anywhere where it may become dried and so liable to be pulverised.

In the house a spitting cup, or other suitable vessel, containing liquid—whether a disinfectant or only water matters little—should always be used to receive the sputa. This should be emptied on to a hot fire, or the contents be well mixed with some germicide solution before being thrown down the drains. Out of doors and wherever the spittoon cannot be used, pieces of linen, or Japanese paper handkerchiefs, should be used to receive the sputa, and at once put into a metal box carried for the purpose until they can be burned, which should be as soon as possible. If the ordinary handkerchief is used, it should be put in a disinfectant solution before the sputum dries, and be allowed to soak until it is sent to be washed. As, with a violent cough, minute particles of phlegm are usually expelled, it is advisable for the patient to hold a handkerchief before his face during coughing. Consumptives should be warned against swallowing their sputa, for by this they risk auto-infection through the alimentary

canal, with increased suffering to themselves, and with greater danger to others from the multiplication of bacilli in each new tubercular focus.

B. As tubercle bacilli are to be found in the milk, tubercular mothers should not be permitted to suckle their infants.

Probably much of the Tuberculosis amongst young children, especially in towns, is due to infection from the milk of tubercular cows. The efforts of veterinary surgeons and Medical Officers of Health are being directed to the stamping out of Tuberculosis amongst cattle used for dairy purposes, and to preventing the sale of infective milk. Cleanliness and ventilation of sheds, removal and destruction of tubercular animals, and inspection and examination of milk supplies are the main means to these ends. But so long as Tuberculosis is common amongst cattle we must seek to protect the individual by boiling or otherwise sterilizing cow's milk before it is taken as food. This is especially necessary with the milk used as food for young children, and imperative where the child is constitutionally predisposed to Tuberculosis.

3. The Modes by which the Infection is conveyed from the sick to the susceptible is sufficiently indicated by what has been said as to the sources of infection. Air and food (especially milk) are the two main carriers of the infective agent.

Fomites are not so largely concerned in the distribution of Tuberculosis as is the case with some of the exanthemata: but we must bear in mind that handkerchiefs

which have been used to receive the sputa, bandages, &c. soiled by the discharges from tubercular abscesses, &c., may carry infection, and should therefore be at once disinfected or burned.

4. **The paths of entry of the bacilli into the body** require some examination.

The bacillus may be implanted in the body before birth; but congenital Tuberculosis is rare.

Direct inoculation by cuts and wounds is possible, and cases are recorded where this has occurred from cuts with broken spittoons, and in the Jewish rite of circumcision. In these cases the disease often remains localised near the seat of inoculation. The cases, not infrequent, where a tubercular husband has apparently infected his wife, or the wife her husband, point to the inadvisability of the marriage of phthisical individuals, and suggest the precaution for those already married of occupying separate beds.

The two most frequent paths of entry are, however, by the air-passages and by the alimentary tract. It is possible that with a large dose of the poison, or when this is very virulent, the bacilli may cause infection through undamaged mucous membrane, but undoubtedly the danger is increased when the endothelial surface is injured or inflamed. The fauces and pharynx may be exposed to infection from either air or food, and there are many reasons for suggesting that the entry of tubercle bacilli frequently takes place in this region. Professor Sims Woodhead has drawn attention to infection by the tonsils

not only as a cause of strumous glands in the neck, but also perhaps leading to tubercular meningitis. In some experiments which I carried out some years ago, I damaged the mucous membrane of the throat in rabbits by the repeated application of a weak solution of iodine and then sprayed some tubercular liquid into the mouth. Tubercle bacilli were afterwards found in the cervical lymphatic glands. I would point out the possible danger in this connection of attacks of tonsillitis and relaxed throat. Further I would suggest that there may be temporarily increased danger of infection in those whose tonsils have been removed, and in those who have been operated upon for adenoids. With regard to the latter I have grounds for suggesting that, occasionally at least, adenoids may be the result of tubercular infection; but my inquiries in this direction are not yet sufficiently complete to enable me to be more definite.

5. Predisposition.—In the preceding section I have indicated that damage to the mucous membrane favours the invasion of the bacillus, and thus produces a local predisposition to Tuberculosis. There may also be some general or constitutional condition of health which renders an individual liable to suffer from infection. General predisposition may be inherited or acquired; local predisposition is usually acquired. Preventive measures must embrace the causes which predispose to Tuberculosis, and must aim at avoiding or removing these. It is in this part of the general scheme of prophylaxis that the medical practitioner has so great a share to perform.

The question of hereditary predisposition is here im-

portant. I suppose the popular and usual opinion is that one could hardly note the way in which Consumption attacks certain families without recognising some potent hereditary influence. Granting this, I do not place hereditary influence in the fore-front of predisposing causes.

My view—expressed before the Royal Medical and Chirurgical Society in 1894—is that the offspring of parents who are constitutionally unsound, whether from Tuberculosis or other cause, possess less resisting power against disease of all kinds, including Tuberculosis. In 1,000 families from about the same station in life, I found there was only an excess of 9 per cent. in the incidence of Tuberculosis on the children of phthisical parents over that on the children of non-phthisical parents, and that this excess might be explained by the former living with their consumptive parents—*i.e.*, with an infective individual. The fact that the daughters—who remain longer at home—were chiefly attacked in these families, whilst the sons were more often affected in those families where the parents were non-phthisical, gives support to this view.

But whatever opinions we may hold on this point, we shall all agree that it is only a *predisposition* which is inherited and not the actual disease.

If this predisposition can be corrected, or sources of infection avoided, Tuberculosis may be prevented. The children of consumptive parents should be kept from too close proximity with such parents. If it is the mother who is tubercular, the infant must not be suckled by her, and in any case the children should not share the parent's bed. To remove the predisposing constitutional weakness we must see that early life is passed under the most

favourable hygienic conditions. We must give attention to the feeding and clothing of the infant; the hygiene of the nursery, especially as regards ventilation and sunlight; the food, exercise, and hours of work of the schoolboy; the selection of occupation and place of residence for the adult.

Acquired general predisposition may result from severe or prolonged illness, depressing emotions, worry, or overwork and unhygienic surroundings. Hysteria and long-continued anæmia may pave the way for Tuberculosis. Measles and influenza are frequent predisposing causes, and in these ailments there is often local predisposition produced in the lungs, in addition to the general want of resisting power. Where full return to health is delayed after these illnesses it is advisable to examine the chest carefully.

I have elsewhere drawn attention to the "pulmonary affections which may lead to Phthisis," such as bronchitis and pneumonia, and have already mentioned some local predisposing conditions in the fauces and pharynx. We are all aware that some strain or other slight injury generally precedes Tuberculosis of a joint. Recognising the conditions which render an individual specially liable to suffer from tubercular infection, we should be on the alert for the first warning of disease in those who are thus predisposed, and by early treatment we shall be able not only to save our patient much suffering but to prevent much danger to others.

In conclusion I must briefly refer to the vexed question of **Notification.**

On theoretical grounds I have no doubt that notification is desirable; as I suggested before the International Congress of Hygiene in 1891, it is in the application of the principle that difficulties arise. Instead of the rigid quarantine which was formerly imposed on those arriving from infected ports in the case of cholera and other diseases, we now allow the travellers to land and proceed to their destination. But a registration of such persons allows of a certain supervision, until the expiration of the incubation period allays anxiety or the occurrence of the disease necessitates special precautionary measures. So with the consumptive or other tubercular individual, notification and registration would render supervision possible. Where the circumstances and habits of the patient will ensure the advice of a competent medical man, and the probability that his directions would be carefully carried out, no interference by the Medical Officer of Health would be necessary, and the person would not feel any disadvantage from a supervision which was non-obtrusive. But all experience shows that amongst the poorer members of society, whose education does not help them to realise the importance of preventive precautions, whose habits are at variance with some of the prophylactic requirements and whose means preclude the provision of others, more active supervision is essential if preventive measures are to be seriously attempted. Whilst work is still possible, such supervision might be sufficient; but with the advanced condition of disease, when work is impossible and when also the danger to others is greatest, removal to a hospital would be better for the patients and better for their

families. For this purpose more special hospital accommodation would be needed than is now available.

I say nothing here about sanatoria—which would only receive the earlier cases, when cure is possible. But the provision of such sanatoria cannot be omitted from any complete scheme for the prevention of Tuberculosis.

CHAPTER VIII.

TO WHAT EXTENT CAN LEGISLATION ASSIST IN DIMINISHING THE PREVALENCE OF CONSUMPTION AND OTHER TUBERCULAR DISEASES?

This title opens out a wide field for consideration, but it will only be possible to indicate the main points at which preventive legislation may be applied.

Some Continental nations, Italy for example, have for years included in their Sanitary Codes certain regulations specially directed against Consumption, a chief provision of which deals with the disinfection of rooms in which phthisical patients have died. Such regulations founded only on an uncertain belief of the infectiousness of Consumption, and not being based on a scientific knowledge of the nature of the tubercular condition, are but crude efforts, and cannot be taken as the basis of preventive legislation. It would be difficult to show that they have had any appreciable effect on the prevalence of Consumption in those countries where they have been in force, and they certainly have not resulted in any special immunity of such countries from tubercular diseases.

With the more exact knowledge of the nature of Consumption and other manifestations of the tubercular process, which resulted from the discovery of the *Bacillus*

tuberculosis, and with the experimental proofs of the transmissibility of tubercle by inoculation and through the injection of tubercular flesh or milk, as well as by the respiration of infected air, we have now some scientific basis on which to construct preventive measures; but beyond this knowledge of the active agent of Tuberculosis, and of modes by which it may gain entrance into the body, we recognise certain conditions of lowered vitality or the like—either of the individual or of certain organs and tissues,—without which exposure to the infective agent may lead to no ill effects, but which, when present, produce in the person a susceptibility or predisposition to receive the infection and develop tubercle. Thus, although we may acknowledge that infection—the inception of the specific micro-organism—is the necessary and essential factor for the development of tubercle in the individual, we recognise that the power for harm of the infective material is so largely influenced by the health of the person exposed to its attack that the predisposing causes assume almost greater importance, from the point of view of preventive measures, than does the mode of infection.

Yet the virulence of the infection may be so increased by concentration of the poison that a robust constitution may be unable to withstand it; and, therefore, whilst we endeavour to remove the conditions which tend to produce a lowered vitality in a community, or a damaged state of special organs rendering them more susceptible to infection, we cannot neglect precautions directed against the concentration of infection, and something may be done to prevent the wide-spread dissemination of infection,

whether from consumptive persons or tubercular animals.

In a former Chapter on the prevention of Phthisis I endeavoured to point out all the various ways in which the infection of tubercle might gain entrance into the body, but for our present purpose we may confine our attention to two chief causes of infection, viz., infected air and infected food. Both these direct causes of tubercular infection are to some extent controllable, and we will discuss them before turning to the consideration of the control which may be exercised upon the conditions which predispose communities or individuals to suffer from these causes.

Although it is probable that air tainted by the emanations from phthisical patients is the commonest infecting cause, it would be of little value to suggest enactments on the same lines as those directed against the acute infectious fevers. This disease is infectious, as far as we know to the contrary, throughout the whole of its often prolonged course, and, if isolation of the consumptive patient were attempted, either we should make his position as bad as that of the leper of old, or more probably we should not have his condition notified until death seemed imminent. The difficulty of recognising with certainty the early stages of pulmonary tubercle would form a ready excuse for neglect of notification until the patient had been poisoning the air for many weeks or months.

And yet there might be great advantage in requiring that a case of Tuberculosis should, under certain conditions, be notified to the health officer. The danger of all infection increases with the close crowding together of sick and

healthy, and with deficient ventilation. Thus the dangers are greatest in tenement houses, single-room dwellings, or common lodging-houses. All these, being for many reasons well known to the sanitarian possible dangers to a community, should be under inspection and control. To existing regulations for such places it might be well to add provision for the notification of illness which keeps the patient confined to bed, or incapacitated from work beyond a certain time (say one, two, or more weeks), requiring in this, as in other cases, medical evidence as to the nature of the disease. When the illness is tubercular Phthisis, it might be advisable to effect the removal of the patient when the sick-room serves also as the living room of several susceptible persons; or in any case to direct the disinfection of sputa, attention to free ventilation, and the efficient fumigation of the room after removal or death of the patient. Similar precautions might be necessary in hotels and lodging-houses, especially at health resorts. The removal of phthisical patients would necessitate provision for their reception, and the present hospital accommodation is obviously insufficient for the purpose. Nor are the existing Consumption hospitals intended for the reception of phthisical invalids in an advanced condition of the disease. These hospitals are mainly provided in the hope of curing or checking the disease, and so enabling the patients to resume their places amongst the workers. Homes for incurable consumptives are few, and not always utilised for their intended purpose. A sanitary district should provide a home for its own advanced consumptives, where the sufferers should be received without thereby incurring the brand of pauperism; these invalids

would thus be prevented from being unwilling dangers to others. In such homes, as in Consumption hospitals, disinfection of all sputa should be strictly attended to.

When we come to the possible dissemination of tubercle through infected food, it becomes extremely difficult to suggest a limit for preventive legislation. If all cattle or other animals which are tubercular in any degree, are liable to seizure and destruction, much loss may be incurred, and the loss must fall on the community, or on the particular owner. It can hardly be proposed to compensate owners for all animals so destroyed. Even partial compensation would entail severe loss on owners, as well as great expense to the nation. If in any case we can contemplate a compensation for such animals, it would be in the case of owners who gave early notice of any suspected animal; it might then serve as an incentive to care.

No compensation should be allowed where a tubercular animal had been slaughtered for food, or the flesh exposed for sale.

But the chief aim of legislation should be the prevention of Tuberculosis in cattle, rather than the destruction of tubercular flesh or milk. More attention to cubic space and ventilation in the sheds, and the early isolation of suspected animals, would probably produce far more important results than the most rigid inspection of meat and milk. Here also inspection of the sanitary circumstances of the animals by competent men is essential, for monetary considerations outweigh regard for the public health with the majority of private persons. This necessitates public expenditure, but the public contributions cannot be better

expended than in preserving the public health. Tuberculosis should no longer be kept out of the list of diseases of animals which must be notified (as in the Contagious Diseases (Animals) Act).

We are still unable to control the possible *importation* of tubercular food, for unless tubercular nodules are found in the flesh the meat cannot be seized, and there is no ready means of knowing that the animal was free from tubercle in the absence of the viscera and membranes. If cattle kept for food were subject to frequent inspection, and suspected animals were isolated, it would not be difficult to prevent the sale of tubercular milk, or at least to prevent its being sold until it had been boiled. The milk from tubercular cows should not be used, even as food for pigs, until boiling had rendered it harmless. Tuberculosis is said to be more common in cattle destined for food than in the general bovine population (*a*), and we may surely refer this to the conditions under which the cattle for food are kept.

But the most promising field for preventive legislation is to be found in endeavouring to combat the predisposing causes of tubercular diseases in a community. We have abundant evidence of the influence of over-crowding, of dampness of the subsoil, and of dusty occupations in determining the incidence of tubercular diseases in a community.

In large towns we have a general over-crowding of the population on a confined area, and in special parts of such

(*a*) *Influence of Heredity and Contagion in the Propagation of Tuberculosis.* Fleming. London.

towns there is a more localised and intense over-crowding of. the poorer inhabitants. Consumption and other tubercular diseases are more frequent in large towns than in more sparsely populated localities, and in the towns it is the densely inhabited, poorer districts, which suffer most. The general over-crowding incident to all towns may be regulated and kept within the requirements of health by enactments regulating the width of streets, the height of houses, and the provision for open spaces, such as are to be found in the Model Byelaws of the Local Government Board. But such enactments should be universally enforced by general legislation, and not dependent for their adoption on the wisdom or fancies of local bodies, whose anxiety for the public weal is sometimes modified by more personal considerations of commercial expediency. However well the construction and surroundings of the houses may have been planned, they may be rendered unhealthy by the over-crowding of the inmates. Sufficient air-space for the inhabitants is more likely to be found in the houses of the well-to-do than in the dwellings of those to whom the question of rent is of more pressing importance than the sanitary condition of the rooms.

When several members of a family work at home, the air of the rooms never gets properly changed, and all evil effects of over-crowding become aggravated.

Where a house is occupied by more than one family, and the owner has a pecuniary interest in packing as many individuals into the house as it can contain, inspection is required in the interests of the lodgers.

Thus, tenement houses should be licensed to contain a certain maximum number of inmates only, and similar

provisions might with advantage be made for ordinary houses let in lodgings, and, perhaps, also for hotels and flats. Such regulations would entail inspection of such houses to prevent infringement of the law. The Local Government Board already has the power to enforce some such regulations in the case of sub-let houses; why should there be any longer delay in taking advantage of it? These regulations also should be general and not merely permissive. Common lodging-houses are already under regulations.

Where a trade is carried on in the house inspection within business hours should be provided, and no trade should be permitted to be carried on in any single-room tenement occupied by more than two persons, and then only if the room is above a certain size. To provide for the workers thus prevented from pursuing their occupation at home, public work-rooms could be provided, where space and facilities for the various occupations (*e.g.*, tailoring, bootmaking, &c.) could be obtained for a nominal payment. Public wash-houses have diminished the periodical using of the dwelling-room as a laundry and drying-room. Certain trades might be scheduled (as dangerous or offensive) which should on no account be permitted in rooms used as sleeping rooms. Again, to be effective, any such regulations must be universal throughout the kingdom, at least in the towns.

If nothing further were done than to apply throughout the country the byelaws and regulations which are now permissive, much benefit would result to the public health, and something would be done to diminish the prevalence of tubercular diseases. We might contemplate with less

regret the present impossibility of dealing with hereditary Tuberculosis, the marriage of tubercular persons, and the suckling of infants by tubercular mothers, if we could by such means as have been indicated diminish the risk of acquiring tubercular diseases; for healthy parents cannot produce tubercular offspring, and healthy children growing up under more favourable hygienic conditions have not the same susceptibility to infection.

The physical training of children in the open air is already cared for in Board schools, and is of vast importance. But healthy recreation for the parents when the day's work is over is not so easily obtained, and perhaps we must look to the efforts of philanthropists to provide inducements to the mother to leave her close room for an hour or two in the evening, and to keep the father from the public-house.

For those who work at dusty occupations protective measures have already done much, but even they would be less liable to Consumption if the infectious particles were not ever present, or if the sources of infection—their consumptive neighbours—were fewer in number.

The question of preventive legislation directed towards the diminution of tubercular diseases is not one to be put aside because of its difficulties, though they are many and great. Where the national good is concerned, means have always been found to grapple with and overcome even greater difficulties than present themselves here.

CHAPTER IX.

THE PREVENTION OF CONSUMPTION (*continued*).

THE broad principles which must underlie all measures for the prevention of Tuberculosis, and particularly for the prevention of Consumption, should by this time be thoroughly well known, since they have been constantly before the profession and the public for the past ten years. They consist (1) in attention to general hygienic rules to counteract any constitutional predisposition, and to lessen the risk of infection; and (2) in special measures to diminish the spread of infection from affected individuals or tubercular animals. Of the latter, two stand out with special prominence, viz., the disinfection of the sputa of consumptive persons, and the sterilisation of the milk of tubercular cows when this cannot be altogether excluded from the market. But before these prophylactic means can be adopted with any practical result, there are many details demanding consideration.

Taking the disinfection of the sputa first, we are met with the initial difficulty that, as Phthisis is often a long-continued malady which does not entirely incapacitate the sufferer, all cases are not under supervision; the sources of infection are not under control. The habit of expectorating is common amongst certain classes in this country, and amongst all classes in certain nations. If those who

are well expectorate without restraint and in all places, how can we control the indiscriminate spitting of those whose ailment demands expectoration?

In a paper on the "Prophylaxis of Tuberculosis" by Professor Landouzy, read before the Académie de Médecine (*La Presse Médicale*, June 11, 1898, p. 222), we have an authoritative article on spittoons, their form, their contents, and the positions in which they should be placed. The subject is important, no doubt, but those who spit from mere habit will not trouble to use spittoons, and those who really need them should provide themselves with receptacles for expectorated matters and have these always at hand. Professor Nocard, in his presidential address before the recent Congress of Tuberculosis in Paris, thought that the provision of hygienic spittoons in all public places, with a conspicuous notice forbidding people to spit anywhere else, would gradually reform the habits of the people in respect to indiscriminate expectoration.

The disinfection of all places in which tubercular patients have lived is another useful suggestion, which is really required to be carried out if prophylactic regulations are to be enforced. But what about the disinfection of public buildings, lavatories, and vehicles, which are often fouled by the expectoration of persons, some of whom may be tubercular? When the consumptive is under supervision, whether in a hospital, sanatorium, or other institution, it is possible—at least, theoretically—to collect and disinfect all the sputa. It follows that the provision of suitable institutions for the care of consumptives forms an important portion of any scheme for prophylactic regulations in a community.

The greater part of the preventive legislation hitherto recommended has reference to the food supply in relation to Tuberculosis, and it is this branch of Prophylaxis which has lately attracted most attention from the majority of Medical Officers of Health who take special interest in Tuberculosis. This may to some extent be due to a feeling that the difficulties in the way of applying even a modification of our present legislation against infectious diseases to sufferers from Consumption are almost insuperable. We have, however, the experience of the Health Department of New York, where notification of Tuberculosis has been in force for some five years, during which time the death-rate from all tubercular diseases in that city has diminished from 3·51 to 2·85.

The Compulsory Notification of Phthisis.—I have seen no reason to alter the opinion, which I expressed in my paper before the International Congress of Hygiene in 1891, that the notification of Consumption is desirable in the interests both of the patient and of the community. Much discretion would no doubt be required as to the action, if any, to be taken on the receipt of a notification; but any one who has visited the homes of some of the poorer consumptives will allow that there is room for some supervision and advice as to preventive management. Notification would also allow of some supervision of lodging-houses and hotels where consumptives are, or have recently been, staying, with distinct advantage to the community at large.

One of the practical difficulties is that many of the cases which should be notified are not under medical treatment,

and it would be difficult to get members of the family to notify. Those who are under medical advice will be told what is necessary without notification; those who are not may avoid notification and may not know what is advisable. In this way the notification of Consumption—like notification in scarlet fever or small-pox—may fail to directly accomplish all that is desired. Indirectly, however, it will effect much; for no more convincing method could be found of bringing home to the consumptive and his family the dangers of the disease, and the necessity for care to prevent it spreading.

The Prevention of Tuberculosis through Food.—Although Tuberculosis transmitted from animals to man by means of flesh, and more particularly of milk used as food, does not, as a rule, primarily affect the lungs, the prevention of Consumption cannot be dissociated from that of Tuberculosis in general.

The danger of drinking the milk of tubercular cows is fully proved by experimental investigations: the risk of using the flesh of tubercular animals as food is comparatively small, but by no means to be neglected. These dangers may be minimised by attention to hygienic requirements in the sheds and quarters where the cattle are kept; by removing sources of infection — tubercular cattle—from the herd; and by preventing the milk, and in some cases the flesh, of tubercular animals from being sold for food. To ensure these points, inspection is necessary. Compensation should be given to those who willingly submit to inspection and its consequences, and penalties must be enforced on those who seek to evade

their responsibilities. This entails much expenditure of public money, but it would not be difficult to prove that the present loss to the country by the high death-rate from Tuberculosis amongst the younger members of the community costs the country more. Denmark, under the advice of Bang, took the lead in preventive measures directed against the danger from tubercular milk and food, and the communication of Bang to the French Congress of Tuberculosis (*Brit. Med. Jour.*, Epitome, p. 26, August 13, 1898) gives many interesting and important facts. He sums up the "ideal prophylactic measures" as follows: (1) The recognition of all animals affected with Tuberculosis, and their compulsory notification, followed by the examination of all beasts which have been exposed to contagion; (2) Destruction of all animals sufficiently diseased to be able to transmit the affection; (3) Complete isolation of animals slightly affected, which must be slaughtered as soon as the disease tends to develop itself in them.

It is in the details for carrying out these principles that difficulties present themselves.

CHAPTER X.

THE PRACTITIONER'S PART IN THE PREVENTION OF TUBERCULOSIS.

The requirements of a general scheme for effecting the limitation or possible eradication of Tuberculosis in this country include much that can be carried out only by combined efforts under central direction.

A disease so widespread, which affects animals as well as human beings, cannot be influenced to any large extent by individual effort. To produce any appreciable result public sanatory bodies—even the central government itself—must put their machinery of organisation in action. Regulations for the inspection of cattle, meat and milk, for the destruction of infective sputa of consumptive individuals in public institutions, and for the disinfection of infected houses or rooms, whether promulgated in a district or operative throughout the kingdom, make an impression on the public mind and may produce the idea that the prevention of Tuberculosis is a matter which concerns the Medical Officer of Health and other public officials only. The general public—perhaps even some medical men also—may consider that the matter can be left to these officials, however interesting it may be to watch the effects of the means which are advocated. But

there is work for all. An undertaking of such magnitude as the prevention of Tuberculosis requires the co-operation of all branches of the medical profession, and the assistance of the public as well.

Although the organised attack of the Medical Officers of Health may for the present overshadow and obscure individual efforts, yet there is much which comes within the province of the practitioner which is important and useful. To the medical man in private practice belongs the duty of instructing those with whom he is brought in contact as to the ways in which Tuberculosis is spread, and of explaining the utility of such prophylactic measures as may be recommended. In this way he assists largely in the education of the public, without which preventive regulations meet with obstruction rather than with willing compliance. It is the practitioner who must explain to the consumptive patient how his disease may be imparted to others if certain precautions are neglected, and it is by him that these precautions must be detailed. Even the most careless will take some heed when these precautions are recommended by his own doctor for the safety of the patient's family and household. The bringing up of delicate children, especially if some member of the family is already consumptive, the counteracting of predisposition when possible, the avoidance of predisposing causes, and the selection of suitable occupation for the susceptible—on all these points it is the advice of the family doctor which is eagerly sought, or willingly accepted when proffered.

It may be useful to summarise some of the more important ways in which the practitioner, as distinct from

the medical official, may assist in the campaign against the most fatal of all diseases in this country :—

1. By educating the public, on the subject of the prevention of Tuberculosis, through the individual.
2. By instructing the consumptive patient as to the necessary precautions for him to observe for the safety of others; and by exercising the supervision which will tend to ensure the efficient execution of these precautions.
3. By the care of those who are specially susceptible, to prevent them becoming consumptive. It is in the families of tubercular patients that this care will be most required; the suckling of the infant by a tubercular mother must be forbidden; the boiling or sterilisation of the milk for the children must be recommended, and the general hygiene of the nursery and school will need attention. Careful instructions may have to be given to those recovering from such illnesses as Measles, Influenza, Pneumonia, for such persons are particularly vulnerable to tubercular infection during convalescence.
4. By the careful examination of doubtful or suspicious cases, to ensure the early recognition of Tuberculosis, especially in the lungs, and immediate treatment when the disease has commenced. We all recognise the greater probability of cure in the early stage of the disease, and we must also bear in mind that the longer a case goes on the greater danger does it become to others. The early cure of cases of Tuberculosis must be looked upon as one of the

important desiderata even from the point of view of prevention. I recommend a periodical and systematic examination of the chest in members of a tubercular family, especially during early adult life.

5. By advising employers of labour as to the danger from tubercular (consumptive) persons working with others, more especially if the office or work-room is ill-ventilated or over-crowded; and by instructing the consumptive workers as to the precautions they should take and the danger to others of neglecting these.

6. By using their influence to bring about the removal to hospital or elsewhere of advanced cases of Consumption living under conditions of danger to other members of the family—as amongst the very poor.

7. By co-operating with the Medical Officer of Health, informing him where houses or rooms might require disinfecting, where disinfectants for spittoons, &c. might be provided free, &c., &c.

The Medical Officer of Health might with advantage draw up short and clear rules to guide consumptive patients, such as are in use at most consumption hospitals; these might be printed and given to medical practitioners for distribution to their patients.

The following will serve as an example of such rules. They are interesting from the date at which they were first issued rather than for their completeness.

NORTH LONDON CONSUMPTION HOSPITAL,

MOUNT VERNON, HAMPSTEAD, N.W., AND
41, FITZROY SQUARE, LONDON, W.

DECEMBER, 1892.

Directions to Out-Patients.

As it is now known that the phlegm coughed up in Consumption contains the seeds of disease—

Do not swallow your expectorations. This habit may lead to consumption of the bowels.

Do not spit about the floor, nor into any utensil unless it contains a disinfectant.

Do not spit about the streets.

Indoors, use a special spitting cup or other vessel in which is some disinfectant.

Outdoors, use a pocket-handkerchief, or else use a piece of rag or paper which must be burned as soon as you get home.

You can get some disinfectant from the Dispenser for use in the spittoon or spitting cup.

The contents of this spittoon should be mixed with some more disinfectant before being emptied.

The spittoon should be emptied into the pan of the w.c. or into a bright fire, but *never* anywhere else, not even into the dust-heap.

If you spit into a pocket-handkerchief, it must be boiled for five minutes as soon as it is done with.

Keep your room well aired, throw the window wide open when you leave the room, and always keep it open a little at the top all night.

If there is a fireplace in the room, do not stop up the chimney but always keep it free for the passage of air.

Keep your room clean, do not allow dust to remain on the floor.

Consumptive patients should sleep alone.

Mothers who are consumptive should not suckle their children.

