Treatment of consumption / by W. Camac Wilkinson.

TREATMENT OF CONSUMPTION

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TREATMENT OF CONSUMPTION

BY

W. CAMAC WILKINSON

B.A.Syd., M.D.Lond., F.R.C.P.

Lecturer in Medicine, University of Sydney
Hon. Physician, Prince Alfred Hospital, Sydney
Formerly Physician to Ear and Throat Department, Sydney Hospital

MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, LONDON
1908

RICHARD CLAY AND SONS, LIMITED,
BREAD STREET HILL, E.C., AND
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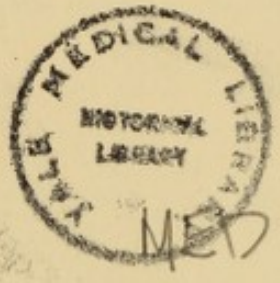

PREFACE

As a thank-offering to my distinguished teacher and as a means of commemorating the semi-jubilee of the greatest and most fruitful discovery of medical science, disclosed to the meeting of the Physiological Society of Berlin on March 24, 1882, I desire to present to the members of the medical profession in their own interest and in the interest of their patients this small work, which embraces an account of the results obtained in the treatment of Pulmonary Tuberculosis with Tuberculin in the different forms offered to us so generously by his Excellency Professor Robert Koch.

University of Sydney, 1908.

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TREATMENT OF CONSUMPTION

INTRODUCTION

WHOSOEVER undertakes to prove that everyone in his own particular society has been wrong, and he alone has arrived at the conscious possession of truth, takes great risks, and the odds are manifestly against him. Yet such a man may happen to be quite right. The glorious path of human progress has ever been illumined by such beacon lights. In modern times, Galileo, Harvey, Newton, Pasteur, and Koch are among these giants of the human race. Their work by its irresistible truth must survive them, and can never be undone. During their life they have been buffeted and persecuted, reviled and ridiculed, and too often have died before even their friends discovered they were right. For generations their enemies have scoffed and sneered. Nevertheless, Heaven has ordained that these discoverers of truth should have some disciples. Since 1884, when I had the honour of being a pupil of Koch, my allegiance has not once wavered, and in publishing this small work, I am compelled by a sense of duty to acknowledge that this great genius has been the source and inspiration of my life's work.

Looking at work with all its trials, its hardships, and its disappointments as part of the religious duty of man, it is encouraging and stimulating to think that one's own work may bring within reach of the poorest human creature the solace and comfort that must otherwise be denied him.

Through the profound genius of Koch, and Koch alone, a certain, safe, and trustworthy remedy has been found for the

treatment of pulmonary tuberculosis, which is but the name for consumption in its first beginnings, and it is high time that this great truth should be acknowledged and inspire the whole of the medical profession to imitate the labours of those who in season and out of season have preached this great gospel of mercy and beneficence.

To all my colleagues in this profession, in every part of the globe, I would say in all humility and earnestness, "Go and do thou likewise."

PART I

THE GREATEST PROBLEM IN MEDICAL SCIENCE

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THE GREATEST PROBLEM IN MEDICAL SCIENCE

Prevalence and Death-Rate of Pulmonary Tuberculosis.

If it is the function of the physician to cure disease, and thus restore the patient to health, a stupendous task lies before those of us who have to deal with the victims of tuberculosis. In England alone there are more than 55,000 deaths recorded each year—more than 150 deaths every day. In New South Wales the records are not so dreadful, but still there are probably nearly 1,500 deaths from tuberculosis each year, for statistical records underrate the incidence of the disease. In tuberculosis the mortality statistics—the death-rate—alone give us no clue to the actual number of persons suffering from this disease. Mortality is not morbidity, and there is not any known proportion between mortality and morbidity in tuberculosis. It is for the physician to reduce the mortality; for the hygienist to reduce the morbidity. Tuberculosis does not always and inevitably cause death. Many suffering from tuberculosis, chiefly in the very early stages of localised disease, get well without the physician's assistance and do not die of tuberculosis at all. There is no reason why these cases should not also get well under sanatorium treatment or specific treatment; and in such cases the recovery may be readily attributed to the particular method. *Post hoc, ergo propter hoc.* We must all plead guilty to this comforting method of reasoning. A still larger number may not get well, but survive for one, two, three, or many years before death brings the disease to an end.

In these cases the morbidity may endure through years, and all this while the victim is more or less maimed or disabled. This represents an *economic loss*, that fails to be recorded in mortality statistics. Further, many cases of Tuberculosis appear in the Registrar-General's report under more euphonious titles--pleurisy, bronchitis, enteritis, laryngitis, even meningitis, pneumonia, and typhoid fever—to which no social stigma attaches. Worst of all, pulmonary tuberculosis, far the commonest and most dangerous form of tuberculosis, exhibits its cruel malignity by laying its withering touch upon men and women in the very prime of life, and too often strikes down ruthlessly the fairest flower of the family. Thereby the economic loss is further intensified. The care, and often the savings, of devoted parents have been lavished upon their children, who have drooped one by one under the blighting influence of this insidious and deadly disease. However much we may dread cancer as a fatal disease, cancer has the saving grace of attacking its victim when the best part of life is over. Most of the victims of tuberculosis are in the beauty of youth or early maturity, and the very bonds of love and affection give this heartless enemy the best opportunity of showing its malignity. In women the most fatal years are from 15 to 35, in men from 20 to 40. These terrible features should make consumption the most-to-be-dreaded of all diseases, but, because its insidious onset deceives and disarms us, and its slow progress gives us plenty of time to get accustomed to its presence, consumption does not impress the ordinary mind as the most potent cause of misery and suffering—of disease and death—in the human race. If one-tenth of the number of deaths due to tuberculosis were due to plague, or cholera, or smallpox, there would be a public outcry against the inadequacy and futility of our measures for protecting the public health against such epidemics. There are every year in England 50,000 deaths from tuberculosis. Imagine the state of public feeling if the *Daily Mail* could have such a heading as "a thousand deaths every week from plague in England." Yet this does not adequately represent the death-roll from tuberculosis at the present time; and that too, let us remember, although the source of infection is well known and has been proved to be

limited in distribution. Is it, then, too much to say that far the greatest and most difficult problem awaiting solution at the behest of medical science in the name of suffering humanity is the prevention and successful treatment of consumption.

The term "consumption" may be used to connote pulmonary tuberculosis in its advanced and infectious form, and this infectious form of pulmonary tuberculosis is the essential, though not exclusive, source of all forms of tuberculosis. Whatever opinions may be held concerning the relative frequency of infection from cows in infancy and early childhood, pulmonary consumption has no such origin. The common disease, consumption, does not come to man from cows or bovines, but from man himself afflicted with the infectious form of pulmonary tuberculosis. Not long ago this was held to be a self-evident proposition