In addition to the part which falls to the duty of the civil practitioner, there is, I believe, a great work to be done by the medical officers of the Navy in the prevention of Consumption. Years ago the prevalence of Consumption in the Army was far in excess of that amongst the civil population, but the altered conditions of service and improvement in barrack accommodation have had a most satisfactory result. In the Navy, the changes in the types of our war vessels have not tended towards more hygienic accommodation for our sailors. This is, of course, to be deplored, but the exigencies of the service sometimes entail conditions which are unhygienic. Theoretical hygiene might suggest separate cubicles with through ventilation for our sailors on board ship, but patriotism comes before hygiene, and we must rule the sea even at the expense of creating invalids. But one consumptive becomes a great danger in a vessel crowded with her full complement of men, and the early detection and removal of such an one is of the utmost importance. My hospital experience has convinced me that this danger is real, and I would refer naval medical officers to a paper by Fleet-Surgeon Bryson, in the Transactions of the Epidemiological Society of London (Vol. ii. 1863-64, p. 142), entitled Epidemic Pleuro-pneumonia in the Mediterranean Fleet, and suggest that it should be read in the light of our present knowledge of the spread of Tuberculosis.

Part III.

THE TREATMENT OF CONSUMPTION.

CHAPTER XI.

THE INFLUENCE OF THE BACILLARY THEORY OF TUBERCULOSIS ON THE TREATMENT OF PHTHISIS.

That tubercular disease of the lung may be cured is proved by the records of post-mortem examinations, as well as by clinical experience. But all observations and experience tend to show that the chances of cure depend upon the stage of the disease, and emphasize the importance of early recognition of the tubercular mischief. It can never be too much insisted upon that the one essential requirement for success in the treatment of Consumption is such care and skill in examination as will make early detection of the disease possible. Failure of early diagnosis can never be made up for by the most complete knowledge of the various drugs and modes of treatment which have been recommended for Consumption. As in all diseases which often baffle the skill of the therapeutist, the number of remedies which have from time to time been brought forward as specially valuable in the treatment of Consumption is enormous. All have their little day and are forgotten, or at best are retained as means to alleviate special symptoms. The bacteriologist theorises on the possibilities of cure by antitoxins, his mind filled rather with the behaviour of the bacilli in a culture tube, than with the histological environment of the bacilli in the

body. The clinician, anxious to miss no chance of finding a satisfactory remedy, carries the experience of the laboratory to the bedside, and realises in his disappointment that the bacilli in the lung are not so easily reached by antitoxins or germicides as those in the culture medium.

Pathological considerations must be taken into account in forecasting the probable success of some of the newer remedies, such as tuberculin, antitubercular serum, or nuclein; but it is by the result of clinical experience that their ultimate place in the treatment of Tuberculosis will be determined. Drugs of all kinds still come and go, but the one point in treatment which is steadily gaining ground is that the destruction of the bacilli and the neutralisation of their products, which we have so far failed to accomplish from the outside, *i.e.* by germicides and antitoxins, may be accomplished from within by the living tissues. Hygienic treatment consists in strengthening the organism to effect its own cure—aiding the *vis medicatrix naturæ*.

But whilst we recognise that hygienic treatment offers the best chances of success in treating Consumption, we must beware of forgetting the important assistance which we may obtain from drugs. At present by far the greater number of consumptive patients in this country have to remain under adverse hygienic conditions, and have to rely on such relief as medicines can give them. The Consumption hospitals can only receive a comparatively small proportion of the poorer patients, and that only for a short period. There is little provision for middle-class patients, who might have a better chance of recovery away from their homes. We want some large institutions in this

country for the hygienic treatment of consumptives, where patients might remain as long as may be desirable; not merely private homes for well-to-do persons, but public institutions, with fees within the reach of the middle-class patients, and free beds for the poor. We have the experience of the large Sanatoria in Germany and America to guide us in construction, arrangement and management of such institutions, and private enterprise has demonstrated, although on a small scale, that the open-air treatment of Consumption can be successfully carried out in this country.

The Influence of the Bacillary Theory of Tuberculosis on the Treatment of Phthisis.—Within the last few years all sorts of antiseptics have been recommended in Phthisis; some as medicines, others for subcutaneous injection; others, again, for use as inhalations or even to be injected *per rectum*. Injections direct into the lung tissue were also practised. Some of the drugs employed had long been used in the treatment of Phthisis, and had already gained a certain repute. Of these creasote and iodine may be taken as examples.

Creasote and its derivative, guaiacol—both used in the form of the carbonate—are now, perhaps, the most fashionable (*a*). Theoretically, the attempt to kill or to prevent

(*a*) The following antiseptics have been used:—(1) By the mouth: Phenol compounds—creasote, guaiacol, salol, benzozol, and their compounds; iodine compounds and iodoform; corrosive sublimate. (2) Inhalations: Carbolic acid, creasote, eucalyptus, bromine, iodine, iodoform, hydrofluoric acid, sulphurous acid, sulphuretted hydrogen. (3) Injections: (a) Hypodermic—creasote, guaiacol, eucalyptol,

the growth of the *Bacillus tuberculosis* within the body, by means of antiseptic or germicidal substances introduced direct to the affected part or carried there by the blood stream, is admissible. But the practical carrying out of this object is attended with great, and apparently insuperable, difficulty. No doubt if creasote, guaiacol, or corrosive sublimate (and many other substances) could be made to reach the bacilli in sufficient quantity the result would be the destruction of the micro-organisms and the arrest of the disease. This, however, would be at the expense of lowered vitality of all the tissues and a consequent predisposition of the individual—a matter of some importance to those undergoing treatment in a special hospital for consumptive patients.

The quantity of any germicide required to make the fluids of the body antiseptic is a bar to complete success. Estimating the amount of the blood at one-thirteenth of the body weight, this would give for a man weighing nine stone nearly ten pounds of blood. To make a 5 per cent. solution, about half an ounce of the antiseptic must be dissolved in the blood, and this quantity must be maintained in spite of rapid excretion. Hölscher showed that we cannot effect this, although both he and Cornet succeeded in rendering guinea-pigs partially immune against tubercle infection by injections of creasote.

But supposing we can get sufficient antiseptic dissolved in the blood, will it reach the bacilli? The pathological changes which occur in the immediate vicinity of these

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iodine; (b) into the lung—bichloride of mercury, iodine, iodoform, carbolic acid, creasote, turpentine; (c) *per rectum*—sulphuretted hydrogen.

organisms result in early obliteration of the blood vessels of the part, and the consequent cutting off of the blood stream from the disease focus. This necessarily prevents any blood-borne antiseptic reaching the bacilli in the tubercular centre. True, the margin of the tubercular area still receives its blood supply, and extension of the mischief might presumably be checked. But the bacilli in the centre of this area, secure from flushing by an antiseptic blood stream, thrive and multiply; and when, in the natural course, the bloodless mass softens and breaks down, myriads of bacilli are suddenly let loose to be carried to other parts. Were it possible to have the whole body so impregnated with antiseptics as to ensure the immediate destruction of these micro-organisms, wherever they may be carried, we should soon clear the body of the bacilli. But how would the living cells of the body thrive on such an antiseptic liquid? These living cells are our natural protectors against the attacks of pathogenic organisms, and we want to promote their vigour and not to poison them with antiseptics.

We must be content with a less strongly antiseptic fluid in the blood vessels, and at the best the bacilli will be "scotched, not killed." Antiseptic remedies cannot be relied upon as curative agents in pulmonary Tuberculosis. Nevertheless, these drugs have a certain value in the treatment of Consumption; and one object of these remarks is to assign to such remedies their proper position, so that they may not be entirely discarded when the inevitable reaction against the antiseptic treatment of Phthisis sets in.

Several observers have recorded beneficial results from

certain of these antiseptic remedies, and this may in part be due to their possessing some action in destroying and nullifying the effects of secondary products of the growth of the bacilli, thus restraining the noxious agents which produce septicæmia. In this way they may prevent hectic, and the distressing symptoms which accompany high temperature. But perhaps the two great uses of antiseptics in Phthisis are to prevent secondary infection by the air passages, and to destroy bacilli which have been expelled from the body. The latter object is attained by disinfecting the sputa; the former is, to a certain extent, possible by using antiseptic inhalations. In my opinion, inhalations are better for this purpose than antiseptic medicines which are excreted by the lungs; for, with the latter, in order to affect one organ we flood the whole body with antiseptics.

The best means we have for destroying the tubercle bacillus within the body exist in the action of the living cells and fluids of the body—the natural safeguards against deleterious germs. If we drench the tissues with antiseptics, we may diminish the vitality of the bacilli, but at the same time we impair the vitality of the "phagocytic" cells, and this not only at the disease centres, but throughout the body. We are then interfering with the natural means of cure, not assisting Nature; and whenever medicine is opposed to the natural process of cure, it is likely to do more harm than good. I have at different times given a fair trial to most of the special drugs which have been found successful by others; but in looking through my hospital records, I have not found one that gave results which justified expectations held out by their advocates.

The hospital to which I am attached is surrounded with pure air, of which we encourage the patients to take full advantage; a dietary is provided which is so generous that it is the despair of the Finance Committee; and as much comfort and cleanliness are maintained as is possible with the class of patients admitted. With such hygienic advantages medicines are chiefly required to help the patients to get the full benefit of these, to stimulate appetite, to aid digestion, and to ease cough—especially so as to give a fair chance of a night's rest. In the ordinary case, a simple tonic (acid and bitter), an expectorant when needed, perhaps cod-liver oil, and an antiseptic inhalation from an oro-nasal respirator worn for an hour twice or three times a day, give better results than any of the antiseptic drugs which we have tried.

The good effects recorded by so many observers from the use of creasote, guaiacol, and other antiseptics prove that these remedies are beneficial in some cases. But there is hardly a drug which has not been used in Phthisis with more or less success. There is much significance in the following remark of one who records his success with creasote carbonate. Dr. Chaumier writes (*b*): "Those of my patients who obtained fresh air to the largest extent, and who tired themselves least, derived the greatest benefit from my treatment with creasote carbonate. With those, however, who lived in bad hygienic surroundings, and, in spite of the disease, were obliged to work the whole day, the condition remained the same, or became worse."

(*b*) Reported in *Deut. Med. Woch.*, No. 25, 1893, p. 611.

These lines would, I believe, apply equally well to any drug treatment of Phthisis.

Hygienic means furnish the most reliable treatment for Phthisis, and the medicines employed should help the patient to derive the fullest benefit from these. The essential importance of strengthening the resisting power of the tissues may be overlooked in attempts to kill the tubercle bacillus by antiseptics.

It is impossible here to quote cases in support of the views I have expressed. These views are, however, the outcome of careful observation of several hundreds of cases, and of a dozen years of special hospital experience.

CHAPTER XII.

SOME CONSIDERATIONS WITH REGARD TO THE TREATMENT OF PULMONARY TUBERCULOSIS, AND THE LIMITATIONS TO THE USEFULNESS OF ANTITOXINS AND ANTISEPTICS IN THE TREATMENT OF THIS DISEASE.

Though clinical observation may lack the precision of experimental research, it has a value to the student of disease which cannot be surpassed by any other means of investigation. Experimental research has cleared up much that might otherwise have remained long obscure, and has given wider meaning to much that is observed at the bedside; indeed, the chief end and use of experiment is to elucidate the clinical signs which it is the province of the physician to appreciate correctly, with a view to effectual treatment. The sum of what may be gained from noting the allied processes occurring in animals only suggests possible explanations of what occurs in the human subject, and these explanations can only be adopted when verified by clinical observation. That this must of necessity be the case is obvious when we consider how differently the same disease may affect different persons; and since the same disease may be modified in different individuals of the same species, it is possible that in animals of another species the same morbific cause may produce quite a

different chain of events. It is the recognition of this fact which has led to more careful consideration of the "personal equation" in our observation of disease. We aim at treating the individual who is ill, instead of applying the same remedies to all cases of a given disease. This is the rational outcome of the closer study of pathology and morbid anatomy. From such study it becomes evident that the conditions of organs and tissues are constantly undergoing changes—apart from any definite diseases—as the mere result of life; but these changes differ somewhat in different individuals according to the mode of life, &c.

Even at birth there are certain subtle differences between individuals—the impress of heredity—which, though not immediately obvious, exert a manifest influence as development proceeds, and stamp their mark on all the subsequent life-changes. Pathologically, persons do not all start in life from the same point; the race of life is a handicap. Even those who are closest together at the start soon diverge, and in a few years their individuality is as certainly stamped on the organs and tissues as it is in face, form, and manner.

Thus a morbid condition attacking the same organ in two persons of the same age would not produce precisely the same pathological picture in that organ in the two cases; the outline might be the same, but there would be differences in the details. Still more would the same disease show modifications in persons of different ages and of different classes or races. But it is not only the "soil" which may cause modification in the manifestations of a morbific agent. The dose and continuity of action of the poison which starts the illness will vary in different cases,

and the virulence of the infective material may be subject to variation.

Thus the same disease is sometimes widely different in its course and effects, not only in different animals, but in different individuals. Recognising this fact, treatment ceases to be entirely a question of "specifics."

One of the most noteworthy characteristics of medical progress in recent years has been the recognition of the modifying influence of the individual on the manifestation and course of the disease—what has been termed the personal equation in disease. Almost before this principle has been thoroughly appreciated we are being drawn again towards the search for specifics. Our *fin-de-siècle* therapeutical armamentarium would almost have satisfied the savage medicine-man or the astrologer of the Middle Ages. Instead of serpent's blood we have Residuum rubrum (arterial and venous), and the extracts or powders formerly derived from rats and toads, or from parts and excretions of animals, are now carefully prepared and elegantly dispensed. Thus we now have—

Ox bile.	Pancreatic substance.		Lymphatic gland substance.		
Pig bile.	Ovarian	,,	Mammary	,,	,,
Bone marrow.	Spleen	,,	Pineal	,,	,,
Cerebrin.	Spinal cord	,,	Pituitary	,,	,,
Didymin.	Uterine wall	,,	Prostate	,,	,,

The search for specifics has always had a fascination, and the more intractable to treatment a disease seems to be, the greater the anxiety to obtain a remedy which shall be universally applicable. Such specifics are continually being advertised for Consumption, but they rarely produce in the hands of others the success experienced by their

discoverers. Perhaps the difference is to be sought in the general management or hygienic *régime* which accompanies the use of the remedy,—showing that the so-called "specific" is at the best only partially responsible for the success. We are all cognisant of the different effects produced by "taking the waters" at a well-organised "Kurort," and that which can be attained by taking the same waters at home.

In the last year or two the search for specifics has received a fresh impetus from our further knowledge of the action of micro-organisms. Antitoxins are being prepared for all diseases—Tuberculosis as well as others. In discussing the use of these antitoxins it is necessary to bear in mind that in the treatment of poisoning more may be required than the administration of an antidote; important as it is to neutralise the poison, shock and local irritation, as well as toxic symptoms, may require attention. It may be that the antidote is itself a powerful poison, and danger may result from an overdose of the remedy. There is in these considerations no argument against the administration of the antidote; they merely emphasize the necessity for care in its administration, and suggest that we must not rely upon it alone. And further, as acute and chronic poisoning require different treatment, so also the success of antitoxins in some acute diseases does not necessarily imply similar success in Tuberculosis.

The combined results of long-continued clinical observations in man, and of comparative observations in animals, show Tuberculosis to be an infective process of long and persistent constitutional effects, and definite

local lesions destructive of the tissues in which they occur. It is associated with and dependent upon a special micro-organism—the Bacillus tuberculosis. In dealing with the accumulated evidence from clinical observation and from experimental research the predominant interest must still attach itself to the disease as it shows itself among mankind. The relation between cause and environment is no simple question, and each element needs separate study. The different behaviour of the same germ in various culture media, and the various effects produced in animals, are not without importance, but we must above all consider the differences observed in different human subjects under the influence of the same disease. Let us trace briefly the pathological changes which take place as a result of the invasion of the Bacillus tuberculosis—giving our attention primarily to the lung.

We will suppose the bacillus to have reached the bronchial tubes. Here, probably through some damaged epithelium, the bacilli get into the peri-bronchial lymph space, and, multiplying, set up inflammation in their immediate neighbourhood.

Cell aggregation and multiplication go on fast, and soon the mass of closely-packed cells replaces all the original histological structure of the affected spot. Vessels are absent in the cell mass, and those in the immediate neighbourhood may become blocked and obliterated. The centre of the nodule is nothing but a mass of cells cut off from all blood supply, and forming a habitat for innumerable bacilli.

Caseation or destruction of the cells provides material on which the bacilli thrive, safe from the destructive action

of living blood or of phagocytic leucocytes. The disease centre, impervious to both blood and lymph streams, is thus cut off from the influence of any antitoxin distributed by these fluids. This fact does not nullify the good effects of such remedies, but it certainly limits their operation. At the margin of the mass the bacilli continue the inflammatory action, and some of them getting into the blood or lymph vessels are carried away to start fresh foci of disease elsewhere. If now it were possible to keep the blood and lymph impregnated with antitoxin or antiseptic, such bacilli as get into these fluids might be destroyed, and extension of the disease be, to this extent, prevented. But even then the bacilli in the centre of the nodule still flourish, and are a continual source of danger. We cannot keep the blood charged with bactericidal substances ready to destroy the bacilli as they from time to time escape, for we should have to keep this up for weeks or months, with probable detriment to the body at large.

Thus, on theoretical grounds, we are led to doubt the possibility of curing Tuberculosis with antiseptics and antitoxins alone.

Whether it be possible to make the blood antiseptic by drugs at all may be doubted.

Again, let us suppose that the infective particles, whether introduced by the food or by the air, are arrested by the viscid mucus on the fauces. With undamaged epithelium the bacilli will probably remain innocuous on the mucous surface, and will be expelled or swallowed with the mucus. If, however, the epithelium be damaged—as by chronic pharyngitis, tonsillitis, or "relaxed throat"—the bacilli may find their way into the lymph spaces below, and pass

into the lymph stream. Arrested at the first lymphatic gland they come into conflict with the lymph cells, and if not quickly destroyed by these cells may cause inflammation, perhaps leading to caseation of the gland (strumous gland). In some experimental work in which I was engaged a few years ago, I sprayed tubercular material into the throat of certain rabbits, in some of which a subacute inflammatory condition of the fauces had been produced by repeated application of a weak iodine solution, others being untouched. The superficial cervical glands of the prepared animals, already somewhat damaged from the throat mischief, were found when the animals were killed to be enlarged, and tubercle bacilli were found in them. In the untouched animals these glands were found healthy. This demonstrates at least the possibility of strumous (tubercular) glands being caused by infection through the mouth. The caseation of one gland, accompanied as this change is with the multiplication of the bacilli, leads to the setting free of bacilli which may be carried by the lymph stream to the next gland, and there produce a similar condition of inflammation. But let us note a significant fact. Out of the many cases of strumous glands which we all meet with, how few, comparatively speaking, develop tubercular disease in other organs! True, we often meet with cases of tubercular Phthisis in persons of adult age—often of middle age—who in childhood have had strumous glands in the neck, evidenced by the scars which they carry through life. Are we to imagine that the bacilli have remained dormant for ten, twenty, or thirty years, and have after all this time become active, or may we not suppose a new infection

from without? Seeing the ubiquity of sources of infection, the latter supposition imposes less severe strain on the imagination than the former. In a patient recently under my care there was an interval of some twenty-five years at least between the strumous glands and the pulmonary Tuberculosis.

The comparative rarity with which sufferers from tubercular disease of the cervical glands subsequently become phthisical has long forced itself on my notice. The significance of this has been explained by experimental research, by which the power of living cells to destroy micro-organisms has been demonstrated. Here, then, is a most important fact. The healthy and vigorous cells in the body may and do safeguard the body from the effects of bacteria. Should these cells, however, be wanting in vigour, they are unable to prevent the development of bacteria which enter the tissues, and so to prevent disease. This is an elementary principle, and one which cannot be neglected in deciding upon the treatment of Tuberculosis. If by antiseptics or antitoxins we could destroy *all* the bacilli in the body, we need take little notice of the vigour of the protective cells; for by the remedy we do the work which they should do, and it matters little then if they are unable to do their duty. But if the antitoxin can only reach a proportion of the bacilli in the body, leaving others out of reach which are actively engaged in their destructive work, we must beware that our remedy does not diminish the activity of the protective cells as well as of such bacteria as come under its influence. In an acute illness it may be possible to check the development and multiplication of the causative bacteria, and so cut short

or cure the disease. If the protective power of the tissues is temporarily lessened by the treatment, as well as by the disease, invasion from without can be guarded against during convalescence. We have seen, however, that the struggle against the invading tubercle bacillus is necessarily prolonged; the invaders have a *pied à terre* from whence they issue at intervals until all are destroyed or until their victory is complete.

We have clear clinical evidence that the tubercle bacillus becomes more potent for harm when the general health of the individual is lowered, or when a particular organ is temporarily or permanently damaged. We saw in the case of the rabbits in my experiments how the bacilli infected those with damaged mucous membrane in the fauces, but not the others. In another series of experimental inoculations with tubercle, of rabbits in whom the lungs had been damaged, the control (or undamaged) animals which were inoculated became affected before the others. The damaged animals were strong and robust before they were operated upon; the control animals were poor ill-conditioned animals, though free from Tuberculosis. This tends to show the greater susceptibility to infection of the weakly animals. Clinically we see the same thing in the cases of Phthisis following influenza, measles, &c., or attacking those weakened by hysteria and depressing or exhausting work (*e.g.*, long residence in hospital).

We might reasonably infer the protective power of healthy tissues from the effects of infection when resistance is diminished, even without the confirmatory evidence of Metschnikoff and others.

Reasoning from this it seems logical to endeavour to get

the patient into good condition, and so aid his tissues to destroy the bacilli, rather than to trust to antiseptics or antitoxins, which may impair the protective power. This is, of course, difficult to accomplish when active Tuberculosis keeps up a drain on the strength, but there is good hope of success in early stages. This line of treatment I have adopted with a satisfactory measure of success after some fifteen years' special experience, during which I have tried almost all the methods of treatment which have been advocated.

To lay down dogmatically the details of treatment is impossible, since, as we have seen, each case must be treated on its merits—the patient, not the disease. It is impossible to treat all patients alike. We cannot treat Consumption; we must treat the consumptive individual. There are fashions in health resorts and fashions in drugs, but blindly to follow fashion is a confession of mediocrity. There is too much generalising in treatment where it is essential to particularise, but the latter requires more complete knowledge. Not only the seat, extent, stage and activity of the tubercular process require to be taken into account, but the temperament and peculiarities of the individual must be considered.

The principles of treatment are to build up the patient's strength, to relieve distressing symptoms, and to avoid infection of himself and of others. To attain the first object a full supply of fresh air and good food, with good hygienic surroundings, are most important.

I would rather have to treat Tuberculosis without drugs of any kind than be restricted as to air, food, and other hygienic requirements, even with an unlimited command

of drugs. The consumptive turned adrift far from civilisation in fresh pure dry air, and obliged to live the life of the woodsman and hunter, is better off as regards his chance of recovery than the miserable dweller in the heart of the richest city of the world, within reach of the most experienced specialists and of every drug, appliance, or "cure" which medical science (or anxiety) can suggest, but hampered by his unhygienic surroundings, by overcrowding, and by the necessary evils of the struggle for existence in a crowd.

Of all the various drugs which have from time to time been in vogue for Consumption in the last fifteen years I have found little to choose between them in their effect on the disease. Some simple mixture, such as an acid with bitter, is often valuable to improve appetite and digestion, and is in my experience the most generally useful medicine in Consumption.

Cod-liver oil and maltine are foods which are especially useful as additions to the ordinary dietary. The symptoms which often require to be treated are ineffectual cough, pain, diarrhœa, fever, and night sweats; most of which, however, generally soon improve under simple tonics, without special medication. For diarrhœa dieting and rest are important; for high temperature rest is essential.

The phlegm is a source of danger to the patient if it be swallowed, or if it is not cleared out of the air tubes. When expectorated it is a danger to others. It is to counteract these dangers that antiseptics find their chief value. By antiseptic drugs, inhalations, or vapours we may possibly do something to prevent auto-infection by sputa of intestine, lung, or larynx. By disinfection of the

spittoon, the linen, and the room we may prevent infection of other persons.

We must, however, not deceive ourselves when speaking of the curative measures in Tuberculosis. When it affects internal organs, such as the lungs, we are obliged to recognise that there is a stage in the disease beyond which we must not expect a cure. Amelioration of symptoms, or quiescence of the disease process, may still be obtained for a longer or shorter period,—but with the ever-present risk of a recrudescence of the active mischief. For the successful treatment of pulmonary Tuberculosis—and probably of all tubercular disease—early recognition of the existence and nature of the complaint is essential. The consumptive in the hands of a man who, with little knowledge of the art of prescribing, and small command of drugs, is able to recognise the presence of the disease in an early stage, is better off than one under the most famous expert, with the complaint in an advanced stage from want of early recognition. To treat successfully, we must be able to recognise clearly. The stethoscope is so easy to use that no medical man is without one; the laryngoscope is less easy to manipulate, and is largely left to specialists.

Now I venture to say that, except for the initial practice in manipulation, it is far more difficult to judge of the evidence obtainable by the stethoscope than of that obtainable by the laryngoscope.

Still, where we cannot cure we may prevent harm to others, and after all the highest aim of medicine should be to prevent disease. True, the longer a person suffers from Tuberculosis the more risk there is of his infecting others,

and thus an early cure has an influence in preventing the spread of the disease. But it is not by improved methods of treatment that the mortality from the most destructive diseases is materially lessened. Is it by improved methods of treatment that typhus has become so much rarer, or that ague has ceased to be a scourge in London and in the Fens? Neither is it due to any improvement in our treatment of Tuberculosis that the death returns have shown a diminution of late years in the mortality from this disease. The greater attention to hygiene and sanitation is surely to be credited with much of the diminution of typhus, ague, and Tuberculosis. Much more may be done to diminish the latter when we direct special prophylactic measures against this disease as has been done in the case of the other two diseases mentioned.

What these measures should be can hardly be laid down in their entirety by one individual, for they must include the management of animals as well as of human beings in their scope, and many interests will be affected and must be safeguarded.

Here personal experience cannot be invoked to endorse all the suggested measures. The success of prophylactic regulations cannot be decided by the experience of any one individual, and hardly by the united experience of a single generation. In deciding upon the measures to be recommended we have to study well the ætiology of the disease, and having learned the causes and mode of spread of the disease we base our regulations and instructions on these. Koch's discovery of the Bacillus tuberculosis, which for the first time demonstrated the infectious nature of the malady, showed the possibility of successful pro-

phylaxis. Hitherto so little has been done in this country in the way of systematic prophylaxis for Tuberculosis that we cannot do much more than theorise, though we have some valuable results in other countries (*e.g.*, Germany) to guide us.

Experience will later on make it possible to improve—by elaboration or simplification—our original scheme. One thing of importance is to teach the public that Tuberculosis is catching, and to get rid of immoderate fear of heredity.

Every physician may, however, draw from his case-book some cases bearing on the ætiology of the disease, and from these we may deduce suggestions which may save others from contracting the disease.

It is in the prevention of the disease that antiseptics will eventually find their true value in our fight against Tuberculosis.

CHAPTER XIII.

REMARKS ON SOME CASES OF PHTHISIS TREATED BY DR. KOCH'S METHOD.

THE time has not yet come (*a*) when a definite opinion can be offered as to the efficacy of Dr. Koch's means of treating Tuberculosis, but the wide-spread interest which has been aroused by the discovery calls for the production of any experience in the employment of the method without waiting until we can record conclusive results.

The excitement manifested over this discovery is to be regretted in that it tends to increase the difficulty of arriving at a just estimate of its value as a means of cure. But though unfortunate it was unavoidable. The success of Dr. Koch's researches in the laboratory justified the use of the lymph on the human subject, and the discovery could no longer be kept secret. Thus, incomplete investigations were communicated to the medical profession, because further and more extended observation was required. No sooner had it leaked out that a remedy for Tuberculosis had been found, which might successfully attack tubercular deposits in the lung, than hope spread the report that a cure for Consumption had at last been

(*a*) A Paper read in 1891.

discovered. What wonder that enthusiasm was aroused throughout the whole civilised world at this announcement, seeing how wide-spread and fatal is Consumption? But to the masses the term Consumption is too often synonymous with *advanced* lung disease, and a cure for Consumption conjures up in their imagination visions of the hectic invalid, shaken by his cough and wasting into his grave, restored to robust health and useful employment. Such high hopes must necessarily lead to disappointment, and already we see signs of the natural reaction. Having selected cases for treatment from my patients in hospital, I was met by refusal to undergo the treatment from more than one, and this when those who could afford to do so were hurrying to Berlin to be treated at once. One patient having read in the papers a description of a Lupus case after injection, imagined that the whole of the signs described were produced by the injection, and feared the treatment would bring her "out all over in sores." Others read of deaths following the treatment, and feared the risk. The publication of unnecessary details in the public press is undoubtedly unwise, and so, too, is the selection of hopeless cases for the early trials of a new remedy.

We must all regret the premature publication of optimist opinions, based on incomplete knowledge and unwarranted by facts, if only for the disappointment which must follow. But we, as scientific men, must not be led away by enthusiasm nor checked by disappointment; our judgment must not be precipitated by popular anxiety nor warped by public excitement.

We must investigate impartially, and only after extended and careful trial form and disseminate a mature opinion.

But even now at an early stage we may bring forward our facts, and thus, by collective investigation, jointly arrive at conclusions more quickly than would be possible to each worker singly. This interchange of views is in fact a consultation in which medical men of all nations assist.

Months must elapse before an opinion as to the value of the remedy as a curative agent can be arrived at, but already no doubt can remain that a notable epoch has been reached in therapeutics, almost ranking in importance with the discovery of vaccination. We have here apparently a preparation which has a selective affinity for the pathological products of a specific inflammation; itself prepared (if surmises be correct) from the micro-organisms which, if not the cause, are at least the constant concomitants of the special inflammation against which the lymph is successful. If such a result has been attained in Tuberculosis, may we not look forward to similar preparations to combat other diseases which are accompanied by specific micro-organisms? No doubt the chances of success are much greater in diseases which have a prolonged course than in acute illnesses of short duration. But even a partial success against one "germ-disease" may well stimulate investigators to attack other diseases of the same class. We must not forget in our enthusiasm over the work of our German *confrère*, that one of our own countrymen seemed five or six years ago to be within measurable distance of forestalling Dr. Koch's success. We may, perhaps, be not very wide of the mark if we take the experiments of Dr. Woolridge to help us to explain the nature of the fluid which Dr. Koch has discovered.

Dr. Woolridge having prepared a pure culture of the Bacillus anthracis in liquid proteid material, separated the liquid medium from the bacilli by filtration through a porcelain plate, and found that this fluid if added even in minute quantities to cultivations of the anthrax bacillus, checked and prevented further growths. Injections of the fluid in animals rendered them non-susceptible to the anthrax virus, even in excessive amounts. His success with anthrax led him, I believe, to commence investigations on similar lines with the Bacillus tuberculosis, but his untimely death put an end to his labours.

It appears, then, that the bacillus of anthrax, and presumably other bacilli also, in the process of growth and reproduction (in other words, as a result of their vital activity) render certain media in which they are living poisonous to themselves and their kind—somewhat as an animal shut up in a closed box eventually renders the air in which it has been living unfit to support animal life.

But Dr. Koch does not claim for his lymph that it directly destroys the Bacillus tuberculosis or checks its development; its mode of action is said to consist in the destruction of the new or altered tissues in which the bacilli are living.

It is claimed, in fact, that this lymph when introduced into the circulation has a special affinity for the sites of tubercular deposit, and produces a necrosis (or sloughing) of active tubercular material. This material being destroyed may be discharged or removed from the body, and with it the bacilli which had found a habitat in it, or it may remain and be slowly absorbed. In the latter case

the bacilli may die for want of nourishment, or some may escape into other parts of the body, where they may eventually settle down and become the foci of further tubercular mischief. The changes which have been noted in the appearance of the bacilli in the sputa of patients after several injections of the lymph may be taken as indications that the bacilli are dying of inanition.

The bacilli may become much attenuated without alteration in length, or they may become smaller in every way, as though the thinned rod were broken into two or three pieces; others, though thin in the centre of their length, have broader extremities, and thus are dumb-bell shaped. Others have several constrictions which produce a nodulated or beaded appearance like a row of dots; this form has been previously described.

If these observations are confirmed (and I have seen all the varieties I have mentioned), we must allow that though the lymph may have no direct action on the bacilli, these do become affected by the injections and may be destroyed. Seeing, however, that the main effect of the lymph is in destroying the tubercular tissue, we naturally ask how is this brought about? Why is only tubercular tissue affected? If I may hazard a theory I would suggest the following: active tubercular tissue may be defined as tissue in a condition of inflammation produced by the specific irritation of the products of the vital activity of a colony of tubercle bacilli. Dr. Koch's lymph is believed to contain as its active principle these products of the bacilli; and though not sufficiently powerful to set up its specific inflammatory action in healthy tissues, it is enough when

added to the actively inflamed (tubercular) tissues, so to increase the morbid changes that death of the part results from the intensity of the inflammation (*i. e.*, by sloughing). Of the lymph itself I need say nothing as it has been frequently described. This liquid is said to contain 1 part of the active principle in 1,000, and for injection purposes the fluid is further diluted.

For first injections a 1 per cent. solution is best used, but for larger doses we may employ solutions of 2, 5, or 10 per cent.

[To illustrate this paper eight cases were described: one, a case of Lupus, which served to show the local changes which take place after the injection; the others were all cases of pulmonary Tuberculosis.]

The case of Lupus is a girl (Rose W.) of ten years old, who was in the Hospital for Sick Children, Great Ormond Street, under the care of Dr. Barlow, to whose kindness I was indebted for the opportunity of using the injections on this patient.

She belongs to an extremely tubercular family, almost every member of which has some tubercular manifestations.

She has a patch of lupus on the nose which commenced twelve months ago as a sore spot inside one nostril, and has gradually spread until it now involves the whole of the tip of the right side of the nose and extends into both nostrils.

There are also tubercular nodules in a scar on the right temple, and in another scar—the result of a burn—on the right wrist. The lymphatic gland above the inner condyle

of the right humerus has suppurated, and there is an adherent scar over the hard and thickened remains of the gland.

There are also nodules in the soft palate and on the right posterior pillar of the fauces.

The epiglottis is much thickened and the false vocal cords seem also swollen, but laryngoscopic examination is difficult.

There is a metallic-sounding cough, evidently laryngeal; the lungs are apparently free from disease.

The first injection on December 31 was two milligrammes.

The temperature began to rise from five to six hours after the injection, and the child felt chilly.

The lupus patch on the nose became red and discharged freely. The patch on the wrist also became swollen and surrounded with a red zone, and some doubtful spots on the left shin became redder and swollen.

There was headache, but no difficulty in breathing and no cough. The highest temperature was 103° F. After the reaction had subsided the patches on the nose and wrist were covered with a scaly scab. A red blush was noticed on the back and leg. With the subsequent injections, four, five, and six milligrammes respectively, similar local reactions occurred, and the gland at the elbow also reacted. With the larger injections, however, the general reaction was much more severe.

The highest temperature reached was 103·8° F., but the pulse was very rapid—I counted it once at 212 per min.—and very small and soft. The breathing became

hurried and difficult; cough was frequent, and distressing, and metallic in character.

The neck was painful from swelling of the glands all down both sides.

The chest was full of rhonchi and moist sounds, and the lower ribs were retracted during inspiration, probably from the larynx being swollen and interfering with the entrance of air.

During the height of the reaction the child looked as ill as it was possible to be.

As the effect of the injection passed off the breathing became quieter and the lungs free from râles, but there was now some diminution of resonance and fine crepitations to be detected at the base of the right lung. All the lupus patches were greatly improved.

Note.—An interesting circumstance in this case was that, though most marked improvement took place at first, there was subsequently an increase or re-crudescence of the disease, and this occurred even though the injections were continued for a short time after apparent cure had been brought about, and actually whilst the injections were still being given.

Having described the cases we may see what general deductions can be drawn, and what difficulties or warnings present themselves.

We note that in all the patients the injection has been followed by marked reaction, showing itself in general symptoms and in local signs.

The amount of the lymph required to produce the reaction is not always the same even at the first, and, in all, a kind of tolerance to the fluid is quickly established.

This is seen in the slighter reaction which results from a second injection of the same amount as the previous one, even to a dose of increased quantity, and finally no apparent effect is produced even with large doses.

This is explained, according to Dr. Koch's theory, by the supposition that the reaction depends upon the amount of active tubercular material in the body, upon which alone the fluid is said to produce any effect. Each injection is followed by destruction of some of this tubercular material, and less therefore remains to react to subsequent injections. When all is destroyed, no reaction follows even comparatively large doses.

In this theory of the action of the remedy we have an important point to guide us in the dosage, both in children and adults. The dose must be proportionate to the amount of active tubercle, rather than to the age of the patient, and our only reason for using smaller doses in children than we should employ in adults with corresponding tubercular mischief, is that children have less resisting power to febrile reaction than older persons. We should expect also that in children the temperature and pulse would be more easily disturbed by the changes set up by the injection than would be the case in adults; but with these allowances, we select our dose according to the amount of tubercle rather than in proportion to the age.

Here we have a marked difference from the ordinary chemical drugs, and some analogy to vaccine lymph, where we use the same amount to vaccinate an infant or an adult.

But the position of the tubercular deposit and the organ or tissue affected must necessarily largely affect the

dose. Acute inflammation and swelling of a tubercular patch on the nose or cheek, will have none of the dangers of a similar condition in the larynx, and we hesitate to set up excessive inflammation in the lung, in the eye, or in the brain, even if we can be sure of its being confined to a local deposit.

In connection with the dosage of the remedy there is an important question which I think requires consideration.

Is the best result to be expected by commencing the treatment with the largest dose that can safely be borne, or shall we obtain equally satisfactory results by injecting first a minimum dose, and gradually increasing the quantity? In the first case we produce a rapid effect in the tubercular material, with a corresponding severity of the general reaction. By commencing with small doses, we produce a certain toleration to the remedy before we have arrived at the injection of large doses. If this toleration is due to the gradual destruction of the tubercular material at the seat of disease, the ultimate effect is the same as that produced by a large dose, and we have the great advantage of effecting this without subjecting the patient to the misery (and perhaps the danger) of a severe general reaction. But if the tubercular tissue becomes gradually tolerant of the lymph without undergoing destruction, it is better to risk the severe reaction in order to ensure the destruction of the morbid deposit.

My observations on this point are not sufficiently complete to warrant any definite answer to the question. In the two lupus cases I have under treatment, and in whom I commenced with smaller doses than those used at first by some other observers, the injections have not yet been

used sufficiently long for a cure to be expected already, but in each case the progress has been satisfactory, and warrants a hopeful prognosis. In two of my lung cases I commenced the treatment with the large dose of 1 centigramme, whilst in the others I began with the minimum dose—1 milligramme. The severity of the reaction in the two former cases gave some anxiety, and so far I cannot think that there was any corresponding advantage in the effect on the lung, over that produced in the patients in whom the dose of 1 centigramme was gradually reached. If large doses are to be used in pulmonary tubercle, we must only select cases in which the amount of deposit is small, and in whom the general strength is good.

If small doses gradually increased are equally efficacious, it may ultimately become possible by carefully regulating the dose to treat patients without interfering with their employment. For the present, patients must be content to give up everything to the treatment for a few weeks.

The time at which the reaction produced by the injection commences, and its duration, seem also to vary with the dose, or rather let us say, with the proportion between the dose and the amount of active tubercular material. In a typical case, the reaction seems to show itself about four to six hours after the injection, the height is reached in another two hours and is over about twelve hours from the time of injection. The reaction lasts altogether about six hours. With relatively small doses (the quantity depending upon the amount of active tubercular tissue), the reaction may be delayed, as is seen in one of the Cases (Walter W.), where no reaction was noted until after the fourth injection (0·004 gr.), and then the reaction was

delayed until the evening of the day following the injection, the temperature then running up to 102·4° F. With relatively large doses the duration of the febrile reaction may be prolonged.

I have already mentioned the symptoms which accompany the reaction when describing the cases, but we may now note more particularly the most important of these.

Headache is a very frequent symptom. It comes on early, and is often the first indication of a reaction. It is usually frontal, but may be felt all over the top of the head.

Nausea and Vomiting are common, and diarrhœa occurred in one Case (Bellamy).

Shivering or Rigors accompany the rise of temperature; sometimes merely a feeling of chilliness, general or confined to one part of the body (*e.g.*, cold feet) at other times a severe rigor which may be repeated.

Pain is often complained of. Severe lumbar pain occurred in several of my patients. Others had general pain or soreness all over the body. Abdominal or epigastric pain has also occurred, and in two children it seemed particularly severe. May this be taken as an indication of tubercular mesenteric glands which have been affected by the remedy?

Local pain at the seat of the disease is almost always experienced, and in the cases of pulmonary tubercle the pain over the affected part of the lung was usually accompanied by tenderness.

Temperature.—One almost constant sign of reaction is the rise of temperature. In none of my cases has a temperature of 105° F. been reached, though a considerably higher temperature has been recorded in several cases here and abroad. When the temperature has reached its maximum, the subsequent fall is often succeeded by a secondary rise before the reaction passes off. Although this febrile condition is sometimes accompanied with a marked feeling of illness, there is sometimes no such sensation.

In the case of children, I have found that by timing the injection so that the reaction occurs during the night the child may sleep through the whole reaction.

In some few cases, instead of a rise of temperature, the injection has been followed by a fall to considerably below the normal, subsequently returning to the normal height.

Pulse.—In my experience the injections have usually a marked effect on the pulse. This I noted in most of the cases at Berlin, and I have confirmed it in my own cases. The pulse at first may be full and bounding, but very soft; later it becomes rapid and small, and very compressible. In children it may reach 190 or 200, and in one case it was counted at 212 in the minute.

The most noticeable peculiarity is the diminution of arterial tension; whatever the rapidity or the volume of the pulse it is usually markedly compressible. In some patients the low arterial tension seems to persist for some days.

It appears to me that we have in this effect on the circulation one possible danger from the injections. The

pulse should be watched during the reaction, and its condition noted as a guide to the amount of subsequent doses.

In two of my cases I gave ether during the reaction because of the condition of the pulse, with apparently good effect, though in this, as in other unfavourable signs which sometimes occur during a reaction, our great safety lies in the short duration of the acute stage produced by the remedy.

The Effect on the Lungs.—In all cases, even when the lungs are apparently sound, increased rapidity of breathing accompanies the febrile reaction, and cough is also produced. In Phthisis the breathing is quickened and dyspnœa frequently occurs. The cough is increased, not only during the acute reaction but generally for a day or more afterwards. It then subsides, and is often less frequent and less distressing than before the injection. The expectoration is increased in quantity at the same time, and may be altered in character. During the height of the reaction, I have seen tinted mucus like the rusty sputa of acute pneumonia. In other cases I have seen blackish or brownish lumps, though I have never seen pieces of lung tissue. Under the microscope, elastic fibres may be easily found, and the number of tubercle bacilli is often largely increased after the early injections, though they may diminish in number or disappear after more prolonged treatment. The increase of bacilli in the sputa is not taken to indicate an increased number in the lung, but is explained as resulting from the expectoration of broken-up tubercular material in which numbers of bacilli had found a habitat. If the bacilli are not killed by the

treatment, we have, in this wholesale removal of bacilli from the lung in the expectoration, an indication for increased care in disinfecting the sputa of patients undergoing this treatment.

As signs of the local effect of the remedy on the lung deposit, we have pain referred to the diseased part, and changes in the stethoscopic signs over the affected portion of the lung.

At first the breath sounds are extremely harsh, and large crepitant râles are heard, resembling the *crepitatio redux* of pneumonia.

Later the sounds become more liquid, and large bubbling râles are heard.

In several cases small deposits of tubercle previously undetected have given marked indications, by pain and stethoscopic signs, of their presence after the injections. The effect produced on the lungs appears, in fact, to be that a pneumonia is excited in patches, presumably around a deposit of tubercular material. In the phthisical cases we are prepared for this; in cases where the lungs were not known to be affected, are we, as in the case of Lupus (Case I.), to accept this pneumonia as an indication of tubercle in those organs?

Amongst the occasional symptoms which I have seen are the following:—

Rash.—Of this there are three forms which I have seen. The one consists in erythematous blotches or patches, which may occur on the back, limbs, or face.

The second form is a fine papular eruption, which may almost cover the body, or may be limited to the limbs.

The third variety consists of large raised red spots, dotted singly, or in groups, over the face, trunk, or limbs. They are much larger than those of the second form, and very much fewer in number. Some of the spots may have a yellow head, forming pustules.

The first form seems to be of short duration, and disappears after a few hours; the two papular varieties I have known to persist for two or three days.

Whatever its form, the rash may only be an accident of the fever.

If we consider it to be a specific inflammation of the skin produced by the lymph, we must allow that the effect of the remedy is not confined to tubercular tissue.

Delirium has occurred.

Collapse gave some anxiety in a patient in the Augusta Hospital at Berlin whilst I was there.

Jaundice to a slight extent has been observed in two of my cases, but passed off quickly.

Albuminuria has been reported, but has not occurred in my patients.

Double Vision was a distressing complication of the first reaction in one of my cases.

This I have not yet seen reported by other observers.

Weight.—In all cases in which febrile reaction follows the injections, I have noted some loss of weight during the first week or two of this treatment. In favourable cases an increase in weight then commences, although there may still be occasional pyrexia after the injections.

The initial loss of weight will surprise no one who has witnessed a severe reaction.

If Dr. Koch's contention, that the remedy affects tubercular matter and that alone, be correct, we must accept his opinion that its value as a diagnostic agent is only secondary to its importance as a means of treatment.

At present we must allow that his contention appears to be justified, but we should not, I think, accept it absolutely until more extended use of the remedy has enabled us to know more about its effects.

Are we, for example, to accept the reports that cases of leprosy react to the injections as a proof of the tubercular nature of leprosy? That such an explanation of the nature of leprosy should ultimately be found to be correct, would not perhaps come as a complete surprise, but we cannot accept the reaction of leprosy to Koch's lymph as a proof.

In two of my cases at the children's hospital, I carefully watched after the injection for any effect in the inflamed (congested) patch produced by a blister, but none could be detected, though in one case a lupus patch close to the blister reacted well.

Auxiliary Treatment.—Whatever reliance we may ultimately be able to place on this treatment for Tuberculosis, we shall probably get better results the more we can sustain the patient's strength whilst the remedy is being employed. We should therefore avail ourselves of such useful aids to cure as cod-liver oil and good food throughout the treatment, and not forget the importance to our patients of a plentiful supply of pure air.

In conclusion, we see that we are dealing with a most powerful agent, which demands all the more care in its use at present from the fact that its composition and its mode of action are as yet imperfectly known. It must be used with extreme caution, and its effects in each case must be carefully noted. Its proper dosage for each case requires anxious consideration, and there is still much to learn as to the rapidity with which the amount injected may be increased, the maximum dose required in any case, and the length of time for which treatment should be continued.

If improvement is brought about for a time, and a relapse occurs later, we may expect that the disease may be again checked by re-commencing the injections, and relapses will be regarded with less anxiety from the knowledge that the disease can be quickly checked.

At present we can only say that in Lupus we can obtain great improvement from the injection, and if later the disease reappears we have the means of again arresting it before much healthy tissue becomes involved.

Possibly also in early tubercular Phthisis we can expect similar satisfactory results. We cannot yet speak of permanent cures. In Lupus we can see the improvement. In Phthisis we have to rely somewhat on the patient's statements, and though we can measure the improvement in the temperature, the increase in weight, or the disappearance of dulness or signs of softening, and can check to some extent the patient's reports on his cough, appetite, or night sweats, we must take into consideration when the patient tells us he feels better, that he is testing a treatment which he hopes may cure him, and is anxiously

looking out for signs of improvement. His expectations may unconsciously colour his report of his condition, and improvement may be more apparent than real.

If the best results are to be obtained from the remedy, we must carefully select our cases, and though I have seen advanced cases of Phthisis injected without harm, it is only early cases that give a reasonable expectation of cure.

These cases, we must recollect, are just those in which we had reasonable hopes of permanent arrest of the disease under other modes of treatment.

The points to guide us in the selection of cases for treatment are as follows:—

Localised deposit with little or no softening in young patients offers the best chance of success.

The nature of the deposit should be determined by examining the sputa for bacilli.

The previous occurrence of Hæmoptysis is not necessarily a contra-indication.

I should hesitate to use the remedy in—

Cases of extensive deposit in one or both lungs.

Cases of extensive softening.

Cases with high evening temperatures (over 102° F.).

Patients who are much debilitated.

Patients with persistent diarrhœa, presumed to depend on tubercular ulceration of the intestine. Here destruction of tubercular deposit in the intestine might risk perforation.

Cases with marked congestion or œdema of the bases of the lungs.

Patients with fatty degeneration of the heart, or marked mitral stenosis or dilated ventricles.

Cases of pneumothorax.

In empyæma and pleuritic effusion, I should think the fluid should be withdrawn before the injections were commenced.

In Berlin I examined cases in all stages of Phthisis who were undergoing treatment, even some with extensive tubercular infiltration of both lungs, and although I saw no ill effects follow the injections in these advanced cases I could not note any improvement, and certainly little permanent good could be expected where much of the lung was already destroyed.

Tubercle in the larynx does not seem a sufficient reason to withhold the trial of this remedy, but it is well to be prepared for tracheotomy or intubation if occasion should suddenly arise.

With extensive laryngeal deposit and swelling, it might be advisable to practise tracheotomy or intubation as a precautionary measure before commencing the injections.

The importance of exercising care in the selection of cases, if we wish to avoid errors in investigating the effect of the remedy, is seen in the following instances. A child at the Hospital for Sick Children who was examined with a view to undergoing Koch's treatment, was rejected on account of its poor condition, and no injection was given; the same evening the child's temperature rose to 104° F., which would probably have been ascribed to the action of the lymph if it had been used. A woman also in my wards at the Consumption Hospital who had been selected for this treatment, but whom it was afterwards decided not to inject, had the same afternoon a rise of temperature to 103° F., though it had previously been normal.

In other cases, during the treatment, a break was made in the injections when the patients did not seem so well as usual, and in two of these an attack of acute tonsillitis produced as great pyrexia as had followed the injections. In the children's hospital an outbreak of measles in one ward, which attacked some of the patients under Koch's treatment, put a stop to further injections for a time.

Professor Ewald—whose courtesy to the foreign medical men who visited his clinique at the Augusta Hospital in Berlin has deservedly called forth grateful acknowledgment from all sides—stated that he had sought for contra-indications to the remedy and could find none. For the present I certainly think we should do well rather to err on the side of over-caution until we can determine the value of the treatment in selected cases.

Note.—The cases, interesting as they undoubtedly were when Koch's tuberculin was only just received in England, are omitted, as the record would have no special interest now. I kept some of my patients under observation for several years, and though some kept well, others came into hospital again after various intervals with a relapse or advance of the disease. I had the satisfaction of using the tuberculin for six months—the time I had fixed as a minimum period for a satisfactory trial—without having to record a death during its employment. At the end of six months, however, I gave up using tuberculin, as it appeared to me to give no better results than I had obtained by other methods of treatment which were not attended with the discomfort to the patient and the anxiety to the physician which the "reactions" of tuberculin entailed.

CHAPTER XIV.

THE ADMINISTRATION OF LARGE DOSES OF GUAIACOL IN PHTHISIS.

The accompanying report, which was drawn up for me by Mr. C. Stanford Read, the resident medical officer at the North London Hospital for Consumption, is, I think, sufficiently interesting to publish. It is, of course, incomplete, as the observations were still in progress when it was written; but it establishes the fact that patients can take pure liquid guaiacol in doses of one drachm three times daily (180 minims in the day), not only without toxic effects, but apparently with decided benefit. Creasote has been given in doses of from 160 to 180 minims a day and carbonate of guaiacol in doses of from 45 to 60 grains three times a day, but I know of no record of guaiacol itself approaching these quantities. The amount of guaiacol in creasote is somewhat indefinite; the carbonate is said to contain about 91·5 per cent. of pure guaiacol. A drachm of carbonate would thus be equal to about 55 grains of pure guaiacol. The liquid guaiacol is not quite pure, so that the amount of guaiacol taken will be much the same with a similar dosage of the carbonate or of the liquid guaiacol. The latter has the advantage of being less expensive. The caustic action of uncombined guaiacol on the mucous

membrane and some fear of toxic effects have prevented the prescribing of full doses of this drug, but it will be seen from the following report that with due care in administration most patients can safely take half a drachm, or even one drachm, three times a day. The drug was given in capsules containing 5 minims each, or in emulsion with glycerine and tincture of orange peel, and was always followed by a drink of milk. The doses were taken after meals. I have not attempted to exceed the dose of 60 minims, or to give more than 180 minims in the twenty-four hours, but some of the patients have continued taking this amount for several weeks with apparent benefit. One patient who has recently been discharged after taking this quantity for three weeks gained about 1½ st. during the three and a half months that he was in hospital, and left in a condition which might well be described as "cured." Short notes of this case will be found at the end of this report. It has been stated (*a*) that after large doses of guaiacol the urine gives a precipitate with hydrochloric acid. I am unable to endorse this statement, for the urine of patients taking large doses of the drug has not given any precipitate with hydrochloric acid in those cases which I have tested.

REPORT on the Treatment of Phthisis by Guaiacol.

By Mr. C. Stanford Read.

Guaiacol has often been administered in the North London Hospital for Consumption in certain cases in the past, usually in capsules of 5 minims each, after the three

(*a*) Eschle. Quoted by Dr. R. Seifert, *The Lancet*, Nov. 14th, 1896.

principal meals of the day. This has hitherto been done in cavitation cases accompanied by profuse or fœtid expectoration with very good results as far as this one particular symptom went. Of late, at the suggestion of Dr. Squire, we have pushed the drug in all stages of the disease to see how much patients could take without ill effect and what good results, if any, would follow. In many cases after starting the treatment one has been obliged to cease the administration of the drug, as other symptoms or complications (not connected with the guaiacol) have arisen necessitating its withdrawal. We have nevertheless forty patients who have taken the drug: six cases took 60 minims, two cases took 50 minims, four cases took 40 minims, six cases took 30 minims, ten cases took 20 minims, six cases took 15 minims, and six cases took 10 minims three times a day after food. Some of these are still in hospital and are gradually increasing the dose. The drug was first administered in capsules, each containing 5 minims, but later was given in an emulsion with glycerine and tincture of orange peel, many patients taking it partly in one form and partly the other. A good few found difficulty in swallowing many capsules; others, again, preferred them much to the acrid liquid; in either case they would drink about half a cupful of milk with the guaiacol, which seemed to greatly add to its being borne well. The dose of 5 minims was at first usually increased by 5 minims every third day, and later more rapidly, till 60 minims were reached. Only one patient felt any ill effects; he, when taking 20 minims three times a day, complained of much stomachic and abdominal pain with a sinking sensation in the epigastrium, passing a small quantity of blood by the bowel. The guaiacol was stopped, but resumed later with 10-minim doses without ill effect. Twenty-six were cavitation cases, and in these the diminution in the amount of expectoration was very marked, beginning to diminish early in the administration of the drug. Some patients whose expectoration was very profuse on admission would later only cough up little "pellets" of sputum first thing in the morning and none after. There has been no marked effect

on the temperature observed when guaiacol has superseded other drugs, but in those taking it continually a steady lowering of the evening temperature has been commonly noticed. Thirty-five of the cases put on weight, most of them well, one putting on 19 lb. in twelve weeks. There was not, as far as I could make out, any definite relation between the increase in weight and the proportional increase of the doses. The cough was in no special way affected that I could see, but night sweats in nearly all cases diminished and then disappeared in a very short space of time. Twelve cases had laryngeal Phthisis as well, but as these cases all had local treatment also one cannot say whether the guaiacol had any special effect or no. The patients complained a good deal either of the emulsion burning their throat or of their difficulty in swallowing the capsules, but by dint of perseverance combined with faith in the drug they managed very well after a time. The effect of the drug, if any, on the number, &c. of the bacilli in the sputum is now being observed.

The following is an abstract of the case referred to in the former part of this paper. The patient was a man aged twenty-four years. He was admitted to hospital on Nov. 18th, 1897, complaining of much weakness, sickness, and cough. The patient had been working harder than usual of late and had got run down. The first symptom was hæmoptysis (one pint) six weeks before admission. Cough with weakness and wasting gradually supervened. On admission he was found to be a cachectic pale man. He was 5 ft. $4\frac{1}{4}$ in. in height and weighed 8 st. 13 lb. 6 oz. He had very profuse night sweats and much sickness; the cough was not very troublesome; he had muco-purulent expectoration. With regard to physical signs, there was very slight impairment of the percussion note at the left

apex, with a few sharp "clicks" at the end of inspiration. He had continuous temperature of about 102·4° F. He was treated with an acid tonic and complete rest in bed. On Nov. 22nd the acid was omitted; quinine and digitalis were given. On the 25th the temperature was gradually falling. He was still sweating much at night. On Dec. 5th the temperature had been remittent, but was continuously high again and was going up. The patient was sick a good deal. Large râles were now heard in the second, third, and fourth spaces in the left front and down the left back commencing from the apex of the lower lobe. On the 10th, 2 minims of guaiacol three times a day after food were ordered. He had gradually lost weight up to now. He was out of bed for an hour and could hardly stand. On Jan. 3rd, 1898, the patient was taking 15 minims of guaiacol three times a day. Fine râles were heard in the left front with some crepitations below. There were a few crepitations at the base in front. Râles heard at the back were much smaller. The temperature kept about 99°. He had put on 8 lb. in weight since getting up (Dec. 10th). On the 31st the râles were gradually drying up. He was taking 35 minims of guaiacol three times a day. He had put on 1 st. in weight. His temperature was normal. He complained of nothing except cough and expectoration the first thing in the morning. From Feb. 15th to 17th there was pain in the left base, friction was heard, and the chest was strapped. One drachm of guaiacol three times a day, all in capsules, was given with no ill effects. His temperature was normal. He was 1 st. 5 lb. heavier than on Dec. 10th. On March 7th the

patient went out. He expressed himself as perfectly well and strong. He had taken 1 drachm of guaiacol thrice daily up to now. He had gained 22 lb. in weight since December 10th, when he commenced guaiacol. There were no tubercle bacilli in the sputa when he left the hospital.

CHAPTER XV.

THE HYGIENIC OR FRESH AIR TREATMENT OF CONSUMPTION.

THERE is nothing new in the "new" treatment of Consumption. Over sixty years ago, Mr. Boddington was ridiculed for treating his consumptive patients in a similar manner to that which is now advocated by medical men, and talked about with wonder and approval by the public at large. But the prophet is not without honour save in his own country, and the work of men honestly practising what they believe to be good is not lost. Jealousy or ignorance around them may prevent their work receiving its due appreciation in their own country, or in their own time, but the force of jealousy diminishes with distance, and is annulled by time. Thus when, years later, the word comes from abroad that the treatment practised by Boddington is good, there is no lack of persons willing and anxious to adopt it. But it was not only the natural hesitation in accepting new modes of treatment, which prevented the open air treatment of Consumption coming into immediate general use. Sixty years ago, Consumption was considered to be chiefly caused by such climatic conditions as are peculiarly characteristic of this country. To expose the consumptive to the weather was to offer him a hair of the dog that bit him; it was placing him in the very midst of the conditions to which his illness

showed him to be dangerously susceptible; it was as though one should attempt to treat opium poisoning by giving an overdose of laudanum.

Believing that Consumption was a climatic disease, the open air treatment was a theoretical absurdity.

In the interval which separates us from Boddington's time, the etiology of Consumption has been the subject of much study and investigation, which has caused the older views of causation to be materially modified, and thus to remove one potent theoretical objection to the open air treatment. The collective observations from distant parts of the world, which the greater facilities for publication and reference made easily available to all, showed the disease prevailing under the most diverse climatic conditions wherever people were crowded together. The greater prevalence of Consumption amongst the dwellers in towns, and especially amongst those following indoor occupations, in itself indicated a dependence on something beyond climate. The belief that Consumption was in some measure infectious had never died out, though no proofs had been discovered that would justify its universal acceptance, till Koch verified what others had surmised, and made a certainty of what others had striven to demonstrate with only partial success.

The tubercle bacillus was shown to be the one determinating cause of Consumption; all else — climatic vicissitudes, occupation, hard living, previous illness, inherited constitutional weakness—all, singly or together, cannot produce Consumption without the infective organism. True, these various influences must still be reckoned as important factors in the etiology of the

disease; their effects render the body vulnerable to the attack of the disease-cause, and weaken the resistance of the tissues to the advance of the invaders. Even in treatment the influence of these predisposing causes cannot be disregarded; cure can hardly be brought about if the conditions which favour the progress of the disease are still operative.

Whilst exposure to the weather was looked upon as the main cause of Consumption, what wonder that sufferers from this malady were advised to keep themselves shut up in warm rooms and to avoid the open air? Even the discovery of the Bacillus tuberculosis and the demonstration of its modes of attack could not at once remove the ingrained fear of wind and rain. We in this climate have good reason to fear its treacherous variableness—apart from any question of its direct influence on Consumption. Thus it is that the open air treatment only becomes popular in England after it has for some time been accepted in other countries.

The chief interest in the open air treatment of Pulmonary Tuberculosis seems to me to consist in the fact that it involves a very important principle. It marks the application to the treatment of disease of hygienic rules, hitherto chiefly restricted to preventive medicine.

It is not only in Tuberculosis that the principle may be advantageously applied, nor is it only in diseases of the respiratory organs that fresh air is beneficial.

The hospital to which I am attached is now carrying out, as completely as circumstances permit, the treatment of Consumption by fresh air. Ten years or more ago, when I attempted to carry out in the hospital this prin-

ciple of treatment—which I have consistently advocated throughout—the windows which I caused to be opened were closed almost before I had left the corridor, and that not by the patients alone. Up to a few months ago, the dread of open windows, so marked amongst the poorer classes of our large towns, made thorough ventilation of the wards distasteful to the patients. A popular interest in a supposed new "cure" effected what no argument of the physician could encompass; and at the present time, although there is still some anxiety at first as to the risk, there is no opposition to the opening of windows even by the new-comers. Patients who previously dreaded an open window lie in bed on the balcony day and night, in rain or wind, and prefer this to being in the ward.

After the initial feeling of distrust wears off, almost all the patients express a liking for the freshness of the air in the wards, even when it leads to a strong wind blowing over the bed. But even with wide open windows it is impossible to keep the air as fresh as in the open, and the "fresh air" treatment only reaches its full development in the "open air," where many of the patients spend the day and some of them the night also. Broadly speaking, the essential factor in the "treatment" is to provide the patient with a full supply of pure air, so that there shall be no irritation of the organs by inhaled dust, and that the damaged lungs may perform their function of oxygenation with no unnecessary strain.

But apart from this *local* advantage of pure air, there is the general effect upon the body. The individual with perfect digestive power may assimilate sufficient nutriment from even "indigestible" foods; his diges-

tive organs may be severely taxed, but so long as they are equal to the strain, general nutrition does not suffer. With impaired digestive power, however, the organs can only accomplish a limited amount of work, and, unless the food is restricted to easily assimilable nutriment, the needs of the body will be incompletely satisfied. So when the lungs are diseased not only is functional rest a desideratum for the organs themselves, but with impaired respiratory power the oxygen-hunger of the body will hardly be satisfied unless pure air alone is supplied. With this provision the tissues may be prevented from suffering from want of oxygen, and retain or regain much of their vigour. Well-nourished cells may withstand the attacks of morbific organisms, and the spread of the tubercular mischief is prevented. If time is thus given for the degenerative changes, which will result in the removal or cicatrisation of the original tubercular focus, to be completed without the development of further centres of the disease, cure, or at least quiescence, may result. But time is needed, and the fresh air treatment is hardly a matter of a few weeks.

The provision of oxygen is, however, only one of the requirements of the body, and in addition to pure air we must supply the necessary nutriment, and keep up as far as possible the functional activity of all the tissues and organs. The selection of proper food in full quantity, the exercise of the muscles—unless high temperature shows an excessive expenditure of oxygen in the body—the proper measure of rest—all these need attention. Drugs may be required to assist digestion or assimilation, to relieve symptoms, to calm anxiety or to promote sleep.

Fresh air alone is not sufficient, and the "fresh-air treatment" includes much beyond living out of doors. As attention to other hygienic requirements of the body is as essential to the treatment as the provision of pure air, I prefer the term "The Hygienic Treatment of Consumption," which I have employed for several years, to the less comprehensive term which is now commonly used.

Sanatorium treatment implies the routine which is so beneficial to most patients, and the supervision which instructs patients to regulate their lives according to hygienic requirements. Patients need to be instructed as to their clothing as well as other things: consumptives are much disposed to over-clothe themselves.

The most successful practitioners of the open-air treatment are those who, having their patients in a well-regulated institution, give the fullest personal supervision to each case. If fresh air were all that is required, supervision would be unnecessary; but that every variation in the patient's condition may at once receive appropriate treatment, personal supervision by the physician is advisable. It must strike all who note the different regulations amongst the many institutions where the open-air treatment is practised, that in one, much stress is laid on the eating of a certain quantity of food; in another, overfeeding is as nothing compared with regulated exercise; whilst in still another rest in bed is the essential. But rigid rules which allow of no relaxation, whether they relate to food, to exercise or to rest, have one point in common; they mark the fact that whilst residing in the institution the patients are under strict orders. Enforced

obedience to one rule engenders a habit of obedience; and the strictest disciplinarians amongst the medical directors of these sanatoria get the best results, because their patients follow out implicitly the smallest details which the physician orders. The private patient at home is less particular.

A question which needs consideration is whether any climate will do for carrying out the open air treatment so long as the air is pure.

Undoubtedly some climates are more favourable than others. Dryness of the air seems to be a great desideratum, and a large measure of sunshine is also advantageous. The temperature of the air apparently matters little.

It must, however, be remembered that different cases require different conditions. Some do best in a bracing air—and perhaps this class includes the great majority of curable cases—others require the warmth of more sheltered situations. Many, but not all, receive benefit at high altitudes; some get rapidly worse at the seaside, whilst others improve. It is in some measure due to the want of full recognition of this necessary selection of climate for each case that such different results are obtained from apparently similar treatment. All cases do not bear life in the open air, in this climate at least. This is what we might expect. We all recognise in theory that the same climatic conditions are not suitable to all cases of Pulmonary Tuberculosis, and that the climate for residence should be carefully selected for each case; though in practice all kinds of cases are sent to the health resort which is at the moment fashionable. If the climatic con-

ditions are not suitable to a particular case the disadvantages will be most obvious if the patient is fully exposed to these climatic influences. The patient undergoing open air treatment experiences the fullest effects of the climate, whether this be beneficial or otherwise, and if in any case we select the climate most suitable for the patient he will undoubtedly derive more benefit by living in the open air than by remaining much of his time indoors. Before advising a patient where to go we have to take into consideration something beyond the relative degrees of popularity of different sanatoria. Even the selection of the most suitable climate for a given condition of tubercular disease does not always settle the matter, for all patients cannot travel where they would. We may allow the superior advantages of other climes, but since so many consumptives cannot go far in search of health it is as well to recognise that fresh air is to be had in these islands, and that the open air treatment may be successfully carried out nearer home.

As the fact of keeping patients in the fresh air, or even in the open, is at the present time attracting attention both within and outside the ranks of the medical profession, I may note a few observations from my experience of this treatment.

Although, as I have already mentioned, all cases do not bear the open air life equally well, I have seen no ill effects from consumptives sleeping in the open.

If the patient is kept warm, there is little risk of catching cold. Colds will occur, but I am of opinion that they come from infection (by visitors) not from exposure. Sometimes cough is increased at first if the air is sharp

and bracing. Exposure to wet appears sometimes to induce bronchitis.

One effect of the exposure is, in most instances, to lower the temperature, and in those patients whose temperature falls considerably below the normal the low temperature may approach that of collapse. This is probably most likely to occur in patients whose temperatures show a wide diurnal range. I would here note that the oscillation of the temperature is an important point to note as well as the absolute height. Patients who have been living for a time in the open air get a somewhat higher temperature if they go indoors for a night or two.

The general results seem to be that appetite is improved, the temperature reduced, and weight increased almost from the first. Improvement in the physical signs, and diminution or disappearance of the bacilli, take several weeks.

Hæmoptysis is not a contra-indication for open air treatment. In my patients who have been living in the open there seem to have been few cases of hæmoptysis, and where it has occurred the hæmorrhage has been comparatively slight. This may, of course, be a mere coincidence.

Two illustrative cases may here be noted, the charts showing the improvement in the temperature under the fresh air treatment. In one case the gain in weight is also recorded.

H. R. Male, æt. 38. Admitted March 9th. Eight weeks before admission had influenza and left-sided pleurisy. A month later he had hæmoptysis (1 pint) repeated twice during the week. He has had an evening temperature of about 102·5° F. until admission.

CHART I.

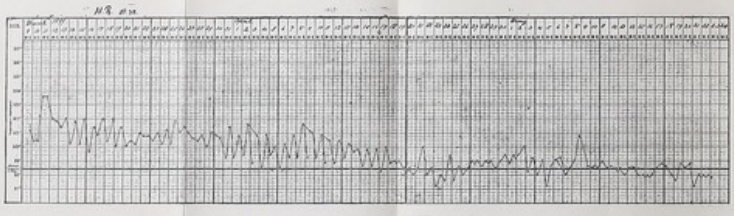
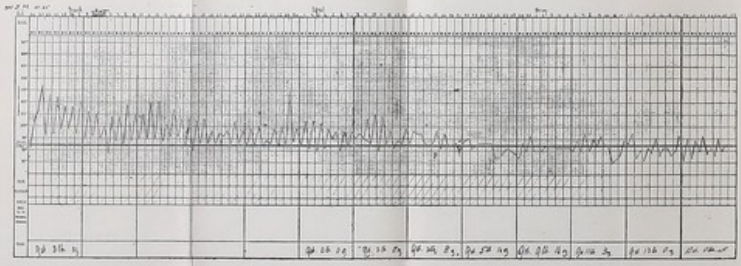
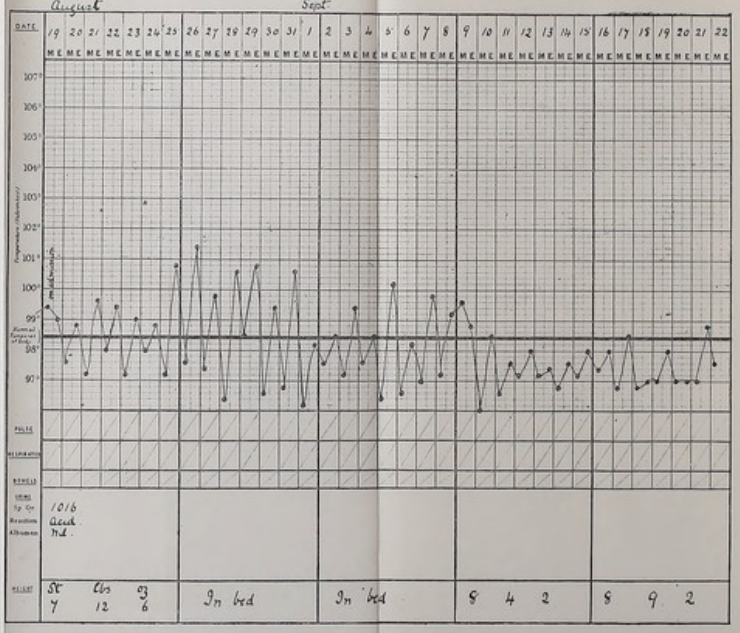


CHART II.



These Charts, I., II., III., NOT to be cut, but folded and fixed to face page 199.

CHART III.



The physical signs showed acute Tuberculosis, involving the whole left lung, and the apex of the right. He spent all day in bed on the balcony (and one or two nights also), and on discharge at the end of May the right lung was clear, and the left side, which was considerably retracted, showed but a few râles. For the first two months in hospital he was too ill to be weighed regularly, but the improvement can be seen by the temperature chart (Chart I).

W. J. M. Male, 25. Had been in the hospital a year ago and left much improved. He continued well for nine months, when he had hæmoptysis and had to give up work.

On admission fluid was found in the left pleural sac. He remained in hospital from February 24 to May 23, when the ward note states "goes out practically cured."

His weight, which kept at about 9 stone until the beginning of April, was exactly 10 stone when he went out. Tubercle bacilli, which had been found in the sputa on admission, were now absent.

The temperature chart (Chart II) further illustrates the improvement.

A third chart of a patient recently in the hospital shows similar improvement both in temperature and weight (Chart III).

These cases have been selected merely because the patients have only recently been discharged from hospital. My case books for the past ten years show similar improvement in temperature and weight, with corresponding improvement in the lung condition in a large proportion of the cases. I have always made a point of keeping my patients as much out of doors as possible, and having the air in the wards kept pure by free ventilation. It is not only in Phthisis that I have relied on fresh air in treatment. In pneumonia, bronchiectasis, asthma, and empyæma, amongst other ailments, I have always advocated open windows and as much fresh air as possible;

and in 1889 I published (*a*) a successful case of gangrene of the lung, where, during a cold March, the windows of the ward were kept wide open day and night. Chronic bronchitic patients, however, cannot usually stand the fresh air to the extent that other patients can do. Even in the warmer weather the bronchitic patients in the hospital avoid, as far as possible, the rooms and corridors through which the wind is blowing, and the wards occupied by these patients have to be kept less freely open to the air than the other rooms.

We must be careful that the "fresh air" treatment is *thorough* whilst it is being employed. It is not enough to let a patient spend a certain portion of the day in the fresh air, if he breathes impure air for the remainder of the twenty-four hours. It should be absolutely forbidden for the patient to enter a close room—even such as the public rooms at an hotel, or a place of public entertainment. There is a prejudice against "night air" which must be overcome. The air is probably purer during the night than in the day, though, in some situations especially, it may contain more moisture, and it is always cooler than during the time the sun is above the horizon. For this reason the patient requires to be warmly covered if in the open air at night. Indeed, one important point in open air treatment, is to keep the patient warm whilst freely exposed to the air. So long as the mouth and nose are uncovered, there is no necessity to expose any part of the body. Cover the patient as much as may be required to keep him comfortably warm, but at the same time

(*a*) *Lancet*, July 20th, 1889.

avoid any restriction of respiratory movements. It has long been a rule with my female patients in hospital that stays are never to be worn whilst they are there. It is not entirely a question of restricted movement of the chest; the work of holding up the body, thrown entirely on the muscles when no corset is worn, strengthens them and soon leads to a better carriage of the body, and tends to enlarge the chest and increase the respiratory capacity. For similar reasons some carefully regulated "gymnastic" exercises in the open air may be recommended in quite early cases, and in my experience have had most beneficial results. Simple gymnastic exercises are most useful in those who are predisposed to consumption, as well as in early cases of tubercular disease of the apex. I have been accustomed for many years to recommend such exercises, and am convinced of their utility.

I am not as yet convinced that forced feeding is essential, or even always desirable. The necessity for a full supply of nourishment is evident, and I encourage patients to eat a good quantity of food and endeavour to assist their appetite, by drugs if required, and by variety in diet. But, however useful or necessary it may be found in a large institution to lay down hard and fast rules to ensure that none of the patients get an insufficient quantity of food, I am not sure that it is "necessary to work by rules," as I have lately seen it expressed—unless, indeed, we are prepared to make numerous exceptions. If we take the text-book description of a disease to represent the rules for that disease—as I think we may do—we find that each patient presents some exception to the rules in one or more particulars. We learn the rules, but we

expect to find exceptions; so also when we lay down rules for treatment, we must be prepared to modify them according to the requirements of the individual. The student, before he has had clinical experience, knows only the rules, and would treat by rule. Experience shows that the individual patient is generally an exception from the rule, and that rules of treatment, necessary as they are as a guide to principles, are not to be too rigidly adhered to.

There is a danger of looking upon a gain in weight as the object to be attained. A patient who is putting on weight is most probably improving, but the gain in weight, though a sign of improvement, is not always a true guide to its extent. I have frequently seen an advance in weight without material improvement of the disease. Forced feeding may perhaps help patients to put on weight at first, and may from this have been credited with an importance beyond its merits.

Of the many drugs advocated at different times for the treatment of Consumption, those which have stood the test of time and experience are those which tend to improve the nutrition of the body, not those which were supposed to have a specific action on the disease-centres in the lungs. The duration of the malady and the pathological changes produced by the tubercular process militate against the success of germicide drugs in curing the lesions of Tuberculosis, though these may be useful in preventing, or limiting, the spread of the disease to new centres within the body.

Drugs are valuable aids to treatment; but so-called "active treatment" is not always essential, and is rarely necessary throughout.

Let us attempt to realise what the fresh air treatment of Consumption connotes. Shorn of all the non-essentials —the accessories which may be added to make it attractive or remunerative—it is the application to one disease of an important principle, which may be applied to many other diseases. It marks the application of hygienic rules to the treatment of disease instead of only to prevention—an era in treatment soon, let us hope, to be extended to other maladies. It is perhaps natural that the value of fresh air should be first acknowledged in the treatment of pulmonary diseases, as dieting in digestive ailments. But physiology teaches us the dependence of all organs on each other and of the whole body on the efficiency of each part. Pathology and clinical observation emphasize this physiological principle. A diseased organ may need the administration of drugs to restore its functional efficiency, but its recovery may be greatly assisted by attending to the physiological requirements of other organs, of which fresh air is by no means the least.

CHAPTER XVI.

THE HYGIENIC TREATMENT OF CONSUMPTION AT HOME.

It has for some time been recognised that attention to hygienic requirements forms one of the most important means of *preventing* Tuberculosis in the individual. With those who are specially predisposed to Tuberculosis, and particularly in the case of delicate children of consumptive parents, much may be done by careful hygienic methods to strengthen the resisting power of the tissues against invasion by the tubercle bacillus. By a well-regulated life in a healthy home the predisposition may be overcome, and the weakly child may grow into a strong and healthy adult. The hygienic regimen adopted to overcome predisposition must be carried out at home in the large majority of cases, and it is possible also to apply similar measures where a consumptive patient has to be treated in his own house. To quote the words I used in a book published seven years ago (*a*), "The hygienic rules so important as preventive measures are equally necessary conditions in the treatment of the disease."

The chief hygienic requirements for treatment consist in the provision of plenty of fresh air and good food, with

(*a*) *The Hygienic Prevention of Consumption.* Chas. Griffin & Co., London. 1893.

properly regulated exercise and clothing; drugs may be required to assist the patient to obtain the full benefit from his food and to relieve distressing symptoms. The open air treatment is a development of the treatment by hygiene which it is not always easy to obtain at home, but fresh air can be admitted into a private house as well as into a sanatorium. It is, of course, true that it is usually more easy to carry out hygienic treatment satisfactorily in an institution, where the patients are under supervision and where they are subject to rules. Few people who have not studied the subject realise the existence of natural laws for healthy living, and, consequently, if they have contracted Tuberculosis they are unable to apply these laws to themselves. They require, therefore, the guidance of careful directions, and constant supervision to ensure that these directions are followed out. So, too, in the varying phases of their malady the rules of life may require modifying from time to time, and here again experience is needed to direct. Residence in a well-ordered establishment, whether hospital or sanatorium, is therefore a great advantage, especially with the poorer class of patients, whose homes are not the most healthy, and whose previous habits are most at variance with hygienic requirements. Even the well-to-do are often in need of instruction as to the manner of life most suited to their health. But all cannot, and some will not, enter an institution, nor can those who are admitted be always kept until completely cured.

Again, there is one great disadvantage in sanatorium treatment which it is difficult or impossible to avoid: rules must be made, and when made must be enforced. All

patients are thus under the same regulations, and no allowance is made for personal peculiarities or for differences in the disease-condition of each individual case. Treatment by routine is, as we all know, less satisfactory than treatment adapted to the individual and his special conditions. It becomes, then, sometimes necessary or advisable to treat the patient at home, or at least in a private house as opposed to a sanatorium. When this has to be done the medical adviser must lay down the rules of life for his patient. The day must be methodically parcelled out, the times for meals, for rest, for exercise, &c., being definitely stated. The daily time-table should be written out or printed, and the patient must undertake to obey the rules laid down.

The following, which is in use in the North London Consumption Hospital, will serve as a guide :—

PATIENTS' ROTA TABLE.

All patients who are undergoing the "open air" treatment and who are up all day must strictly observe the following table unless ordered otherwise.

7.30 to 8 a.m.	Up, washed and dressed.
8.15 a.m.	Breakfast.
8.45 a.m.	Rest on seats and couches.
9.30 a.m.	Recreation (reading, writing, smoking, &c.).
10 a.m.	Lunch.
*10.30 a.m.	Exercise in grounds or on Heath.
12 noon.	Rest on seats and couches.
12.30 p.m.	Dinner.
1.30 p.m.	Rest on seats and couches.
2 p.m.	Recreation (reading, &c.).
*3 p.m.	Exercise on Heath or in grounds.
4.30 p.m.	Rest on seats or couches.
5 p.m.	Tea.

5.30 p.m.	Rest on seats or couches.
6 p.m.	Recreation (reading, &c.).
8 p.m.	Supper.
8.30 to 8.45 p.m.	Bed.

* During wet changeable weather the times of exercise may be altered.

Hats are not allowed in the grounds, and patients are advised not to wear thick overcoats when taking exercise. Patients must not talk, except to officials, during meals and after going to bed. Smoking is only allowed during the recreation periods. Patients who are up for a portion of the day only must follow the table during the time they are up.

Supervision will generally be required and is always advisable at first. If there is any difficulty in getting the patient to observe the necessary routine, a nurse who understands what is required is desirable—preferably one who has had experience of the treatment in some institution. Instructions as to the time to be spent out of doors, the ventilation of rooms, the diet and the clothing must be clearly detailed.

Change of scene is sometimes useful, and with a good and reliable nurse or attendant a patient may move about the country, making a stay of a few weeks or months in each place when the weather is satisfactory, and moving on if the weather becomes unsettled—"chasing the sunshine," as I have often expressed it. I examined this summer a young lady, whom I first saw in the country six or seven years ago with acute tubercular softening at one apex. On my advice she followed the above plan for a year or two, and finally settled at Bognor, where she is now enjoying life and is practically cured. Occasionally, also, there may be an advantage in leaving home to get

away from other members of the family. We may persuade the patient to keep his rooms thoroughly ventilated, and after a short time he will probably prefer to have the windows always open. In the cooler weather this free ventilation may be too much for the other members of the family, who either want the windows closed when they visit the patient, or persuade him to sit for an hour or so with them in the evening in a warm and probably close room. This is neither good for the patient nor for the other members of the family, and it may be best for all if the patient leaves home for a time.

It is probably true that we cannot carry out the hygienic treatment so completely at home as in a sanatorium, but in my experience such treatment can be carried out with success. Even in London, where the conditions are not the most favourable, much can be done in this direction. In cases where it is impossible to get all the hygienic requirements, it is well to insist on attention to such as can be obtained, for much good can be done by even a modified hygienic treatment. In any case, and under any treatment, attention to hygienic rules is necessary and helpful. We may, and do, meet with opposition to our suggestions for open windows, cleanliness and other matters, on the ground that the patient has always lived under certain conditions, and that others have done so also without harm. We must remember, however, that whilst a person in health may adapt himself to his surroundings, it is necessary in treating disease to modify the environment to suit the individual. The sound man may retain health under conditions which would make it impossible for the sick to regain health.

Part IV.

CLINICAL OBSERVATIONS.

CHAPTER XVII.

PHYSICAL EXAMINATION AND DIAGNOSIS.

The importance of detecting Tuberculosis of the lung as early as possible—since only in the early stage is absolute cure to be anticipated—is so great that we must not risk overlooking physical signs of the disease by incomplete examination. Microscopical detection of tubercle bacilli may be useful to confirm an opinion arrived at by physical examination, but until there is cough with expectoration this mode of investigation is generally impossible. But cough with expectoration is not always—one might say not frequently—one of the earliest symptoms of pulmonary Tuberculosis, and we must endeavour to make a diagnosis and to commence treatment without waiting for sputa. The physical signs on which we have to rely in these early cases consist in slight deviations from the normal, which, though very distinctive when recognised, may escape notice if due care is not exercised in examination. This being so, it is essential, where there is any question of Tuberculosis in the lung, that the chest should be fully uncovered, and the examination be conducted thoroughly and methodically.

I have spoken above of deviation from "the normal." It must be remembered, however, that the sounds heard

in health on percussion or by auscultation differ somewhat in different individuals. The size of the thorax and the thickness of its muscular and fatty coverings will make some difference apart from anything else. There is no universal standard to which we can refer as the "normal" sound, either in percussion or in auscultation. We have therefore to determine the normal for each case.

I have frequently pointed out in clinical teaching that in a quite early stage of Tuberculosis of one apex, it is difficult, or sometimes well-nigh impossible, with a single examination to say which is the diseased lung, although it is quite possible to say with certainty that there is mischief in one. I have known competent observers differ as to the lung which was affected, though they agreed that there was tubercular disease of one apex. The explanation is simple, if we recognise that we need to establish the "normal" for each case; it is easy to say that if one side is normal, then the other, which differs from it, must be abnormal, though it may be difficult to say which must be taken to represent the healthy standard.

For physical examination, the chest must be fully exposed, otherwise we are unable to see any slight flatness on one side, or asymmetry of any kind; neither can the equality or otherwise of respiratory movements on the two sides be appreciated. Articles of clothing not only obscure inspection, but they interfere with free respiratory movement, and may give rise on auscultation to sounds which are at the least misleading. I have noticed the disappearance of a well-marked friction sound when a pair of leather braces was taken off. So, also, every part of the chest must be examined, not merely the apex only;

it is not enough to note the mere fact that there is mischief in the lung; its distribution and the stage to which it has advanced at any one part must also be determined.

The examination should, then, be conducted systematically, taking in order Inspection, Palpation, Percussion, and Auscultation.

I am in the habit of commencing percussion on that side of the chest which, on inspection, appears to be of good shape and to move most freely in respiration. This being presumably the more healthy side, the percussion note obtained on this side is most likely to indicate the patient's normal note; it will at least approximate more nearly to the normal than the note over a portion of the chest which shows flattening or diminished movement. Where there is a scar or other result of previous mischief in one apex, it may happen that the percussion note is more resonant over this place than on the opposite and undamaged lung. This is then due to compensatory emphysema around the collapsed or consolidated area of damaged lung tissue. The deficiency of movement and the character of the sounds heard on auscultation over the damaged apex will, however, correct any misapprehension caused by the percussion note. So, also, with auscultation, I listen first on that side which has given the better percussion note in order to establish as far as possible the standard breath-sound for the particular case.

In percussion it is often of value to make direct percussion on the clavicle, or on the ribs in thin subjects: when there is any doubt whether the note is equal in pitch over the apices on the two sides, this direct percussion is of

great assistance. It is difficult to ensure exactly the same amount of pressure of the pleximeter finger on the chest, and a difference of pressure will make some difference in the note elicited on percussion even on the same spot. The bone, however, keeps the same relative position with regard to the subjacent parts, and partial consolidation only suspected on percussion upon the finger becomes manifest without reasonable doubt when direct percussion upon the clavicle is employed.

Any instrument for percussion is less good than the fingers, as we lose the valuable aid of the sense of resistance, which by practice becomes as important as the sounds produced. For demonstration purposes a plessor and pleximeter may be useful to bring out the sounds so that they can be well heard by a class; but for the purposes of making a diagnosis the hands alone should be used for percussion.

Just as the note elicited by percussion over a damaged apex may sometimes seem to be better than that over the sound lung, because it approaches more nearly to what we should expect to find in a healthy person with a good chest, so we may also be misled at first by the breath-sounds heard on auscultation. We naturally carry in our minds some idea of what we consider a normal breath-sound as regards tone and loudness. In a patient whose lungs in health give a very weak sound on respiration we may, when tubercular mischief has commenced at one apex and has caused the breath-sounds there to be louder from better conduction, consider these exaggerated sounds to be more nearly like our theoretical standard than the weak breathing which is the individual's "normal." If, however, we

pay due attention to the relation between the inspiratory and the expiratory sounds we shall be able to correct this fallacy, which will be fully exposed if adventitious sounds (*râles*) be heard. These *râles*—or the significant moist "click" at the end of inspiration—may not be heard on quiet breathing, and the patient should therefore be directed to take a "deep" or "full" breath.

In listening to the breath-sounds the patient should not be asked to take a deep breath until the breath-sounds during quiet breathing have been noted; the mere fact that these are almost inaudible is in itself important to note.

In auscultation the one point which is common to all abnormal breath-sounds is the prolongation of the expiratory sound. We must not neglect to note undue conduction of heart-sounds, especially on the right side of the chest. At the base a thickened pleura will conduct the heart-sounds round to the axilla, and may suggest consolidation of the lung; this may occur even when there is fluid in the pleural cavity.

For whispering pectoriloquy I usually get the patient to whisper the words "fifty-five," using the words "ninety-nine" to bring out the resonance of the full voice-sounds.

But although a careful method of conducting the physical examination is essential, it is not in itself sufficient to ensure a correct diagnosis. We need training and experience to appreciate the various physical signs and to deduce from them the condition of the organ under examination. Training teaches us to recognise the various signs, to distinguish one from another, and to give to each its name. Experience alone enables us to draw

from the physical signs a mental picture of the organs within the thorax, and of the pathological conditions which give rise to the sounds which we hear. Except for purposes of teaching or of discussion, the exact name which should be given to any sign is of little moment; what the signs mean—the interpretation of the signs—is of the utmost importance. To teach us to interpret aright there is nothing so valuable as to follow the autopsy of cases which we have carefully examined during life, and in the opportunities afforded for doing this lies one of the main advantages of a hospital appointment.

The process of physical examination may be illustrated by the following clinical lecture to students on a case in hospital.

This youth—a shop porter aged sixteen years—was sent here said to be suffering from Consumption; and, so far as his general appearance on admission was concerned, it seemed probable that this supposition might be correct.

But although we may often get valuable indications from a patient's appearance and physiognomy, I constantly point out that hints we may obtain from sight are by no means sufficient to guide one to a diagnosis. During the progress of the examination of a patient, we frequently come upon some sign which seems fully to explain the illness; but though we may form theories as we go on, we must be prepared to throw them over if further examination fails to corroborate them. Not until we have completed our examination—not until we are in possession of *all* the facts which are available—are we in a position to give an opinion on the patient's illness.

The processes through which a physician has to go in examining a patient may be compared to the judicial procedure in a court of law; and these processes should be worked out as systematically, as carefully, and as thoroughly in medicine as in law. The issues at stake are fully as important to the patient as to the individual standing his trial.

To follow up the simile, we first hear the indictment—that is, the idea of the patient or of his friends as to what he is suffering from. We then proceed to take evidence. First, such symptoms as the patient is able to describe, *e.g.*, pain, dyspnœa, cough, etc. Such matters require corroboration from the observation of others (doctors, nurses, or friends) to make them of much value. Evidence likely to bear on the case is also sought, both from the patient himself and from friends, by asking the family history, and the personal medical history especially with regard to the present illness. We then proceed to collect such evidence as we can derive from methodical physical examination of the patient. Having elicited all the available evidence, we proceed to sum up—sifting and weighing the evidence and assigning to each fact and circumstance its due value. Then, and not till then, we can proceed to pronounce the verdict, and follow this by the advice and treatment suitable to the case.

Pursuing this course with the patient before us, we are called upon to decide a case presumed to be one of Consumption. The evidence of the patient tells us of slight cough and dyspnœa, emaciation, and some sweating at night. The history of the onset is as follows. The lad enjoyed good health up to the present illness. A month

ago he felt giddy one day, and as he also had a bad cough he went to see a doctor. He was ordered to bed. He had a good deal of shivering, repeated but not severe rigors. He has never had any marked shortness of breath, nor has he ever expectorated with his cough. He has been "feverish." There has been no pain in the chest, and he can lie in comfort on either side. So far the evidence is not opposed to the presumption of Phthisis; but we may note that the onset of the illness was somewhat sudden and was attended with rigors, a history more suggestive of pneumonia or pleurisy. We may incidentally remember that the onset of pneumonia is generally marked by a single severe rigor, whilst at the commencement of pleurisy the shivering fits are less intense but often repeated. The family history shows a freedom from Consumption; but in my opinion the family history is more important for prognosis in Phthisis than as an aid to diagnosis. The patient's occupation is not one which specially predisposes to Phthisis. We now come to the physical examination, which furnishes us with the most reliable evidence of all, for here there is no chance of the patient misleading us, whether intentionally or unwittingly.

The lad's appearance on admission a few days ago showed marked pallor and emaciation—severe but not acute illness. Even in this short time there is considerable improvement in his colour, and he looks brighter.

The chest is well shaped and symmetrical, except that the right side seems very slightly fuller than the left. The tape measure shows a difference of half an inch only.

The movements in respiration are much diminished on

the right side, and especially so in the lower part of the chest. In Phthisis the diminution in respiratory movements is usually most marked in the upper portion of the chest.

On percussion we find good resonance over the whole of the left side.

On the right side in front there is resonance over the upper portion of the chest of a peculiar tympanitic character suggesting the stomach note (Skodaic Resonance); below this is dulness, getting absolute as we pass downwards, and continued over the liver to three fingers' breadths below the costal margin. The upper margin of this dull area slopes upwards from the fifth space near the sternum to the third space in the anterior axillary line.

In the axilla the dulness extends as high as we can reach for percussion, and at the back its upper margin slopes downwards to the spine. The heart's apex is felt beating about one inch immediately below the left nipple, the heart being slightly displaced outwards.

We are now justified in doubting whether the presumption of Phthisis covers the whole case, or can even be sustained by any evidence at present obtained. The curved upper limit of the dull area on the right side, rising to a point in the axilla, already suggests the presence of fluid in the pleural cavity—a hint which must not be neglected, but which is worthless without confirmation from further examination.

Auscultation over the left lung furnishes no morbid signs, though the breath sounds are puerile.

On the right side we hear the breath-sounds down to and below the upper margin of dulness, losing them

altogether at the base. Note that even when there is a considerable quantity of fluid in the pleural cavity we may hear the breath-sounds—distant but distinct—and the heart-sounds may be plainly conducted over the dull area, especially if the pleura be thickened. On admission there were a few crepitant râles to be heard in the upper part of the right lung, but they have disappeared now.

Vocal fremitus is diminished over the dull area on the right, and is quite absent at the lower part of the chest on that side.

We are now convinced that there is fluid in the right pleural cavity, but that is by no means all we have to decide before we can suggest the treatment or forecast the probable result of the illness.

The further points of importance for decision are (1) the nature of the fluid—whether serous or purulent—and (2) the possible dependence of the effusion on Tuberculosis.

There is only one certain means of deciding the nature of the fluid, viz. the withdrawal of a small quantity by aspiration; and the resident medical officer will obtain this for us while we discuss the indications which might give us some idea of what to expect.

These indications cannot be relied upon, and with such a simple means of definitely deciding the point as by aspiration it is not necessary to say much on the subject. The marked appearance of illness of the patient on admission suggested the possibility of empyæma, but he has improved greatly in the past few days.

The absence of *pectoriloquie aphonique* has been said to indicate the purulent nature of the fluid—this sign we fail to find in this case. The temperature, which ran up to

103° F. on the evening of admission, still reaches 100° and 101° every evening, though the lad has been ill over a month, and he perspires at night. The pulse is rapid, beating 152 times a minute.

All these signs point to the fluid being purulent, yet as we see now the fluid is clear serum. I confess I am both surprised and relieved.

As to the possible association of the pleurisy with Tuberculosis, we have as yet little to guide us in this case. That many pleurisies are tubercular is undoubted; and that many persons, apparently free from tubercle when attacked with pleurisy, subsequently develop Consumption is unquestionable.

In the present case the absence of signs of Tuberculosis in the lung, and the freedom of other organs from disease, justify a hopeful view.

It is too early in the disease to base any opinion on the temperature, and in the absence of expectoration we cannot look for bacilli. The ultimate prognosis must in any case be guarded, in view of the increased liability to Phthisis which results from an attack of pleurisy with effusion.

The treatment will be mainly tonic, and we shall not attempt to remove the fluid by aspiration until we have given time to see if absorption takes place.

Working out this case in a methodical fashion, it seems impossible that anyone could have failed to diagnose pleuritic effusion. But you must not make the mistake of supposing that the detection of fluid in the pleural cavity is always an easy matter. I have seen several cases in consultation lately where the presence of fluid has escaped the notice of careful observers. I have also come across

instances where, the chest having been examined at the commencement of an illness, further examination has not been considered necessary, and fluid has subsequently been effused unnoticed.

The former accident may happen to any of us, the latter oversight is incompatible with proper professional care.

I can also recall recent instances where persons have been treated for "rheumatic" pains in the side, which have been explained when a short time later fluid has been found in the pleural cavity.

CHAPTER XVIII.

PHYSICAL EXAMINATION AND DIAGNOSIS (*continued*).

A Case of Heart Disease.

In the previous Chapter the importance of systematic examination is emphasized; but even with careful examination it is not always possible to detect mischief which may be suspected from the history of the illness. The following clinical lecture illustrates this difficulty in a case which presents several points of interest, and in which a Tuberculosis of the lung, suspected from the character of the temperature chart, could not be detected on physical examination, as the signs were masked by bronchitic râles. It not infrequently occurs that the signs of a Tuberculosis in the lung are so masked by loud bronchitic râles, and only become manifest when the bronchitis clears up. In this case death occurred before there was time for this to take place, and the autopsy showed an old and encapsuled tubercular area which had recently broken out afresh.

This patient, who has been admitted since my last visit, is a broad, well-built man of 46 years of age. He is 5 ft. 7 in. in height, and weighs only 9 st. $3\frac{1}{2}$ lb.—his ordinary weight in health having been 10 st. 11 lb. He

complains of cough and of weakness. As he lies in bed he has some obvious difficulty in breathing, the respirations being rapid. Cough is frequent and irrepressible; the expectoration is abundant, white and frothy, with some clear glairy mucus. The temperature shows a wide diurnal range, with evening fever (103° F.).

The history is interesting, but gives no very clear indications to guide us in seeking the explanation of his illness. For seventeen years the patient was a soldier, and served fifteen years in India (Bengal). Here he had malarial fever—tertian ague. From India he was ordered to South Africa, and spent two years in the Transvaal before returning to England. Since leaving the army, nine years ago, he has been employed as a labourer on the Great Western Railway, with somewhat heavy work. Three months ago he was seized on one occasion whilst at work with sudden palpitation and faintness, or feeling of suffocation. This has not recurred. He has had occasional cough during the last two years; but before this had enjoyed good health.

Let us see what points we may gather from this history. The two clear medical facts are the malarial fever—which may have left its mark—and the anginal attack a few months ago.

The patient has been fortunate in that he has had no return of ague since he left India. Very often, persons coming home direct from a malarious district get a bad attack of "fever" on reaching the colder weather in the Channel; possibly, in the present case, the two years in a warm but not specially malarious country, saved him from this frequently noticed effect of a sudden change from

India to England. It is worth remembering that invalids coming home from malarious districts may advantageously break the journey for a few months in a warm sanitorium, so as to avoid arriving in England during the cold season.

This man's long service in the army, much of it in a warm climate, suggests the possibility of a free consumption of alcohol, and though there is no reason to suppose that he has been intemperate, we must not exclude the possibility of finding some evidences in the internal organs of the habitual use of alcohol.

Service in the army has been said to predispose to certain forms of disease of the heart or of the large arteries, or even to cause these. This has been explained as due partly to the over-distended condition of the chest, which the recruit is made to assume in learning the "position of a soldier," and which becomes habitual as part of the martial bearing. The exertion of long marches, with the weight of the soldier's pack, is also held responsible as throwing strain on the circulatory organs.

We will now examine the patient.

The chest is well developed, and the thorax rounded. Movements in respiration are fairly good, and equal on the two sides. When the hand is placed over the cardiac region evidence is felt of over-action of the heart, the impulse is diffused, and a thrill is experienced apparently synchronous with the heart-beat. This observation gives us a special reason for a careful examination of the heart; but we will follow our usual practice, and commence with the lungs.

The percussion note over the front on both sides is

resonant, and though slightly high-pitched is of good quality. One notices, however, that the resistance of the chest on percussion is greater than would be experienced on a normal chest. At the back we have similar signs in the upper part on both sides, but towards the bases of the lungs the note becomes higher-pitched and "sharper" or "shorter"—*i.e.*, less resonant. This is particularly noticeable at the right base, where we have a very instructive series of gradations of sound. Above, the percussion note is fully resonant, a partially "sustained" note; below this we notice that the pitch of the note is raised slightly, and with this the sound is shorter and less sustained, or more *staccato;* below this we come to the almost wooden sound over the liver; there is still a "tone," but it is very sharp—*staccato*—a momentary note on the tap of the percussing finger, not prolonged by resonance as were both the former though in different degrees.

In percussion we have always three points to observe: (*a*) the *pitch* of the note produced; (*b*) its *duration*—whether prolonged by resonance, and to what extent; and lastly (*c*) the feeling of *resistance* to the hand, which is placed on the chest, when struck by the percussing finger. This last point is necessarily lost when any contrivance replaces the finger as a pleximeter. Dr. Ewald, in the Augusta Hospital in Berlin, was accustomed to use a thin piece of hard india-rubber (ink-eraser) placed on the chest, which was struck with an ordinary percussing hammer (plessor) with an india-rubber tip; and for demonstrations to a large class this was most effective in producing a note that could be easily heard by all.

Whilst on the subject of percussion let me draw your attention to the essential importance of placing the left hand *firmly* on the chest; the efficiency of percussion depends quite as much upon this as upon the firmness of the blow. The percussing finger should strike firmly, but at the same time lightly, the wrist being kept loose; above all avoid a pushing blow. One advantage of direct percussion over a bone—clavicle or rib—without the intervention of the finger of the left hand, is that the bones have a fixed relation to the underlying lung, whereas the finger may not always be equally firmly placed on the chest. Thus percussion on the clavicles is a most valuable means of comparing the apices of the two lungs, as minute differences in the percussion note are then easily distinguished. With the finger placed on the chest we may have the note altered by differences in the pressure on the chest, even over the same spot, and thus the two sides may give different sounds on percussion from causes outside the lung, *i.e.*, depending upon the examiner.

To continue our examination of the patient—*auscultation* confirms what we might suspect from percussion. I may perhaps be allowed again to digress for a moment to remind you that in all examinations of the chest each stage should be proceeded with systematically; inspection, palpation, percussion, and auscultation; and though at each stage we mentally form some preliminary ideas of the condition of the organs within, these ideas must be subject to correction by the result of the further stages of the examination. They may be confirmed, but we must be prepared to throw them over otherwise; no *conclusions* are possible until the *whole* of the evidence is before us.

In this case the weak breath sounds and prolonged expiratory sound show loss of elasticity in the lungs, and tend to confirm the suspicion, raised by the character of the percussion note and the resistance felt on percussion, viz., that the lungs are somewhat fibroid and emphysematous. Further, we hear over the right side numerous large liquid râles of bronchitis, and some sonorous and sibilant rhonchi are found on the left. At the bases behind, over the area of diminished resonance, we hear the large crackling râles of congestion, also most marked on the right side. Here we have evidence of inefficient pulmonary circulation.

On examining the *heart* we find the apex beat directly under the left nipple, instead of well within that position. The impulse, though forcible, is heaving and ill-defined, and a thrill, or rather a rub (friction fremitus), is felt. We notice, now that the patient has placed one hand behind his head, that the brachial artery is clearly visible, tortuous and pulsating.

The area of cardiac dulness is enlarged towards the left.

Auscultation discloses a loud and prolonged murmur at the apex, commencing apparently before the beat and replacing the first sound. Coming to the base, two murmurs are heard; and following the aorta up to the second left costal cartilage we lose the apex murmur, and distinctly recognise a systolic murmur of different pitch from that at the apex, and, in addition, a clear diastolic murmur. There are no signs of enlargement of the aorta (aneurism). The visible pulsation of the brachial artery prepared us for an aortic regurgitant murmur; but in

addition we find aortic obstruction, with regurgitation and probably obstruction also at the mitral orifice.

Here, then, we have the explanation of the anginal attack mentioned in the history. It is, however, curious to note how, with this exception, there is nothing in the patient's symptoms or history to lead us to expect so serious a condition of the heart. This case exemplifies the importance of thorough examination; without it we might have considered bronchitis to be the chief, if not the only trouble.

We will now complete our examination by learning the condition of the abdominal organs. The *liver* is apparently slightly diminished in size, the percussion dulness extending from the seventh rib in the nipple line to the costal margin. We have then probably a cirrhotic liver, as well as cirrhotic lungs; and the hard, tortuous arteries suggest that fibroid degeneration has also affected not only the vessels but many, if not all, of the organs of the body. The *spleen* is found to be enlarged downwards, though not much forwards—a reminiscence of the malarial poisoning in India. The urine is clear, acid, specific gravity 1020, and free from albumen or sugar, so there is no interference with the renal functions. The temperature is peculiar and somewhat puzzling, the large daily range—from 97·4° to 103° F.—suggesting something apart from the bronchitis; but as the patient has only been in hospital two days we may reserve further comment. The lung trouble we may put down as merely an incident due to the disorganisation of the circulatory system; but as the constant cough is distressing, it is a symptom which requires immediate treatment.

Let me, in this connection, remind you that cough is the natural effort to remove some irritation in the air-passages. This irritation may be caused by some foreign substance on the surface of the mucous membrane—such as mucus; our treatment is then directed towards making the expulsive efforts of the cough effectual, and expectorants are employed. On the other hand, the irritation may be due to a diseased condition of the mucous membrane itself—there is nothing to expel; here we try rather to lessen the sensitiveness, and, by checking the cough, give the congested or inflamed parts the rest which is essential to their return to the normal condition.

Note.—Two days after the above remarks were made at the patient's bedside, he had an attack of dyspnœa in the morning with cardiac pain. The pulse was irregular. At 10 a.m. he complained of feeling faint, but rallied somewhat after a dose of Æther mixture. At noon he was again very faint and complained of choking. Dyspnœa was urgent and cough ineffectual. The pulse was very weak. Half an hour later he suddenly collapsed and died quietly.

Extract from Notes of Autopsy by Dr. Smale, *Resident Medical Officer, N.L.C. Hospital.*

Right Lung: No adhesions; on removing lung white scar (size of four-shilling piece) noticed on parietal pleura, about one inch inside angle of third rib, partly over rib and second costal space. Pleura dissected off, structures of chest wall found to be normal under scar. Slight amount clear yellow serum in pleural cavity. Lung not collapsed, grey coloured with black mottling, lower lobe rather purple. At back of apex of upper lobe, corresponding to white scar on pleura, white puckered scar; lung around tinted

deeper grey, and felt more solid. On incising upper lobe through scar, small oval greyish-yellow mass found, size of a pigeon's egg, lying just under scar; lower part breaking down; mass surrounded by a firm, fibrous capsule, and outside this a red congested area. Small millet seed, greyish-white and white tubercles scattered sparsely through upper lobe, some becoming yellow and breaking down. Lung tissue around œdematous; some fluid in tubes. Middle lobe œdematous and congested. Lower lobe—centre deeply congested, purple colour, and carnified in centre; towards surface brighter red, under pleura greyish-white; all very œdematous.

Left Lung: Apex not adherent. Lower part of upper lobe and upper part lower lobe adherent by thin white fibrous adhesion bands, almost matting lung to chest wall. In separating lung from wall, space containing clear yellow serum opened. Base—no adhesions. Lung not much collapsed; both lobes very œdematous, and lower lobe somewhat congested but not much discoloration.

Both lungs emphysematous. No enlarged glands in mediastinum about roots of lung.

Heart: On opening pericardial sac, barely an ounce of straw-coloured serum escaped. Parietal pericardium studded with small white elevations—circular, flat, only slightly projecting—from pin's head up to threepenny piece, chiefly in upper pouches; base free. In front and at back, a few flat whitish patches, irregular in outlines with smooth surface.

Similar white smooth patches on surface of ventricles and auricles (patches roughly corresponded). Along lines of coronary arteries, whitish streaks. No adhesions.

Heart much enlarged: Much external fat. On opening heart, cavities contained dense whitish clot.

Right Auricle: Walls thickened, endocardium smooth, tricuspid valve normal size, flaps thickened at edges, chordæ slightly thickened.

Right Ventricle: Enlarged, walls normal, endocardium smooth, pulmonary valves normal, walls of pulmonary artery normal.

Left Auricle: Walls thickened, endocardium smooth, appendix long and twisted, mitral valve opening enlarged, admitted three fingers easily, valves much thickened and contracted, and chordæ thick and shortened, endocardium of valves smooth.

Left Ventricle: Cavity enlarged, walls hypertrophied, endo-

cardium smooth, except below aortic valves where roughened and thickened.

Aortic Valves: Right Anterior—edges smooth, perforated by circular opening destroying most of substance of valve, white clot adherent to edges of perforation.

Right Posterior—edges rough, calcareous, large calcified vegetation adherent to edge.

Third Cusp—(cut through in opening heart) slight vegetation.

Above valves, walls of aorta markedly atheromatous and calcified. Opening of coronary arteries calcified and stenosed. In arch of aorta several larger atheromatous ulcers. Calcification, &c. extended whole length of aorta, and into branches.

CHAPTER XIX.

COMMENTS ON THE PRECEDING CASE OF DISEASE OF THE AORTIC VALVE AND PULMONARY TUBERCULOSIS.

The patient, a labourer aged 46, was admitted into the hospital, where he had applied for relief for a cough. He had served fifteen years as a soldier in India, and had there suffered from malarial fever; since leaving the army, ten years ago, he had been employed as a labourer on the railway. His work had not been excessively heavy.

There was no history of rheumatism nor of syphilis. With the exception of malarial fever in India, he had had no illness, and enjoyed good health until the last five years. He had had a dry cough for the past two years. Three months ago he had an anginal attack whilst at work.

On admission there was much cough, with frothy expectoration. The temperature was raised in the evening. Bronchitic râles were abundant in the right lung, with signs of infiltration (congestion) at both bases. The cardiac dulness was increased towards the left; the apex-beat was felt directly below the left nipple. A friction rub was felt over the cardiac area towards the apex. At the apex a prolonged murmur replaced the first sound. At the base two murmurs (systolic and diastolic) were

heard, the systolic being distinguished by a difference in tone from that at the apex. There were no signs of aneurism. The brachial arteries were tortuous, firm, and visibly pulsating.

Four days after admission, dyspnœa and faintness, with sensation of choking, preceded collapse and death.

At the autopsy the heart was found to be enlarged. The mitral orifice was dilated, and the curtains of the valve were puckered, with beaded edges. There was no roughness of the endocardium.

Of the aortic valves, one had its margin roughened; a second had a large oval perforation; and from the free edge of the third a large calcified vegetation, about three-quarters of an inch in length, projected upwards into the aorta. The vessel itself was markedly atheromatous, and in some parts of the arch atheromatous ulcers were found.

In the lungs, besides the conditions recognised during life, there was, in the right apex, some undetected tubercle. A scar on the surface of the organ marked the seat of an ovoid caseous mass, about $1\frac{1}{4}$ inches in its longest diameter. This was surrounded by a well-marked fibrous capsule, and outside this was a zone of congestion. Scattered through the upper lobe of this lung were a few much smaller tubercular nodules, some already breaking down.

The spleen was enlarged in its long diameter to about 9 inches, but not thickened. Its lower end was especially firm and dark-coloured. The liver was cirrhotic, and the kidneys also fibroid and cystic.

The case is interesting from several points of view. It is curious to find such serious lesions in both heart and

lung, with so little inconvenience to the patient until a week or so before death. With the exception of an "attack of giddiness" three months ago, the man had apparently felt no marked inconvenience from his heart.

The causation of his cardiac disease offers a good field for conjecture. There are several circumstances incident to army service, both at home, and more especially abroad, which would favour endocarditis and atheroma.

The right lung presents an interesting example of tubercular disease, advancing to caseation, but then becoming quiescent through limitation of the mischief by fibrous overgrowth round the affected portion of the lung, and being lighted up again later, probably on the escape of some of the imprisoned bacilli.

The Tuberculosis of the lung was not diagnosed during life. The temperature chart gave rise to suspicion, but when I examined the chest, the bronchitic râles were sufficiently abundant to mask any auscultatory evidence of the active tubercular changes found after death.

The presence of the large caseous mass in the apex was not suspected. This mass is worthy of close inspection. The central caseous portion I believe to be much older than the smaller tubercular nodules observed in other parts of the same lung. When the mischief happened there is little or no evidence in the history to show; probably it was some time in the last five years. The fibrous growth around this shows an attempt at natural cure by "encapsulation." Then we have the recent congestion (inflammation) around. Is this a not yet completed part of the process by which the capsule is formed, or does it indicate the escape of bacilli through the fibrous wall?

That bacilli have escaped may be inferred from the recent nodules of still increasing tubercular disease scattered through the upper lobe of this lung. This exemplifies what is very liable to occur after apparent cure of Tuberculosis, viz. a re-infection from the quiescent tubercle. The co-existence of Tuberculosis of the lung with heart disease is of common occurrence.

CHAPTER XX.

CLINICAL OBSERVATIONS.

Anæmia and Phthisis.

Here is a case of pulmonary Phthisis with no very noteworthy features, either in the symptoms or course of the disease. Yet it is worthy of a few words of comment, because it is an example of a very common history.

The patient, a young woman in business, first came under my notice elsewhere as an out-patient. She complained of little that was definite, giving such an account of herself as we are accustomed to hear from the anæmic girls who attend in such numbers in any out-patient room. Like so many of these anæmic patients she had a slight cough, not prominently mentioned amongst her complaints. Examination of the chest revealed an unsuspected patch of tubercular deposit in the left apex.

The mischief extended instead of resolving, and I, therefore, had her admitted here. The chest now shows diminished resonance at each apex, only above the clavicle on the right side but extending much further down on the left. Over the whole upper lobe on the left side we hear dry crackling râles, and there are some creaking sounds in the right apex.

The mischief had involved the whole of the upper lobe

of the left lung, and the apex of the lower lobe; the apex of the right lung was also affected. She has improved considerably since her admission here, and the disease seems now to be quiescent in the lung, but there is some commencing mischief in the larynx.

I draw your attention to this case to emphasize the necessity of examining the chest whenever cough is complained of, especially in young adults. It is useless to attempt to treat a cough unless we know the cause of the symptom. It is easy enough in many cases to stop a cough by sedatives, but it is by no means always wise to do so. Purely symptomatic treatment is a confession of ignorance, or a makeshift of the man who is too hurried to examine. We may get some clue to the cause of the cough from the patient's description of the symptom, and from the character of the expectoration.

In a young person who brings up thick and yellow expectoration with the cough, never omit to examine the upper part of the chest. A complaint of the usual symptoms of anæmia by a young person whose pale face and lips confirm the history, might lead us to prescribe without examination; but anæmia does not exclude the possibility of Tuberculosis, it rather predisposes to this disease. The early detection of tubercular mischief in anæmic subjects is of vital importance, for in my experience the disease in such persons tends to advance rapidly.

Another case, which I saw in consultation some few years ago, illustrates the dangers of incomplete examination, and impressed itself strongly on my memory. A girl was pale and out of sorts, and superficial examina-

tion showed the signs of anæmia, for which treatment was adopted. Later on, at school, she became languid and complained of pains in the chest, but her condition was attributed to some hysterical tendency. Partly, perhaps, owing to the objection to complete examination of a girl at school, and partly with a view to avoid making a fuss over what, if due to hysteria, might have been intensified by making much of the girl's complaints, again no thorough examination was made. At last, during a holiday, when her lassitude caused some anxiety, she was examined and found to be suffering from pulmonary Tuberculosis which had already advanced too far for successful treatment, and a few months later she died.

We have, in the male corridor, another patient to whom I would draw your attention also, in connection with the ætiology of Phthisis. He is a footman, 19 years of age. For five years he has been in service in the same family, and for this period he has lived, as male servants are so often obliged to do, in the basement of the house. He describes his bedroom as being close and stuffy, and without a window. I have elsewhere (*a*) commented upon the danger of inattention to hygienic requirements in servants' sleeping rooms—a fault which exists even in some of the best town houses. I have had several cases of Phthisis in men-servants where the disease has been traceable to the predisposing influence of living in unsuitable rooms. In this patient there is slight deposit in both apices, the left side being the more marked. There is every reason to

(*a*) *The Hygienic Prevention of Consumption.* Chas. Griffin & Co., London.

hope that the patient will improve considerably under treatment.

Infection of One Lung from the other.

These two women will serve to illustrate an interesting point. The early detection of tubercular mischief in the lung is of such importance—in view of the greater success of early treatment—that it is well to know where we may expect the first signs of Tuberculosis to show themselves.

As is well known, the apex of the lung is the most frequent seat of tubercular disease when this first attacks the chest.

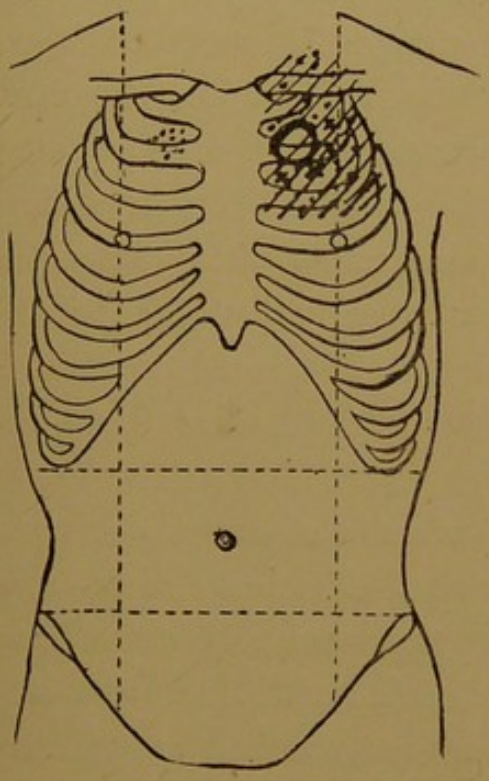
In this first case, a woman aged twenty-five years, who has had a cough more or less always, had a slight attack of hæmoptysis about twelve months ago. A casual observer might pronounce her lungs to be sound. You, however, will hear some crepitations—such as we have become accustomed to associate with tubercular mischief—in the right supra-spinous fossa. This is just the position in which we so frequently find the first signs of tubercular disease in the lung.

But it is a curious and interesting fact, which I have long noted and frequently confirmed, and which I have never seen mentioned by others, that when, with active mischief in one lung, the other lung becomes infected, the first signs of this accident will not be found in the extreme apex of the secondarily affected lung, but in situations which I will now indicate.

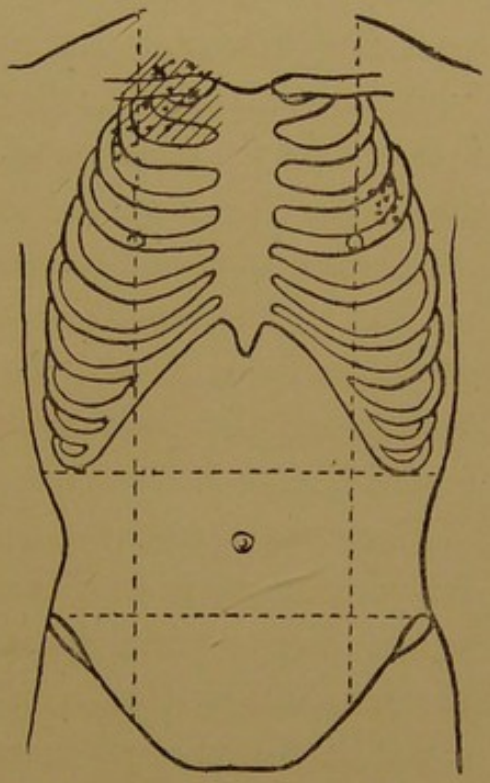
When the primary mischief is in the left lung, and the right lung becomes infected (probably from the sputa

passing into the bronchus), you will find the first indications of this by examining over the second right costal cartilage near the sternum.

If, however, the left lung becomes infected from active Tuberculosis of the right, the position in which you may expect to find the first signs of this auto-infection is in the anterior axillary line just above the level of the nipple—about the third intercostal space.



Infection of right lung from the left. L. N., æt. 27.



Infection of left lung from the right. S. L., æt. 23.

This second patient, L. N., illustrates the correctness of my "position of selection" in infection of the right lung from the left.

A draper's assistant, twenty-seven years of age, has been suffering for two years with Tuberculosis of the left

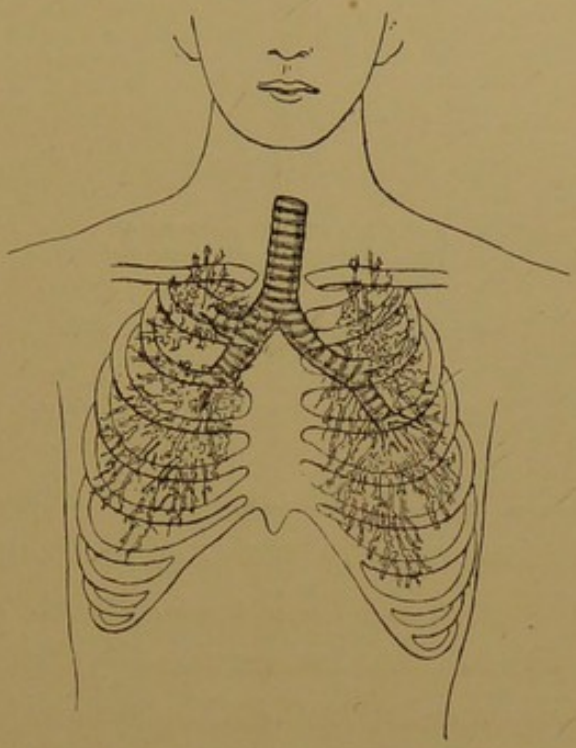
lung. On admission six weeks ago she had extensive disease involving almost the whole left upper lobe. There was acute breaking down of the lung, and already clear signs of cavity.

The right lung gave no evidence whatever of tubercle. For a week after her admission the temperature rose every evening to about 100° F., and then for a time it never rose above 99° F. Ten days ago the temperature again rose to 100° and 101° at night, and on examining over the second right costal cartilage—my "selective point"—we heard crepitations indicating tubercular infection. Counter-irritation by iodine on the chest was at once ordered, and after five days of evening fever the temperature steadied down, and now again varies little from the normal. On examining now you will not hear any crepitations, but a single "click" at the end of inspiration marks the position where the tubercular focus was established.

Here we were prepared, when we saw the rise in temperature, to find a new centre of infection, and knowing where to expect it if it was in the previously sound lung the mischief was at once detected, and I think we may say that the prompt treatment has checked any further advance in this lung, at least for the present.

The explanation of the facts above noted is to be found in the arrangement of the bronchus as it enters the lung, and the position of the first subdivision into the larger branches of the bronchial tubes. Casts of the bronchial tubes have been made by several observers, all of which agree very closely in shape and appearance, and may be taken to represent accurately the branchings of the air-

tubes. The accompanying figure shows how these branchings differ on the two sides.



MAIN BRANCHES OF THE BRONCHIAL TUBES—modified from a cast by Birch-Hirschfeld—shown in their position within the chest.

It will be seen that on the right the bronchus passes almost horizontally into the lung, and after a short course divides into a number of branches. The position of this subdivision is shown by the figure to correspond with the situation I have indicated for the first signs of infection of the right lung by sputa passing down the bronchus. The sputum would be checked where the tubes suddenly become smaller and the straight direction of the bronchus stops. On the left side the bronchus passes less horizontally and reaches further into the lung before it breaks up into its branches, thus bringing the place of subdivision more under the anterior margin of the axilla.

CHAPTER XXI.

CLINICAL OBSERVATIONS.

The Temperature in Phthisis.

The temperature chart in a case of Consumption gives most valuable assistance in gauging the condition, and in estimating the progress of the complaint. Even in the early stage of the disease, when the physical signs are few and but slightly marked, and before tubercle bacilli can be found, the temperature chart will show a deviation from the healthy standard. There may be a slight rise to about 99° F. in the evening, itself a sign of divergence from health when the rise occurs day after day. With this rise in the later part of the day the morning temperature is subnormal, and thus the diurnal range is increased. (Chart IV.) The time of day at which the rise and the low temperature respectively occur may not always be the same, but a daily rise above the normal with subsequent reaction, indicates some active morbid change which has weakened the patient. We may note that any physical activity of the patient increases the febrile condition, and perhaps also leads to a greater reaction, so widening the diurnal range. Conversely we find that rest in bed diminishes the range of temperature. It is common to

Chart IV.

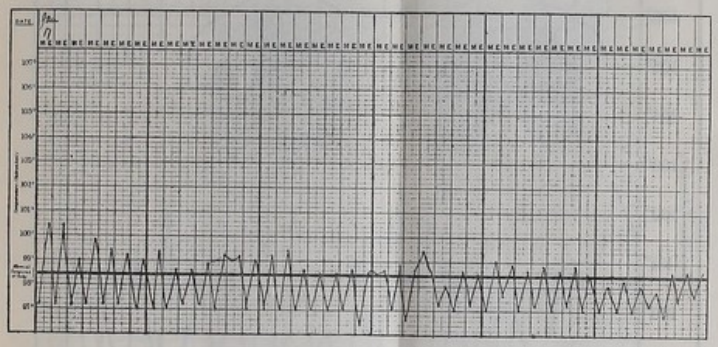
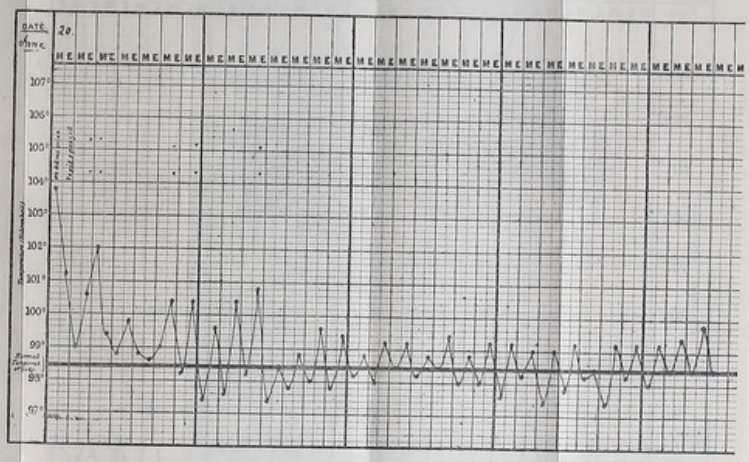


Chart V.



Charts IV., V. to face page 242.

CHART VI.

Name: William B. *Age:* 23. *Disease:* Phthisis pulmonalis. *Admitted:* May 8th, 1888.

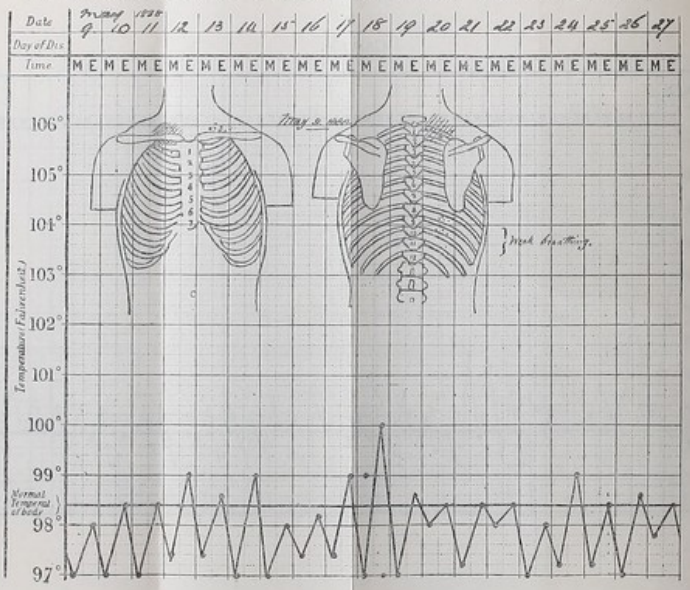
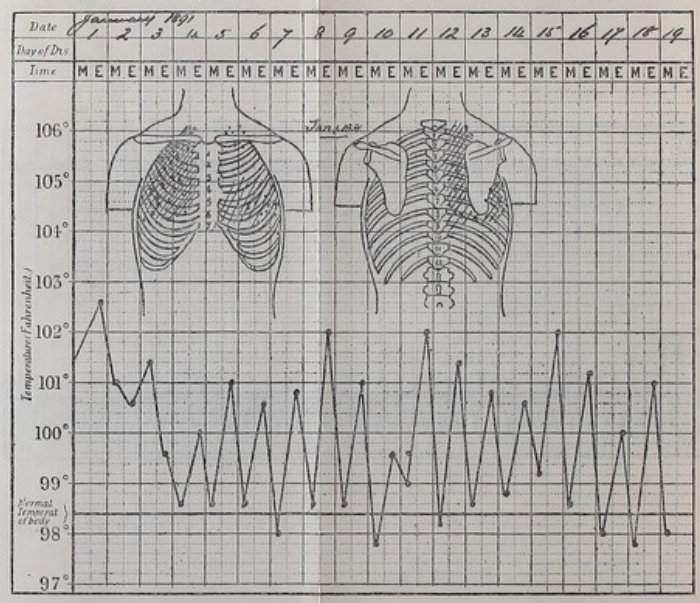


CHART VII.

Name: William B. *Age:* 26. *Disease:* Phthisis pulmonalis. *Admitted:* Jan. 1st, 1891.



These Charts, VI., VII., NOT to be cut, but folded and fixed to face page 243.

find the temperature of patients higher on admission to hospital than at any subsequent period of their stay. (Chart V.) In some cases, even when the physical signs and the presence of tubercle bacilli in the sputum shows the disease to be active, we may get a temperature chart continually below the normal line. This is perhaps most frequently found in chronic cases. As the disease advances the temperature chart will alter its character; not only does the temperature tend to rise higher, but the daily range becomes much increased. This is well shown in the two charts (VI., VII.) from the same patient taken with an interval of three years, during which time the disease had advanced considerably. When cavities have been formed there will probably be a mixed infection, and the temperature chart resembles that of septicœmia, with a daily range of three or four degrees and even more than this. This cannot be considered a chart of Tuberculosis, but it is the typical chart of advanced and active Consumption. Some years ago I endeavoured to obtain a curve which should represent the typical daily course of the temperature in such an advanced and active case of Consumption. I found a suitable case in a girl of twelve, who was admitted in an apparently hopeless condition, and was, three months later, discharged fat and healthy-looking, and with hardly any active mischief in the lungs. Her temperature on admission showed in a marked degree the large diurnal range which accompanies active Tuberculosis with mixed infection, ranging from 105° to 96° in the twenty-four hours. For a week or two before she left, the daily range was only between 98° and 100°, only occasionally touching

this latter, and she was walking about and feeling well. For nearly three weeks, when the temperature fluctuated largely, I had it taken every hour both day and night, so that we might take steps to keep it down when it got too high, and give necessary stimulants when it fell below normal, for the depression then became extreme.

Chart VIII. shows the hourly temperature for six days in this case; the next figure shows the curve obtained by taking the average of each hour's readings for eighteen days, and may be taken as the temperature curve of acute Consumption with mixed infection.

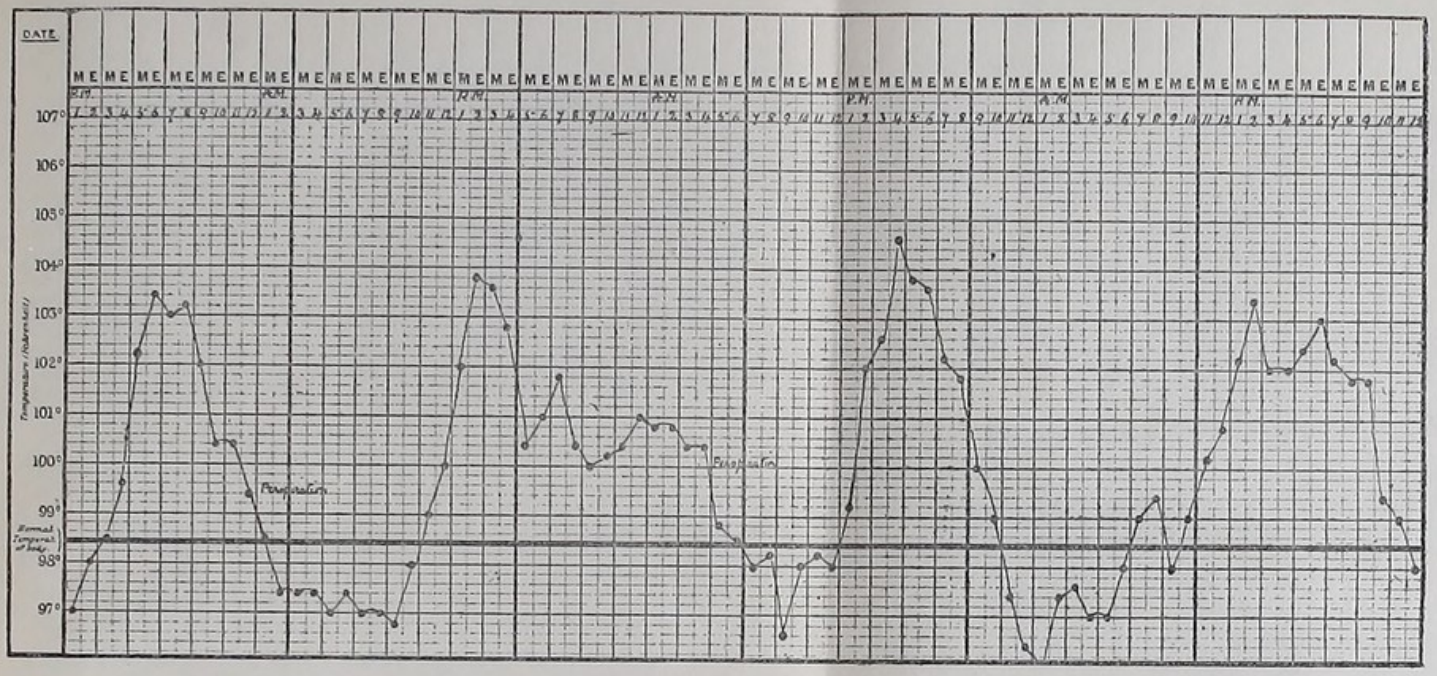
It is interesting to find that, although the temperature is so irregular and that the highest and lowest points occur at different times in the day, the average of a large number of observations shows a regular curve. (Chart IX.)

It may be noted that there was as much as 4·2° F. variation within one hour, and a range of 8·6° F. within twenty-four hours.

Such a chart, however, does not show the temperature variations which are caused by Tuberculosis alone. These wide variations indicate a mixed infection and practically represent septic poisoning. The temperature in uncomplicated Tuberculosis rarely rises as high as is shown in this chart, and although the daily range is greater than in health, it does not usually exceed two degrees Fahrenheit.

In the cases of mixed infection the varying temperature is the cause of much distress and discomfort to the patient, and various suggestions for relief have been put forward.

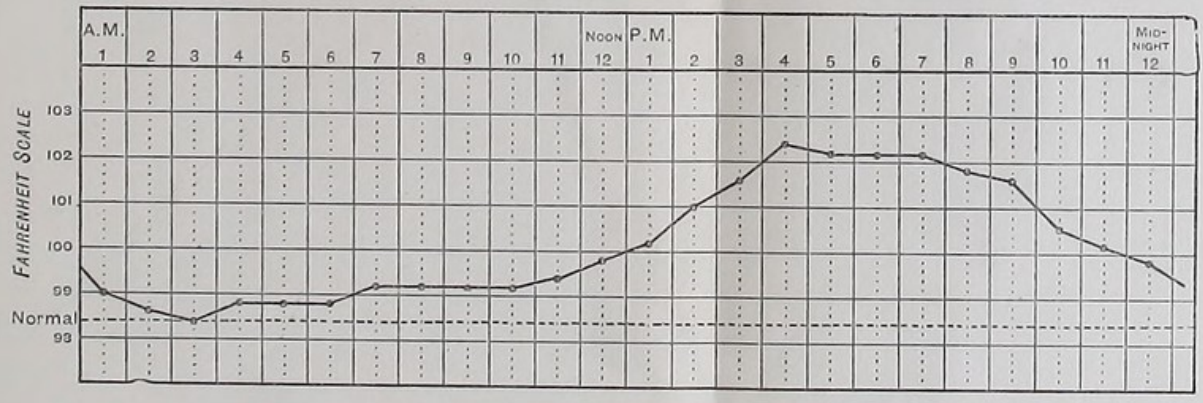
Chart VIII.



A portion of the Chart from which the temperature curve (Chart IX.) was obtained.

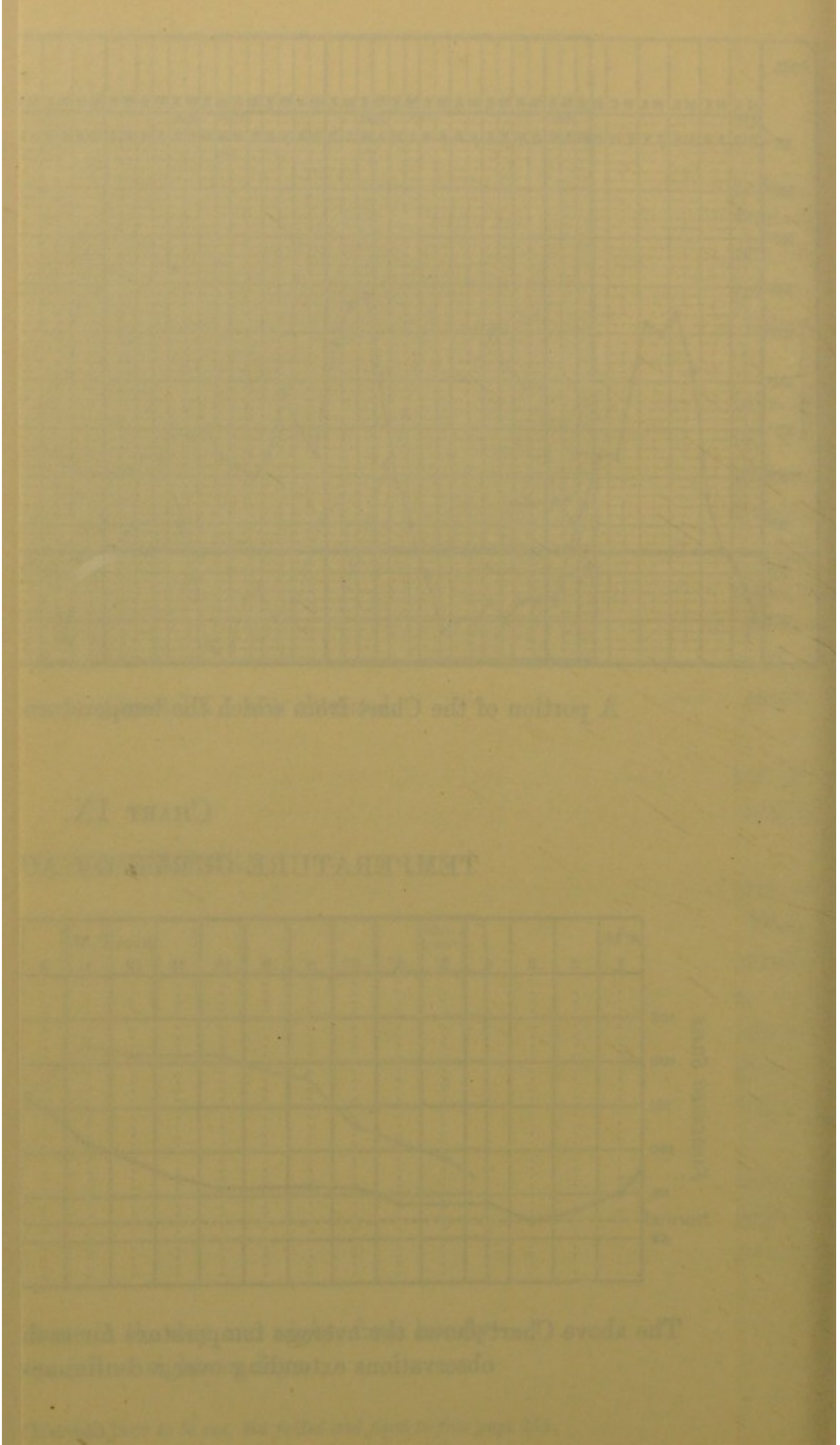
Chart IX.

TEMPERATURE CURVE OF ACUTE PHTHISIS.



The above Chart shows the average temperature for each hour in the day and night, from hourly observations extending over a continuous period of eighteen days.

[*Charts VIII., IX.*, NOT *to be cut, but folded and fixed to face page* 244.



The chief aim has been to reduce the temperature when it rises above a certain point—say 102° F.—and different antipyretics have been employed for this purpose. I believe, however, that the majority of the antipyretic drugs are rather dangerous than useful in such cases. I found, in making observations on this point some years ago, that, though it was possible to delay the rise by giving the antipyretic when the temperature was rising, this merely postponed but did not prevent the high temperature, which occurred later. I have found that sponging with tepid water, with perhaps a little vinegar added, gives relief when the temperature is high, and is much more satisfactory than any drug.

The patient just referred to appeared on admission to be too ill to justify any hope of advantage from a stay in hospital, and one felt disposed almost to resent the occupying of a bed by an apparently hopeless case. But I have had so many pleasant surprises with apparently hopeless phthisical cases that I am unwilling to acknowledge that any case is too bad to admit of the hope of considerable benefit.

You will probably wish to know what was the treatment which brought about such a satisfactory result. The treatment card will help but little to show this, for the rest, fresh air, suitable food and careful nursing are as important as any medicinal treatment. The first prescription is a tonic which I frequently use, which aids appetite and digestion: malt is a food. The antiseptic inhalation I employ to prevent auto-infection rather than with any curative idea.

Acid. Nitrohydrochlorici dil. . .	15 minims.
Sp. Chloroformi	5 ,,
Tr. Gentianæ Co.	40 ,,
Aquam	add to 1 ounce.

T. d. s.

Essentia Malti	1½ ounces.

Cum cibo ter in die.

Inhal. Acidi Carbolici cum Ol. Pini Sylvestris.

Acidi Carbolici	20 minims.
Ol. Pini Sylv.	add to 1 ounce.

Ten drops poured on the sponge of a respirator to be worn, for an hour at a time, three times a day.

I have frequently noticed that when Tuberculosis of the lungs affects children before puberty—which may be delayed if the child is ill at the time when this might be expected—the prognosis is much more favourable than in those who become affected after puberty is reached. The girl referred to above was undeveloped, and a very similar case in an undeveloped girl of seventeen in my wards when I was House Physician at University College Hospital, who improved to an unexpected extent and was discharged to a convalescent home, illustrates the same fact when puberty is delayed by the illness.

Quiescent Phthisis.

In this man, who is shortly leaving the hospital, and whose condition has greatly improved since his admission, we see how the chest falls in when a much damaged lung

underneath contracts as the disease becomes inactive. He is twenty-two years of age, and has had a cough since an attack of pleuritis two years ago. On admission his weight was only 7 st. 4 lb., and he had occasional pyrexia reaching 103° F. There was deficient resonance with fine crackling and tubular breathing over the right apex, and on the left side deficient respiratory movement, dulness almost to the base, with crackling and bubbling râles heard all over that lung.

There was thus very extensive mischief with acute softening in the left lung, which had been previously damaged by the pleurisy.

Now, after about six weeks in the hospital, his weight is 7 st. 10 lb.; his temperature normal or subnormal; he has only a slight cough, and scanty expectoration.

The right side gives much the same signs as on admission.

The left side of the chest is, as you see, much fallen in; there is cavernous breathing above the scapula and at the base, with whispering pectoriloquy. A slight crackling râle at the apex is the only adventitious sound to be heard. The apex beat of the heart is now $1\frac{1}{2}$ inches outside the nipple line in the fifth space, and there is visible pulsation all over the cardiac area.

On measurement of the chest as high as we can get the tape in the axilla, we see there is a full inch difference between the two sides (right $15\frac{1}{2}$ in.; left $14\frac{1}{2}$ in.) Here the softened tubercle has been got rid of, leaving cavities in the lung. These are diminishing in size by contraction of their walls, and the heart has become uncovered by the retracted lung, whilst the chest wall sinks in. The sequence

of events in this case must be regarded as favourable. The mischief gradually dries up, and the signs of softening disappear; cavities are formed, which gradually contract. There is now little or no active mischief. Unfortunately, we cannot call the disease cured; it is only quiescent. It may at any time light up again and extend. But extension would then be less rapid than at first, for when the activity of the tubercular process has been reduced, even if its advance be not absolutely checked, the lessened activity leads to fibroid changes instead of the destructive change (softening) of acute tuberculization, and the fibroid tissue acts as a barrier to rapid extension of the disease.

There is much of interest to be noted in the shape of the chest in lung diseases.

Compare these two cases.

In the one (tubercular Phthisis) we see a flattened chest, thinly covered. The superior margin of the trapezius muscle stands up as a sharp edge behind the supra-clavicular fossa. The movements of respiration are slight, and chiefly carried out by the lower part of the thoracic walls, and by the diaphragm; the lower part of the lungs has to do extra work.

The other case presents a rounded thorax, also thinly covered with wasted muscles, but the upper part of the body looks heavy, instead of slight and wasted as in the first case. The margin of the trapezius seems to be curved forwards, as though overhanging the supra-clavicular hollow; the shoulders are rounded behind. Here also the movements of respiration are slight, but the whole thorax seems to share equally in the diminished

movement. This is a case of fibroid Phthisis; the expansion of the lung is deficient throughout, not only at the apex. The auxiliary muscles of respiration acting on the upper part of the thoracic wall are constantly at work, and having developed by use, give the heavy appearance to the upper part of the body. There is a man in the ward with spasmodic asthma, and in him you will see still more marked the heavy shoulders and rounded back, which result from constant effort to draw air into the lungs.

"Vicarious" Hæmorrhages from the Lungs.

I have seen several cases in which hæmorrhage from the lungs in women occurred with a periodicity closely corresponding with the menstrual period. In some I have not been able to detect any tubercular mischief in the lung; in others there has been unmistakable Phthisis. The following examples serve to illustrate this hæmorrhage in tubercular patients:—

(1) E. K., æt. 28, parlourmaid, single, was a patient in the hospital in 1883. My notes show a history of Amenorrhœa for two months before admission, and an attack of hæmoptysis on one occasion a month previous to admission, when she was suffering from a second attack. This second hæmorrhage commenced on July 19th, and recurred on the 21st and 22nd, the amount of blood being about two ounces each time. This is about the time when she should be unwell. The blood comes up with very little cough, and is frothy from admixture with air. Physical examination of the chest showed dulness on percussion at the left apex, with moist clicking in the left supra-spinous fossa. There was no further hæmorrhage until August 14th, when about a teacupful of blood was coughed up. There was some more bright blood on the 17th and again on the 19th.

(2) Mrs. D., æt. about 30, seen in consultation, June, 1894, with active and extensive tubercular mischief in both lungs. The history as regards hæmoptysis was as follows:—

1st Attack...April 30th, 1893. About half a teacupful of blood. About 25 days before the birth of her child.

2nd ,, ...July 20th, 1893. Slight bleeding at 9 a.m.; more copious at 2 p.m. and again at 5 a.m. the next morning. Menstruation July 22nd.

3rd ,, ...Dec. 28th, 1893. Menstruation Dec. 31st.

4th ,, ...Jan. 28th, 1894. Menstruation Feb. 1st.

5th ,, ...Feb. 25th, 1894 (very trifling). Menstruation March 1st.

6th ,, ...May 2nd, 1894. This attack followed menstruation, which had commenced on April 26th, and had ceased.

7th ,, ...May 14th, 1894. Not serious.

8th ,, ...May 20th, 1894. Rather severe. Menstruation May 24th.

In this case the hæmoptysis occurred with menstruation: in the first case it occurred during a period of amenorrhœa, at about the time when menstruation was to be expected.

CHAPTER XXII.

A CARDIO-PULMONARY MURMUR; OR A RESPIRATORY MURMUR RHYTHMICAL WITH THE HEART BEATS.

In March, 1895, I had the good fortune to be in the chair when Dr. William Ewart read an interesting paper, on the Dorsal Auscultation of Heart Sounds and Murmurs, before the Harveian Society. In opening the discussion I took occasion to make the following remark (*a*) :—

> In reference to murmurs heard in the back, there was a murmur, systolic in point of time, which was well heard just below the angle of the scapula and sometimes in other situations, and which was produced in the air tubes, and therefore not strictly cardiac, though caused by the beating of the heart. It was a "whiffing" sound, and was most plainly heard during inspiration or only during this act. It was well to bear this murmur in mind, as it might be mistaken for a true cardiac murmur.

I subsequently collected a good number of cases illustrating this murmur, but unfortunately sixteen months ago an attack of scarlet fever caught me with the notebook containing the references to these cases in my pocket, and disinfection destroyed the germs of an article on the subject as well as any microbes. I have, however, been able

(*a*) *British Medical Journal*, March 23rd, 1895, p. 645.

to find notes of some of the cases, and have added others within the past few months.

In examination of the chest it is noticeable how, in certain conditions of the lungs, the heart sounds may be heard in parts far removed from the cardiac area. Consolidation, from whatever cause, conducts sound from the heart or from large vessels to the surface of the chest, so that such sounds are heard in unexpected places, and when so heard furnish valuable information as to the interposed organ. Thus we may have the heart sounds conducted towards the right apex in tubercular disease of the lung of that side, and we may hear the heart sounds clearly in any part of the chest wall over the position of a good-sized lung cavity. When, therefore, we hear sounds corresponding in rhythm to the beat of the heart, at some distance from that organ, or from any large vessel, we are at once led to suspect that there is some consolidation of the intervening lung.

There is, however, as I have already indicated, a source of fallacy, which, though frequently present, has received little notice from writers and is, perhaps, less recognised than it should be. It, however, deserves wider recognition; for the physical sign of which I speak may, if misinterpreted, mislead not only as to the condition of the lungs, but also as to the condition of the heart. This physical sign is, in fact, a *bruit*, rhythmical with the heart beat, but produced in the lung—a respiratory sound which may easily be mistaken for a conducted cardiac sound. Heard, as it may be, at some distance from the heart or great vessels, it might convey an erroneous impression of the state of the lung; added to this, the character of the

sound would certainly suggest some lesion of a valve of the heart if the sound were supposed to originate in this organ. The subject is, therefore, of importance, the more so that, as I have already mentioned, it is not of rare occurrence. I propose in this article to instance some cases in illustration of the physical sign under consideration, and to discuss the causation of the sign and its significance.

The sign is a blowing *bruit* or whiff, generally high pitched, but not always so, synchronous with the heart beat, and therefore rhythmical; sometimes heard throughout both inspiration and expiration, but more frequently only audible during some portion of the respiratory cycle. The *bruit*, though simulating a blowing cardiac or arterial murmur, is, as I shall show, caused by the movement of air in the lungs and air tubes, though its rhythm is communicated by the movements of the heart, and perhaps sometimes by the distension of a large artery.

CASES.

Case I.—R. W. R., male, aged 34, first seen in May 1892. Tubercular deposit right apex. In February, 1893, I noted "a high-pitched blowing murmur synchronous with the heart beat (systolic) heard over the right side below the nipple level and into the right axilla." There was also a loud systolic murmur over the aortic cartilage during inspiration, absent during expiration. A month later the first murmur was "nearly gone," but still audible. The lung condition became quiescent, and the patient was in fairly good health when I saw him last year.

Case II.—Mrs. G., 25, consulted me in 1894, with tubercular consolidation at the right apex. On the left, at the base behind, there was a soft systolic murmur during inspiration only; not heard over the cardiac area. Six months later the Tuberculosis

had become quiescent. There was a loud *bruit de diable* and arterial murmur in the neck, and a "systolic inspiratory murmur just inside the tip of the left scapula." Heart sounds clear.

Case III.—L. P., female, 16. Left pleurisy Christmas, 1894. In November, 1895, she had cough with harsh breathing at the left apex. A loud systolic *bruit* was heard immediately below the tip of the left scapula, always present, but louder on inspiration. No murmur heard over the cardiac area. A fortnight later fine crepitations were noticed at the left apex, and the *bruit* at the base had disappeared.

Case IV.—C. L., female, 19. Tubercular deposit right apex. Systolic "whiff" heard just below tip of left scapula, and in third space in front on right side. *Bruit de diable* in neck.

Case V.—Mr. S., aged 19, clerk. Seen in 1895 with rather over-acting heart, faint systolic murmur over pulmonary cartilage (second left), but no murmur over cardiac area. Apex beat just below nipple. Impulse forcible. In left axilla and over left back up to about level of fourth dorsal spine, a systolic blowing murmur is heard only during inspiration (and chiefly during the latter part of this). It is a pulmonary (breath) sound and not a vascular sound.

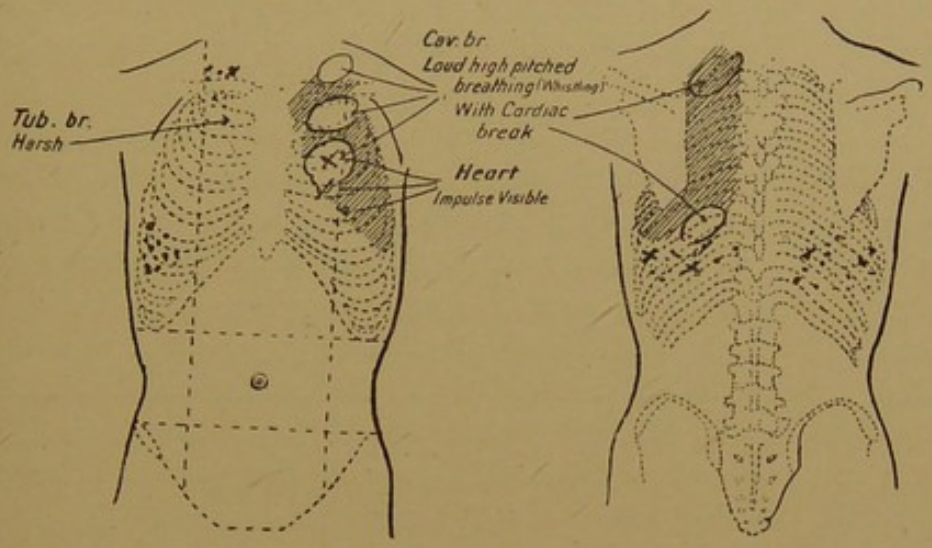
Case VI.—Mr. W., aged 21, seen March, 1895. Recovering from left basic pneumonia. A systolic murmur heard over left base behind, on inspiration, not during expiration.

Case VII.—Miss McK., aged 20. Anæmic; has had pleurisy twice at left base. Faint systolic murmur heard over left base, especially during inspiration. Hæmic *bruit* over cardiac area.

Case VIII.—G. S., male, aged 25. Cold and cough (catarrh) four days. Much blackish phlegm. Later, systolic inspiratory whiff in left axilla.

Case IX.—L. N., female, aged 27. Was in hospital under my care in 1895 with extensive tubercular mischief of the left upper lobe. In October, 1897, she came to see me. There was much cavitation of the left lung, and extension of the disease to the right side. Whilst talking to the patient one notices a peculiar sound produced with the breathing: a blowing or puffing noise is produced during inspiration and at the beginning of expiration, rhythmic with the heart beat. This can be heard at some distance

(about six feet) from the patient. With the stethoscope this sound is heard with greatest intensity under the left clavicle anteriorly, and just below the angle of the left scapula posteriorly. It is a dry, metallic puff, taking place at the same time as the first sound of the heart. The accompanying diagram will show that there



are signs of cavities at the points where the systolic whiff is heard loudest (*b*).

Case x.—M. C., female, aged 20. Thin. History of Phthisis for eighteen months. Cavity left apex with deposit to base of upper lobe. Right side free. Inspiratory whiff heard below the right clavicle.

Case xi.—C. H. J., male, aged 20. Phthisis nine months. Acute. Cavity in right apex with active softening; deposit to fourth rib in front and sixth spine behind. Harsh, jerky respiration in left front to third space. Along the anterior axillary border on the left side a respiratory murmur (systolic) is heard during inspiration; absent during expiration. Breath sounds at this spot good; no whispering pectoriloquy. Heart sounds quite clear. A week later systolic whiff in second space right above and just within the nipple, chiefly inspiratory. Also similar sound,

(*b*) A case resembling this very closely is published in the *Lyon Médical* for September, 1897 (t. lxxxvi), p. 39, by Dr. L. Bonnet under the title "Bruits Pulmonaires Rythmés par le Cœur."

deeper in tone below angle of left scapula, chiefly expiratory. Whiff noted above still audible. Fourteen days later inspiratory systolic whiff to be heard all over the left front, very well marked just outside the apex of the heart. Chest is thin, flat, and poorly covered. A week later the two whiffs noted above still plainly audible; that on the right seems now chiefly expiratory, and that on the left chiefly inspiratory and postsystolic. No murmur over heart or aorta.

Case xii.—F. H., aged 31, male. Admitted December 31st, 1897. Phthisis—active; deposit scattered throughout both lungs. Cavities in both apices; larynx affected. On right side a systolic blowing *bruit* is heard in second, third, and fourth spaces from border of sternum to mid-axilla—an area as big as the hand. It is heard through both inspiration and expiration, sometimes missing one beat at end of expiration. Not conducted along aorta; aortic sounds clear. Breath sounds healthy, with few adventitious sounds over above area. The whiff goes on when patient is holding his breath, but then with to and fro sound—double. The respiratory systolic sound still heard as above three weeks later.

Case xiii.—H. S., male, aged 34. Fibroid Phthisis—chronic. Heart sounds normal. Inspiratory and expiratory systolic whiff left front second space in the nipple line, also just inside the right scapula, here only inspiratory.

Case xiv.—S. S., female, aged 36. Thin, poorly covered chest; tubercular deposit both apices, especially left. Chronic condition. Laryngeal papilloma in interarytenoid region. January 27th. Systolic *bruit*, inspiratory and expiratory, heard to-day down left back, from the middle of the interscapular region to just below the angle of the scapula; from there to the base, only the inspiratory part of the *bruit* is heard. March 3rd. Whiffs still heard in left back; well marked opposite eighth and ninth spines, about tip of scapula, where it is only inspiratory. Heart sounds clear.

Case xv.—E. H. P., male, aged 47. Thin, poorly-covered chest. Fibroid (chronic) Phthisis, ulceration of epiglottis. January 20th. Systolic respiratory whiff left front at level of nipple (fourth rib) during whole of expiration only. Heart sounds at apex quite clear. March 3rd. The respiratory whiff is now heard very distinctly over the whole left front (except cardiac area), and most marked towards the left shoulder. Also heard over first two spaces on

right front. On the front (both sides) the whiff is well marked during the whole of expiration and doubtful during inspiration. At left base behind—below tip of scapula—a similar whiff is heard only during inspiration. March 7th.—Whiff heard when he holds his breath, either after inspiration or after expiration. Much louder in latter. With the expiratory sound there is a distinct systolic *bruit* (whiff). With the inspiration a sort of jerky respiratory sound, the interruptions being rhythmical with the heart beat, but greatest intensity postsystolic.

Case xvi.—S. B., male, aged 21. Three months' history, dating from influenza in January, 1898, followed by pleurisy—fluid to the sixth rib behind. March 17th, 1898, left side clear. Left front, all over, inspiratory sound is loud and harsh and interrupted in synchronism with the heart beats. Expiratory sound much softer, with distinct systolic whiff. Heart sounds clear. No murmurs over either aorta or pulmonary artery.

Case xvii.—A. A. W., male, aged 41. Tubercular deposit, left, to fourth rib, January 6th, 1898. Systolic high-pitched whiff only heard during inspiration. Heard in the third space just inside the nipple line, left side. March 3rd. Still whiff in third left space as before. Below inner end of left clavicle there is cavernous breathing, with whispering pectoriloquy, and here the inspiratory sound is interrupted in synchronism with the heart beat.

Case xviii.—L. S., female, aged 20, shop assistant. March 23rd, 1898. Palpitation and anæmia. Nothing pulmonary. Soft systolic murmurs heard at cardiac apex and at the pulmonary cartilage; *bruit de diable* in the neck. A systolic (respiratory) whiff is heard just below the tip of the left scapula, easily distinguished from the cardiac murmur, being rougher and of lower pitch.

Case xix.—F. P., male, aged 18 (admitted March 17th, 1898), clerk. Ill seven months. Good deal of wasting. Tubercular deposit in left lung (both lobes). Slight consolidation right apex. In first and second spaces the breath sounds give a "rhythmical jerky inspiration," not synchronous with heart beats; but with expiration the interruptions are with the beat, and constitute an expiratory systolic whiff.

Case xx.—A. P., female, aged 32. Anæmia and debility; no Phthisis. Just above the cardiac area there is a systolic expiratory

whiff. Heart sounds clear; beat forcible. Five minutes later, when the action of the heart quieted down, the respiratory whiff was no longer heard.

Case xxi.—J. M., male, aged 47. Fibroid lung with superadded Tuberculosis. Very marked systolic respiratory murmur above cardiac area, throughout the whole respiratory cycle, simulating a cardiac murmur. Heart sounds clear.

Case xxii.—Mr. R. G., aged 49. Has been twenty-seven years in India (Civil Service). Slight fibrosis of lung, but apparently no tubercle. Systolic inspiratory whiff heard below the tip of the right scapula, where there are some dry crackling *râles* to be heard also. Over-acting heart; no murmurs.

Case xxiii.—Occasionally when lying on my left side I can hear a high-pitched whiff, rhythmical with the heart beats, which seems to come from the trachea. It occurs only towards the end of inspiration and seems to be rather after the heart beat (postsystolic) It continues when I hold my breath and is then heard with each beat of the heart. It is not heard when I am lying on my right side. Richardson's theory, referred to later on, of a piece of lung being pressed between the heart and the chest-wall might explain the effect of position on this whiff.

The cases noted above show auscultatory phenomena produced in the lung, and modified by the adjacent circulatory organs. We have other evidence that the sounds produced in one organ in the chest may be modified by the action of other organs in that cavity. As an example of the modifying action of the respiratory system on vascular sounds the following case may be given:—

Case xxiv.—M. T., female, aged 22. Anæmia with enlargement of the thyroid, especially of the right lobe. In the root of the neck on the right side there is a continuous, high-pitched, musical murmur (venous hum), which is much louder during inspiration. Here the negative pressure in the thorax during inspiration causes an increased flow through the vein, with consequent intensification of the murmur.

Of the twenty-three cases illustrating the cardio-pulmonary murmur, thirteen are males and ten females. In fourteen of the cases (nine males and five females) there was Tuberculosis of the lung; in nine (five males and four females), there was no tubercle detected. The murmur was heard only during inspiration in ten cases; only during expiration in four cases, and during both inspiration and expiration in seven cases. In two cases the part of the respiratory cycle is not noted. It will be seen, then, that in this series of cases the murmur is most frequently inspiratory. It is not permanent, sometimes being present at one examination and not to be heard at another.

We may note that in Case II the *bruit* was distinctly audible over the position of the heart's apex, and was thus very liable to be taken for a cardiac murmur. In Case XIV the sound was heard down the left spinal groove along the course of the aorta. In both cases it was easily demonstrated by careful auscultation that there was no murmur in the heart or in the aorta. It will be observed that the lung condition varied greatly in the different cases. The *bruit* may be heard over a cavity and over apparently healthy lung tissue. I have heard it in cases where there was no suggestion and no evidence of lung mischief; and, given a certain laxity of tissues, a general debility, I believe this *bruit* may be frequently found without disease of either lungs or heart. That most of the instances here recorded are in patients with lung disease is probably only due to the field of observation which my own practice chiefly affords.

As to the causation of the *bruit* under consideration, it

is clearly caused by the passage of air through the air tubes. It is only the ordinary respiratory murmur momentarily modified in intensity at the systole of the heart. Jerky respiratory sounds (cog-wheel breath sounds) are often heard in morbid states of the lung or as a result of nervousness; in the latter case the jerkiness is due to irregularity or want of smoothness in the action of the respiratory muscles.

The sound which I am now discussing is a form of jerky respiration, limited to a portion only of the lung, and due to a local cause. Its importance lies in the fact that the jerks, or inequality in the intensity of the respiratory murmur, occur at intervals corresponding to the beats of the heart—a rhythm which may cause the whiff, or breath sound at its point of maximum intensity, to be mistaken for a cardiac murmur.

The simplest explanation of the influence of the heart beat on the respiratory murmur is that the heart kicks against a portion of the lung and forces some air out of that portion. This would explain some of the *bruits* heard during expiration. The sudden pressure, which during expiration would momentarily intensify the breath sound and so produce this systolic whiff, would act somewhat differently during inspiration. Alternating differences of pressure on a part of the lung in contact with the heart might produce jerkiness of the respiratory murmur in both inspiration and expiration, but in inspiration the greater intensity of the breath sound should coincide with the period of least pressure, and be cardio-diastolic in time. I have not been able to satisfy myself that this is so, though in some cases the sound seemed to be postsystolic.

Parts of the lung distant from the heart might be influenced by the movements of the heart if consolidated lung intervened; and pleural adhesions might cause movements in the vicinity of the heart to be felt in more distant parts of the lung. Similarly the alternating distension and recoil of a large artery might affect parts in direct contact, or, more rarely, at a distance.

I have little doubt that these considerations will explain the broad fact that breath sounds may be rhythmically modified in intensity by the movements of the heart, but I cannot pretend to explain the physical detail in all the various conditions in which this sign may be noticed.

A less common form of this modification of the breath sounds by the impulse of the heart is exemplified in the following case :—

Case xxv.—A. F., male, aged 19. Large, over-acting heart, with mitral disease from rheumatic fever. The respiratory sounds are jerky all over both sides, back and front, with the rhythm of the heart beats. The interruptions are only noticeable during inspiration, and are especially distinct towards the end of inspiration. A murmur or whiff is thus produced, which is heard most clearly below the tip of the left scapula.

Here we see the connection of the whiff with the interrupted or jerky respiration which is sometimes noticed with an over-acting heart. The occurrence of such jerky breath sounds is referred to in most text books on examination of the chest, and when, as in the foregoing case, the jerky respiration is heard all over the chest, its significance is not likely to be misunderstood. When, however, the distinct whiff or murmur, which was here present below the left scapula, is alone audible, without jerky breathing

elsewhere, there is more risk of mistaking the meaning of the sign.

There is little significance in the sign *per se*, though I believe it is always associated with a general debility. The real importance of a recognition of the origin of the murmur lies in the risk of mistaking it for a sign of disease of the heart valves, and of suspecting also some lung mischief from the apparent conduction of a cardiac murmur to a distant part.

It is not to be supposed that a physical sign which is so frequently present should have hitherto escaped notice, but I can find little comment on this *bruit* in medical literature. The late Sir B. W. Richardson (*c*) drew attention to this cardio-pulmonary sound under the title of an auscultatory sound produced by the action of the heart on a portion of the lung, and recorded three cases which had come under his notice. All these cases ended fatally, and Richardson was from this led to consider that the sign might have some serious significance. In each case the murmur was heard on the left side of the chest, in the neighbourhood of the nipple, and *post mortem* a piece of lung was found to be drawn down between the left ventricle and the thoracic wall, where it was held by adhesions. In one of the cases the pericardium was also adherent to the lung. Woillez (*d*), in reporting a case of thoracic deformity, notes that the vesicular respiratory sound was reinforced at each pulsation or dilatation of the aorta, and refers to Richardson's paper for examples of a similar modification

(*c*) *Medical Times and Gazette*, 1860, i. p. 187.
(*d*) *L'Union Médicale, tomes* 5—6, p. 515.

of the breath sounds. Richardson's cases are still referred to, and apparently few cases of this common physical sign have been recorded.

Before the introduction of instruments for auscultation, and even up to the time when the binaural stethoscope came into use, this sign would probably have escaped recognition except by the masters of auscultation. Now that we have means which are in the reach of all for intensifying the chest sounds, it becomes more necessary to call attention to sounds which may mislead the auscultator. Most physicians with experience of chest diseases must have noticed this sign frequently, and have referred it to its proper cause. I know that this is so with some of our best-known specialists. But amongst the greater number of practitioners to whom I have mentioned or demonstrated this respiratory "whiff," its occurrence seems to have been unsuspected.

The intensity of the sound varies considerably. Sometimes it is only heard by careful attention, and would be easily overlooked; at others it may even be heard at some distance from the patient (as in Case IX). It is in the more obvious cases that the sign is the most misleading.

Note.—Since the above article was written, a new work on diseases of the lung has been published (*Diseases of the Lung*, by Kingston Fowler, M.D., and Rickman Godlee, F.R.C.S.), in which this respiratory "whiff" is mentioned. Dr. Fowler, however, appears to consider that it occurs only in cases of Tuberculosis of the lung; the cases recorded in this paper show that this is not the case.

PNEUMONIA.

CHAPTER XXIII.

SOME CLINICAL REMARKS ON PNEUMONIA.

The latter part of the present century has so advanced our knowledge of the influence of bacteria on most pathological processes that a study of their relation to the more important pulmonary diseases claims special attention. The various parts taken by micrococci and by certain distinct kinds of bacilli, and how far they share in the initiatory and sequential phenomena of the differing forms of inflammation of the lung, have to be considered. Not only has the germ theory of disease given increased interest to pathological investigation and to experimental research, but it has resulted in larger and clearer views of ætiology and has important bearings upon clinical medicine both in prognosis and therapeutics. In the latter sense I do not here refer to the attempts at treatment by bactericidal drugs; this is now in the actively empiric stage, but it is one thing to kill bacteria in a test-tube and another to destroy them in the body. In the study of disease causation by the light of our knowledge of morbific organisms, two factors demand attention—the seed and the soil; not only must the micro-organism be studied, but the conditions under which it will flourish. This is

well recognised by the bacteriologist in preparing his culture media, and to the clinical observer and practical physician the influence of the soil on the life and virulence of the disease-germ are of the greatest importance. The "personal" equation in medicine has received a new interest and an increased importance from the discovery of the association of bacteria with certain diseases. The influence of the "personal equation" in disease is no new discovery, but we may note in the present day a greater tendency than was formerly the rule to treat individuals rather than diseases. In pneumonia the influence of the personal factor is well seen. Inflammation of the lung in a healthy child is a very different matter from the same accident occurring in an adult of impaired or shattered constitution, even though the initial lesion were of less extent or intensity. It is this individuality of each case of pneumonia which, whilst it renders the disease exceptionally interesting to the clinician, makes the management of each case a matter of peculiar anxiety. I propose to go through my case-book and touch upon some points of interest which my experience has furnished.

Ætiology.—It is now almost universally agreed that acute lobar pneumonia must be considered as a general, not merely a local, disease. And although there are difficulties in the way of fully accepting this view, there are many features of the illness which give it strong support. The discovery by Friedländer and Fränkel of the micro-organisms with which their names are now associated seemed to furnish the proof of the general nature of the illness. We must, however, remember that similar or

identical organisms are associated with other diseases besides pneumonia, and are to be found in the saliva in health, and probably exist in the lungs and other organs without producing ill-effects. When some local change in the tissues furnishes a suitable soil these micro-organisms develop rapidly and produce inflammation, whilst the toxic substances formed by them give rise to general symptoms. These local changes may be caused in various ways – for example, by traumatism or by chill— and they are more readily induced in organs of lowered vitality, whether this is permanent or temporary. Thus we find alcoholism predisposes to pneumonia, and that inflammation of the lung is a common accident in many illnesses where the general vitality of the tissues is impaired—*e.g.*, typhoid fever and influenza. So, too, the fatigue consequent upon excessive exertion renders a chill more dangerous; the overdone competitor in a long-distance bicycle ride finishing in a cold drizzle is more likely to get pneumonia than the spectator who has stood shivering to watch the result. It is a fact, sometimes imperfectly recognised, that over-fatigue is an important factor in the production of pneumonia. I have seen more than one case of pneumonia following a chill during the latter part of a long bicycle ride. The changes in the lung tissue which favour inflammation are probably the result of nerve influence which may originate at a distance from the affected organ. We know that in animals inflammation in the lung will result from injury to the vagus. The influence of chill to the surface of the body probably affects the lung by reflex nerve action. Such a result is perhaps most likely to ensue with exhausted nerve control,

and pneumonia from chill is thus more prone to happen during fatigue. The pneumonia of alcoholism has been considered to be a trophic neurosis originating at the vagus centres, and possibly the poisons of enteric fever and influenza may act in a similar manner. On the other hand, the lung tissue may be directly damaged by local irritation. This may be mechanical, as from a blow, fractured ribs, or inhaled mineral particles, or the local irritation may in some cases be due to the toxic effects of micro-organisms. It may be that even local irritation produces its effects by reflex nerve action. These considerations may help us to understand the occurrence of pneumonia under such widely different conditions as traumatism and chill, or as a secondary result of disease.

Just as fatigue increases the risk from chill, so may the debility after an illness render a chill more dangerous. In the following case we have a good example of the effect of measles in predisposing to pneumonia from chill. A barrister aged about thirty-five years had an attack of measles, which went through its course without any noteworthy characters until the patient was sufficiently well to sit up in his bed-room for an hour or two during the day. Being so well, he sent the nurse out for a walk, and in her absence he took a bath, and then, without completely dressing, he threw open his window, although it was in March, and began to arrange some papers. That evening he had a rigor, and the temperature, which had been normal for some days, ran up. He had a sharp attack of pneumonia. The readiness with which convalescents from measles suffer—and often suffer severely—from chill, though well known to medical men, is imperfectly appreciated by

others, and the large mortality after measles is due to the preventible complications and sequelæ. The case I have just mentioned is one of idiopathic pneumonia following after measles and is quite different from the pneumonia which occurs in measles as a part of the disease, coming on before the original fever has disappeared. The lungs are always affected to some extent in measles (as a part of the disease) and are liable to remain for a time in a condition which renders them a suitable soil for the development of any micro-organisms which may be present in these organs already or which may get to them from outside. Thus we find measles often an antecedent to pulmonary Tuberculosis.

I have seen several cases of pneumonia associated with enteric fever and some of them have had this point of special interest—Was the pneumonia present the substantive disease and the actual cause of the illness or a complication not infrequent of enteric fever? The recognition of the signs of pneumonia has, in some cases which I have seen, been taken to explain the illness and so misled as to the true nature of the case. Whenever, in obscure cases, signs of pneumonia are detected it is important to consider whether the condition of the lung will sufficiently explain the course of the illness as well as the existing symptoms. As a good instance of the importance of this I may refer to a case I saw recently in consultation. I was asked to see a boy aged about eleven or twelve years because pneumonia had been diagnosed. On examination there was no doubt as to the presence of pneumonic patches in both lungs, especially the right; but the lung trouble did not seem to me sufficiently intense to explain

the serious condition in which the boy obviously was at that time. Further examination disclosed pericarditis with effusion. Pericarditis may complicate pneumonia, but here on further inquiry I was led to a diagnosis of acute rheumatism, which the further course of the illness seemed to justify. The boy recovered, but with an endocardial murmur, which has now, after three months, almost disappeared.

The pneumonia of influenza presents some peculiar characteristics. The sputa may be rusty and the symptoms severe, but the physical signs are often obscure and the course of the disease unexpected. One marked peculiarity which I have noted is the tendency of the pneumonia to appear in some distant part of the lung or on the opposite side when resolving at the original seat. Here is a characteristic case. A young man about twenty-five years of age was seen in consultation in the country. On the right side of the chest the percussion note was "tubular" (high-pitched and sharp, with diminished resonance, but not dull) from the apex to the fourth rib, with tubular breathing. The rest of the right front was clear except for a few crepitations. The right back was dull almost all over. The left side was clear. Expectoration was very tenacious and had a slight brick-red colour, not typically pneumonic. The medical history showed that the pneumonia commenced at the right base, and as that resolved the apex became affected with another rise of temperature. This is quite different from what is found in typical acute pneumonia.

With reference to the influence of malaria on pneumonia, in my experience at Suakin in 1885 malaria was not a

frequent cause of illness, except in those cases which have been called "typho-malarial," and which I believe to be dependent upon malarial poisoning. In these cases, as well as in some of the cases of enteric fever there, pneumonia occurred as a complication. A malarial pneumonia has been described; but Osler, with his large experience of malaria, says he has never yet met with a case of pneumonia which seemed to be in any way connected with paludism. The debility which is induced by a malarial attack may, however, predispose to pneumonia, as in the following case. A man aged thirty-three years arrived in England in April from the West Indies, where he had been for about five months, and had been eight weeks seriously ill with malarial fever. I saw him in consultation the day after he landed and found pneumonia affecting the right apex. When I saw him again, three weeks later, the chest was clear, the pneumonia having completely resolved. Tubercle had been suspected as the cause of the lung mischief, but no bacilli were found in the sputa, and the complete resolution within three weeks is in itself almost sufficient to confirm my opinion that the pneumonia was not due to Tuberculosis. The patient recovered and still keeps well after nearly three years. Whether malarial fever predisposes to pneumonia or not, the cachexia left after severe malarial poisoning adds to the danger of a subsequent pneumonia. Some years ago I attended a man aged thirty-nine years during an acute attack of pneumonia, from which he died on the tenth day. The patient had served in the marines and had suffered from malaria. The illness with the pneumonic attack was very acute, the temperature was high, with delirium from the fifth day.

In this case I believe the previous malarial poisoning had left him without power to resist any severe illness, for his age and habits were not unfavourable to recovery.

The foregoing remarks will emphasise the importance of the "soil," in addition to the causative bacteria, as a factor in pneumonia. There is this analogy between pneumonia and Tuberculosis: in both a micro-organism is the essential cause, but the germ is harmless unless the soil is favourable to its development. The difference between the two diseases depends upon the different characteristics of the causative micro-organisms. The pneumococcus or pneumobacillus, developing rapidly, quickly produces its full effects, and sufficient toxines are formed to cause an acute illness and usually to poison the germs before they can become widely distributed. Thus we have a sharp but short illness usually terminating by crisis. The tubercle bacillus also produces a pneumonia, but develops slowly; the consequent small total output of toxines in the early disease leaves the blood-stream non-poisonous to the bacilli, and they are carried by it to other parts, where they set up fresh centres of disease. In pneumonia, as in Tuberculosis, the various predisposing causes, such as cold, exhaustion, or debility, render the individual susceptible; the character of the "tissue soil"—to quote Osler (*a*)—has been so changed that the specific germ of either can grow and produce its ultimate effects.

Course and Symptoms of Pneumonia.—There is a very interesting point on which opinions are not unanimous—

(*a*) *The Principles and Practice of Medicine*, 2nd edition, p. 547.

namely, whether there are cases which justify a diagnosis of acute congestion of the lung. The French consider active congestion of the lung to be a distinct disease (maladie de Woillez), but most authorities in this country consider the cases so described to be cases of pneumonia. Cases undoubtedly do occur, commencing suddenly with the symptoms which mark an attack of pneumonia, which either terminate fatally before the lungs become consolidated, or which resolve without going through the ordinary course of pneumonia. Such cases may be termed "abortive pneumonia," and these are the cases to which the term "acute congestion of the lung" is sometimes applied. I select two of my cases bearing upon this point—one in a child, the other in an adult. A little girl aged four and a half years, who was said to have had congestion of the *left* lung the previous winter, was taken ill rather suddenly with feverish symptoms and slight cough. I found the temperature considerably raised and the base of the *right* lung dull. A mustard poultice was applied, and the next day the dulness had cleared and the temperature went down in two days. In the other case a lady past middle age, having just got over an illness of a few days' duration (supposed to be influenza) was found to have a return of fever with signs suggestive of pneumonia. When I saw her the next day the temperature was 101° F., and there was diminished resonance over the right base up to the angle of the scapula, with weakened breath sounds and fine crackling over the affected area. A mustard leaf was applied. I again saw her three days later. The temperature was down and the patient felt well. The right base was resonant, the breath sounds still somewhat weaker than on the left, but there

were no crepitations. This was hardly a hypostatic congestion, for it was accompanied with a rise in temperature and the conditions which induce hypostatic congestion were absent. These two cases, though presenting different clinical phenomena, have this important characteristic in common—viz., that in both the febrile illness was short and the consolidation of the lung was rapidly absorbed, whereas the consolidation of pneumonia usually takes some time to resolve after the febrile illness is over. In this sense they may both be described as "abortive" pneumonia.

The pathological conditions found in pneumonia may be referred to in explanation of such cases. These, as given by Dr. Douglas Powell, consist "first of an acute hyperæmia of the affected lung resulting in a fibrino-corpuscular exudation into the alveoli, sometimes including the smaller bronchioles and forming a film upon the pleural surface. This exudation, secondly, coagulating *in situ*, fixes the lung in a state of immovable expansion more densely solidified than it could be by any artificial injection with coagulable fluid" (*b*). The facility with which these exudations may be absorbed varies with the nature of the effusion and of the corpuscular elements and the degree of coagulation obtained. In the cases of which I have just been speaking the fluid exudation is presumably absorbed before coagulation takes place. We have thus only the first stage of the pneumonic process.

It is unnecessary here to describe the symptoms and course of an ordinary case of pneumonia; but I propose to

(*b*) Dr. Douglas Powell: *Diseases of the Lungs*, 4th edition, p. 221.

mention one or two peculiarities or divergencies from the normal course which have come under my notice. We sometimes find that in cases of acute pneumonia the fever subsides gradually instead of by crisis, and it has seemed to me that this is more common in hospital practice than in cases seen at home. Possibly removal of the patient may have something to do with this. If a patient is taken to hospital when the disease is well established—say from the third day onwards—the febrile period becomes somewhat prolonged and the crisis is delayed or wanting.

When consolidation of the lung has occurred resolution necessarily takes time, and in the most favourable cases is not complete until some days after the temperature has fallen. We meet with cases, however, in which this resolution is delayed for a considerable period, it may be several weeks or even months. I have notes of a case of a girl aged twelve years in whom resolution was not complete for nearly two months, but it may be delayed longer than this. Such cases may get absolutely well, as this girl did, the lung eventually clearing up completely. In other cases where resolution is delayed we find some further mischief, either as the cause or a result of the delay; for example, tubercle or pleuritic effusion, or an abscess may form in the lung. I have in a previous paper (*c*) before the Harveian Society drawn attention to the risk of Tuberculosis after pneumonia, especially in those cases where resolution has been long delayed. The pneumonic process nearly always leaves the lung in a condition favourable to the growth of germs which may subsequently reach the

(*c*) *Medical Press and Circular*, April 20th, 1892.

organ; thus we find that, unlike what is the rule in a general disease, one attack renders the person more liable to suffer again from pneumonia and predisposes also to Phthisis. Some years ago I had a case of gangrene of the lung following pneumonia (*d*), and I have lately had an interesting case of pneumonia affecting the upper lobe of the left lung in which a pyo-thorax developed which was also localised in the upper portion of the left chest. The patient, a boy ten years of age, was one of three inmates of a school who developed pneumonia. In due course the crisis occurred in all, but whereas two of the boys had an uninterrupted convalescence the temperature of the third, after being down for a day or two, ran up again and showed the large diurnal range of septic fever. The left chest was immovable in respiration and quite dull on percussion. The boy had a tubercular family history, and I took him into hospital to have him under observation. Being satisfied of the presence of fluid in the pleural cavity, I got Mr. Watson Cheyne to open the chest. A large quantity of offensive pus was evacuated and the boy made a good recovery. I have had several cases of empyema following pneumonia, but this case of empyema localised at the apex is exceptional.

The case just quoted leads one on to refer to cases of apex pneumonia. Pneumonia affecting the apex of the lung is not necessarily more severe or more dangerous than inflammation occurring at the base. Cases of apex pneumonia are sufficiently common to make it unnecessary for me to quote instances here. One important point of

(*d*) *The Lancet*, July 20th, 1889.

interest in these cases is the difficulty of distinguishing between a simple pneumonia in this situation and Tuberculosis. The apex of the lung being the most frequent seat of commencing Tuberculosis, we may be led to diagnose Phthisis when we get consolidation of the apex without fully considering the history and physical signs. It is important also to remember that pneumonia may be induced by a patch of tubercle, the presence of which is not recognisable until the surrounding pneumonia subsides. I have elsewhere referred to cases illustrating this and other points in reference to the connection between pneumonia and Tuberculosis (*e*).

With regard to the sputum in pneumonia, viscidity is perhaps a more general characteristic than the rusty colour. In catarrhal pneumonia the rusty colour is not infrequently absent, and it may be so in lobar pneumonia. On the other hand, I have seen the characteristic rusty sputum when careful examination failed to detect any dulness; here the pneumonic patch was probably deep-seated. The prune-juice expectoration, which is often a sign of danger, is not always so. The change from marked viscidity to a more liquid as well as darker sputum is more significant of danger than the deepened colour alone.

Diagnosis.—Theoretically the signs of pneumonia are sufficiently characteristic to be recognised easily and with certainty, but practically we find ourselves often obliged to weigh carefully all the evidences obtainable before we

(*e*) *The Hygienic Prevention of Consumption.* Charles Griffin & Co., 1893.

can give an opinion. I have mentioned the difficulty occasionally met with in deciding between Tuberculosis and pneumonia, and I have on more than one occasion found a pleural effusion where pneumonia has been diagnosed. Pleurisy may follow pneumonia, and when the signs of pneumonia have been correctly recognised in the first place dulness on percussion at subsequent examination has been interpreted as indicating non-resolution when it was in reality caused by fluid. Careful auscultation should be practised throughout the course of a case to avoid such an error, and repeated testing of the vocal fremitus must not be omitted; but even with this care the mistake is not always avoidable. The dulness of hypostatic congestion or of œdema from heart or kidney disease and the dry crackle of emphysema may at first suggest the presence of pneumonia. But with the aid of the thermometer and consideration of the history and general symptoms, there should be no difficulty in forming a correct opinion. The marked alteration in the pulse-respiration ratio—due to the quickening of the breathing out of proportion to the acceleration of the pulse, till we may have one breath to every two pulsations instead of one to four—may often guide us to the detection of even a limited pneumonia. Finally, we have to remember the possibility that the pneumonia may be symptomatic of some disease—such as typhoid fever—requiring the fullest attention, or an accident of a Tuberculosis which would modify prognosis.

Treatment.—I strongly advocate the stimulant treatment of pneumonia. We have here an illness which is of short duration but often of great severity, and recovery is almost

certain if we can keep up the patient's strength till the fever is past. As most of us know by experience, exhaustion may come on so rapidly that if we wait until it shows itself treatment may be too late to counteract it. We may, however, do much to prevent this excessive exhaustion by commencing with stimulant treatment from the first. By stimulant treatment I do not mean necessarily or solely alcoholic treatment, though I consider alcohol one of our most important drugs in the treatment of pneumonia. A full supply of nourishment in suitable form is necessary as well as stimulant drugs, and good nursing is essential. In many cases the nurse is what the practitioner makes her; and if the medical man shows a knowledge of what good nursing means and gives minute instructions to the nurse, taking measures also to satisfy himself that his instructions are carried out, he will probably have little cause to complain of want of attention on the nurse's part. In severe cases I am in the habit of writing out a time-table for the nurse, stating not only the hour at which nourishment, stimulant, or medicine shall be given, but the nature and quantity of each to be administered on each occasion. I then expect the nurse to keep a diary which I may compare with my time-table at every visit. Whether it is ever allowable to cease giving a patient food, drink, or medicine on the grounds that it can do no good and only bothers the dying patient we need not now discuss. It is certainly never justifiable in a case of pneumonia. I can recall two or three instances of recovery after a patient had been "given up," in which any relaxation of the details of nursing would have taken away the last hope. I cannot too strongly insist on this point.

Local applications over the affected portion of the lung are, in my opinion, very useful. Whether they exert any appreciable influence on the disease may be questioned, but they often ease the patient's discomfort and soothe him. I prefer a poultice as generally most comfortable to the patient, but ice-bags are also recommended. The inhalation of oxygen is often of service, especially when a large extent of lung is consolidated; and I have seen marked improvement in breathing and in general symptoms from placing a patient with high fever in a tepid bath, after which the patient dropped off into a quiet sleep. As to drugs, there is room for individual choice within a certain range, and symptoms will guide us in our selection. I have a liking for carbonate of ammonia, to which digitalis or strophanthus may be added if there is any danger of cardiac failure. A caution is to be observed as to the use of even simple aperients. Of course the patient must not be allowed to get up, and sudden danger may result from undue action of the bowels. An enema may be needed, and this means of giving both nutriment and alcoholic stimulant also must not be overlooked.

With regard to alcohol, I prefer to begin to use it early in small doses (two to four ounces in the twenty-four hours), and to increase the quantity on the first indication of increasing weakness. Many cases will complete their course without needing any increase of the initial amount, but I have known cases where the alcohol has had to be given in large amount for a day or two. I sometimes tell the nurse in serious cases that if exhaustion is great an extra half-ounce of brandy should be given before calling the medical attendant. I well remember when I was a

house physician, a patient with double pneumonia who took about thirty ounces of brandy in one twenty-four hour period. The case was pronounced hopeless by the visiting physician, but I and the nurse decided that we would keep the patient alive over the crisis if it could be done by stimulant, and we succeeded. This experience coming so early in my medical career made a great impression on my mind. I believe there is no better stimulant for use in the exhaustion of pneumonia than alcohol; others I know prefer not to use alcohol at all, but if they are wise they make use of some other stimulant in such cases as the one to which I have just alluded. As I have already intimated, your pneumonic patient will probably recover if you can keep him alive over the tenth day. The personal equation has such a marked influence on the character of the disease that, though we may lay down some general principles for the treatment of pneumonia, we must be guided as to details by the individual peculiarities in each case.

CHAPTER XXIV.

SOME FURTHER REFLECTIONS CONCERNING PNEUMONIA.

At the present time this disease—always a subject of medical interest—has a special claim to our attention. Deaths from "pneumonia" have recently been numerous, especially in the aged, and as a result of influenza. Pneumonia also figures as a cause of death amongst our troops in South Africa.

The question what is pneumonia has been discussed over and over again, and although we should probably say, in this country, that the matter is definitely settled in favour of the view that it is a general disease with a local manifestation in the lung, there is evidence that the discussion is by no means at an end.

The *Practitioner* for January devotes considerable space to articles on pneumonia by well-known men; Sir William Broadbent giving a general sketch of the disease, Sir William Gairdner discussing the treatment, and Dr. Hector Mackenzie dealing with the effects of age and season on its prevalence and mortality.

Whilst pneumonia affects individuals of all ages, its mortality is largely influenced by age, and at the extremes of life its proportionate mortality is very great. As to its seasonal prevalence, it is not so much in the coldest months

that pneumonia is most common, but in the season of rapid and marked changes of temperature, and of high winds. The largest number of cases occur, in this country, in May rather than in December.

Sir William Broadbent, speaking of ætiology, says "the most common antecedent is exposure to cold, and the fact that the disease is the work of a microbe must not make us oblivious of the important part played by chill in the causation of pneumonia."

Dr. Mackenzie, on the other hand, concludes, from his examination of statistics, that cold and wet do not play such important parts in the causation of pneumonia as is popularly supposed. Only 14 per cent. of the cases admitted to St. Thomas's Hospital give a history of a chill or wetting.

These slight differences of opinion are quoted in support of the opinion expressed above, that we cannot yet have done with discussions on the ætiology of pneumonia.

The term "acute lobar pneumonia" is at the present time generally restricted to the acute febrile illness of almost definite course in which inflammation of the lung is the most prominent pathological sign. In this illness, the inflammation of the lung is not considered to *constitute* the disease, but merely to be its most noticeable expression.

Undoubtedly, the usual regularity of course and duration noticed in cases of acute lobar pneumonia suggests a specific general disease; and the course of the illness suggests a microbic origin. One difficulty, however, which stood at first in the way of accepting the theory of a microbic origin for all cases of acute pneumonia was the apparent dependence of the disease upon chill as an exciting cause.

That the illness sometimes follows a chill so closely as to justify a belief that their association is that of cause and effect is a clinical experience which cannot be denied. The discovery of micro-organisms in association with pneumonia points, however, to a microbic origin.

We have a somewhat analogous state of things in acute tonsillitis. Here the high temperature, general illness and almost definite course suggest a specific general disease rather than a purely local inflammation. Cold and chill are looked upon as a sufficient determining cause, though there is evidence of microbic association in some cases at least.

In both illnesses it is difficult, with our present knowledge, to avoid a belief in more than one possible mode of origin.

We know that pulmonary inflammation may be caused in various ways; and perhaps I shall best explain the difficulties in accepting any single theory of causation as a sufficient explanation of all cases of acute lobar pneumonia by a very few words on lung inflammation in general.

(A) Inflammation, in the lungs as elsewhere, may be brought about by nerve-influence originating at a distance from the inflamed organ. This is evidenced by the results of damage to the vagus nerve. This nerve-influence may apparently be also brought about reflexly, by chill or cold to the surface of the body.

Such a result is, perhaps, most likely to ensue with exhausted nerve control, and pneumonia from chill is, as we know, most likely to happen during fatigue.

The pneumonia of alcoholism has been considered, and

probably correctly so, a trophic neurosis, acting at the vagus centres; and possibly the poisons of influenza, measles, typhoid, &c., may act in a similar manner.

(B) But, in the second place, the injury to the vascular walls and other tissues, which causes the pathological conditions known as inflammation, may be due to local irritation. This may be purely mechanical, as from a blow, fractured rib, or inhaled mineral particles; or the local irritation may be caused by micro-organisms, whether cocci (Friedländer's or Fränkel's), tubercle bacilli, or other organisms. It may be that the pneumonia of measles, influenza, &c., is thus caused by local irritation instead of through the nerve centres.

Even local irritation may act through reflex nerve action rather than by direct injury; if this be so, then both modes of origin come under the heading of neuroses. The intensity and character of the inflammation differs with the different causes indicated above.

Arguing from the general to the particular, it appears to me possible that acute lobar pneumonia may be caused (a) by nerve-influence—either originating in the nerve centres or conveyed to them by afferent nerves (as from the surface of the body), and reflected to the lung. Such pneumonia might fairly be classed as a neurosis.

In addition, it would seem that acute lobar pneumonia may also depend upon (b) a local irritation—in all probability microbic. In this latter case the cause of the inflammation of the lung would also affect the blood, and would produce a general disease with a local manifestation in the lung.

The morbific micro-organisms might presumably be

conveyed from the sick to the susceptible, and infectious or epidemic pneumonia would be thus explained.

The non-specific causes of inflammation—chill, irritation, &c.—might prepare the lung for the reception and development of the specific bacteria.

It may seem to some rather late in the day to suggest that the term "pneumonia" requires clearer definition, and that differences of opinion will often disappear if the meaning of the term be agreed upon by the disputants.

"Pneumonia," as used in this country, may refer to inflammation of the lung however produced. We seem to want some general term to include all lung inflammations, and to limit the term "pneumonia" to the acute disease which is so well marked in its clinical manifestations.

Sir William Broadbent evidently had some such idea in his mind when he writes:—"Pneumonia is a very remarkable disease. Whatever criterion may be applied, it must be looked upon as an inflammation, but there is no other inflammation of an important organ which at all corresponds with it in the suddenness of its onset, the rapidity of its progress and the abruptness of its termination."

Dr. Andrew Smith, of New York, writing on the treatment of pneumonia as based upon recent views as to its pathology (*The Scalpel*, Jan., 1900, p. 2), expresses what at first sight appears to be a direct contradiction of Sir William Broadbent's view, though, as we read further, we find the difference is chiefly a matter of definition. Dr. Smith says:—"Pneumonia is not an inflammation of the lung," but his next sentence shows that he uses "pneumonia" in its limited sense, and employs the word "pneumonitis" as the general term to signify inflammation

of the lung whatever its cause. He continues:—"You can create a pneumonitis at will in any of a dozen or a hundred ways. There is only one way in which you can produce a pneumonia. You may bruise, tear, pierce, or scald the lung, you may force acrid gases into it; nay, you may introduce any foreign substance into it, including, with one exception, any conceivable pathogenic organism, and you will not get pneumonia as the result. You may get inflammation, suppuration, even gangrene, but it will not be pneumonia, and it will not have the clinical history of pneumonia. But there is just one thing which, introduced into the parenchyma of the lung, will produce pneumonia always and without fail, and that is the pneumococcus." There is here a very definite distinction drawn between the disease "pneumonia," and the pathological condition of inflammation in the lung. Pulmonary inflammation is an almost invariable concomitant of pneumonia, but it is also not infrequently a symptom or complication of other disease, such, for instance, as enteric fever and influenza. It is the inclusion of "secondary" or "symptomatic pneumonia" in descriptions of the disease, which most frequently leads to apparent divergence of opinion.

But again, if pneumonia is not primarily inflammation of the lung, but a disease caused by the pneumococcus in which inflammation of the lung is generally one of the morbid results, should we not call all disease produced by this micro-organism "pneumonia," whether the lung becomes affected or not?

We call all morbid effects of the tubercle bacillus "Tuberculosis," whatever part of the body is affected. A

case of double empyæma in a child, cured by operation, is recorded in the *British Medical Journal* (Jan. 6th, 1900, p. 17), which is properly a case of pneumonia in which empyæma was one of the morbid effects of the disease; a result of the activity of the pneumococcus. Empyæma is not infrequently caused by the pneumococcus, and is probably preceded by or accompanied with inflammation of the lung—the disease is a "pneumonia." It is, however, open to question whether a classification of diseases based primarily upon the causative organism is desirable. Preventive medicine deals primarily with causes; general medicine deals with effects. For the practitioner whose aim is to treat these effects, the best classification would appear to be one based not so much on immediate causation as upon the pathological results which are produced. Whatever the basis of the classification, however, treatment must take causation into consideration; and though the main divisions should be based upon pathology, the sub-divisions would indicate causation. There is no doubt that a classification of disease which is based upon symptoms or clinical characteristics requires to be modified, if not entirely replaced, as knowledge of ætiology and of pathology advances. The following remarks are taken from some comments made in my note-book, several years ago, upon a term then recently coined, and serve to illustrate the thought expressed above.

The term "pulmonary hypertrophic osteo-arthropathy" is a sort of descriptive title. As each morbid state is modified in the individual according to the constitutional condition of the patient, we might have a different descriptive name for almost every case we see. Already such

names are being multiplied, and this multiplication will, perhaps, gradually lead to a reaction and result in a simplification of our classification of disease. Such a simplification seems to me to be desirable, but will be delayed by the difficulty in breaking away from the old text-book classifications. The new nomenclature must be based on pathology, not on symptoms; jaundice, dropsy, aphasia, asthma, &c., &c., must take their place with pain, cough, &c., as symptoms only.

Pulmonary hypertrophic osteo-arthropathy appears to be a bony hypertrophy following the partial stagnation of blood in the extremities from interference with the circulation, here due to obstruction in the pulmonary circulation. The simpler results of such obstruction are seen in the clubbed finger-tips so common in chronic lung disease; but here (pulmonary hypertrophic osteo-arthropathy) there would seem to be also some peculiarity in the blood—possibly a microbe—which causes bony hypertrophy. In agromegaly there is a similar state of bony overgrowth. These conditions would come under diseases of the circulatory system (venous obstruction).

After jotting down a very incomplete classification under the heading of "Venous Obstruction," I followed the idea of classification by an illustration from the lung.

INFLAMMATION OF THE PULMONARY PARENCHYMA.

(A) *Simple* (or non-specific).

1. Injury to nerve (vagus), or centre in medulla.
2. *Irritation* from
   (*a*) Inhaled gases or fumes—chlorine, ether, &c. —generally acute.

(*b*) Inhaled particles—dust, metallic or mineral particles—generally chronic.

3. *Traumatism.*—Injury and wounds of lung.

(B) *Specific* or *Microbic.*

1. Idiopathic Pneumonia (pneumococcic).
2. Septic Pneumonia (streptococcic).
3. Secondary Pneumonia (influenza, enteric, &c.)

} generally acute.

4. Tuberculosis.
5. Cancer.

} generally chronic.

The resulting pathological changes in the lung depend partly upon the intensity and acuteness of the inflammatory process; if short and sharp, we get the well-known pathological changes associated with acute pneumonia, and usually ending in resolution; if slower and less acute, we get fibrosis. In addition, we find in the microbic inflammations specific effects of the special toxines (*e.g.*, caseation of Tuberculosis). The simple inflammations extend from the original centre by contiguity only. Specific inflammations may, in addition, be started in fresh centres in distant parts by infection carried by the blood and lymph streams or along the bronchial tubes.

The thoughts expressed above—immature in themselves and incomplete in their expression—indicate the need for a more definite application of the term "pneumonia." If it be restricted to the acute disease caused by the pneumococcus, we require another term to express pulmonary inflammation of different causation; or, if pneumonia is to

include all inflammation of the lung, a new name is required for the acute effects of pneumococcic infection.

Such discussions are not purely academic; a clear understanding of what is the pathological condition referred to under the term "pneumonia," and of the immediate cause of this condition, must determine the principles of treatment.

Whatever the ætiology, there seem to be two clear indications for treatment:—

A. To soothe the local condition.

B. To maintain the general strength against the exhausting effects of the illness.

Locally, poultices give relief, and are thus valuable.

The stimulant general treatment, which seems to me the most satisfactory, includes a sufficiency of nourishment, as well as medicines.

Carbonate of ammonia is, in my opinion, one of the most useful drugs, and with this digitalis (or strophanthus) may often be needed to prevent cardiac failure.

Alcoholic stimulants are in my experience most valuable aids in the treatment of acute lobar pneumonia. They should be given *to prevent* exhaustion, not reserved until collapse is imminent, when they may come too late. I have seen several apparently hopeless cases of acute pneumonia recover with stimulant treatment and good nursing—which latter is essential. For the reduction of pyrexia, when excessive, sponging the body with vinegar and water will often be sufficient, and I have seen good results from tepid baths. Antipyretic drugs are most of them less satisfactory than sponging; and some of them, from their depressant effects, are injurious.

CHAPTER XXV.

EXPERIMENTAL RESEARCH.

During 1891 and 1892 I was engaged at the laboratories of the Royal College of Physicians and Royal College of Surgeons in some experimental investigations with reference to the ætiology of Tuberculosis.

My first series of experiments had for their object to test the influence of damage to one lung in determining the seat of Tuberculosis on infection. I endeavoured to produce pneumonia on one side by crushing the vagus of that side; and to imitate the effect on the lung of pleuritic effusion, by injecting fluid into one pleural cavity. I expected to find that when tubercular material was inhaled, the damaged lung would become tubercular more readily than that which was undamaged.

I experienced great difficulty in the preliminary preparation of the animals (rabbits were used). First as to crushing the vagus in the neck. I decided to damage the right vagus to avoid interfering with the heart.

In all cases, however, there was an immediate and considerable acceleration of the heart beats directly the nerve was damaged.

In one rabbit, where the right vagus was divided, the animal died on the sixth day, and the conditions noted after death were: pericarditis, pneumonia of the right

lung, (hypostatic) congestion of both bases, especially the left, congestion of the mucous membrane of the trachea, bronchitis on both sides. After several failures I succeeded in getting two rabbits with apparently right-sided pneumonia from crushing the vagus.

Next as to fluid in the pleural cavity. I found it possible in the dead animal to force 100 c.c. of water into the right pleural cavity, but much of this ran out as soon as the syringe was withdrawn. I considered that perhaps 50 c.c. might be retained. I injected then 15 c.c. of sterilised Lucca oil into the pleural cavity of a live rabbit, and three weeks later a larger quantity—the exact amount retained could not be determined, but was probably nearly 50 c.c.—into the same side. I endeavoured to fix one side of the chest in another rabbit, but failed. The looseness of the skin was one difficulty, and the animals—being, as the attendant expressed it, "very handy with their feet"—always displaced any contrivance I could fix on the chest.

In another series I wished to produce Tuberculosis of the cervical glands (strumous glands) by infection through the mucous membrane of the pharynx; this being, in my opinion, the probable course in strumous glands in the human subject. I previously rendered the mucous membrane of the pharynx susceptible to infection, by painting at intervals for days with a weak solution of iodine, to produce slight inflammation. My first iodine solutions were too strong and produced acute inflammation. A solution of 1 gramme to 100 c.c. water was then used three times on the same rabbit; the throat was then apparently sore and feeding was difficult; it was used a fourth time, and the animal died the next day.

Another rabbit, however, on whom the solution of the above strength was used to paint the pharynx several times, survived and was sprayed (see Table). As will be seen by the following Table, the throat case was successful, but the other cases produced no reliable confirmation of the theory I wished to prove. An unexpected result in the control animals is of interest.

The result in rabbit A is important. The early death of the control animals D and E is interesting from the fact that when these animals were selected, the whole stock was in poor condition, and several had died. I examined some that died and convinced myself that the cause of death was not Tuberculosis. But the control animals were not such strong ones as those previously taken for preliminary operations. These strong animals, even when damaged, were less affected by the tubercular infection than the weakly but undamaged animals. This tends to show that general ill-health is a more potent predisposing cause of Tuberculosis, than is damage to a lung in an otherwise healthy individual. I have noticed clinically that in persons who trace the onset of Phthisis from catarrh or from damage to a lung without previous ill-health, the disease advances less rapidly and is more easily checked than is the case in constitutionally delicate persons who become consumptive.

Animal.	Preliminary Operation.	Operation on June 16th, 1892.	Date of Death.	Result.
A Rabbit.	Pharynx previously painted several times with Iodine solution, 1 gramme to 100 c.c.	Tubercular material sprayed into throat.	Killed July 26.	Cervical glands enlarged. Bacilli found.
B Rabbit.	About 50 to 60 c.c. of sterilised oil injected into left pleural cavity, July 10.	Do.	Killed July 26.	Left lung completely collapsed.
C Rabbit.	Right vagus nerve crushed, May 31.	Do.	Killed July 8.	Right vagus divided. Cervical glands enlarged. Left lung healthy. Right lung congested.
D Rabbit.	Nil. (Control.)	Do.	Died June 25.	Fluid in right pleural cavity. Right lung—lower lobe deep red and carnified. A larger patch of carnification in left lung. Catarrh of larynx. Inflammation of tracheal mucous membrane.
E Rabbit.	Nil. (Control.)	Do.	Died June 24.	Lungs, larynx and trachea as in **D**.
F Rabbit.	Nil.	10 min. of same tubercular material injected into peritoneal cavity.	Died June 26.	Intense peritonitis.

NOTE.—The tubercular nature of the inflammatory conditions found in **D** was shown by the presence of bacilli in microscopical examination. Also in **A**.

INDEX.

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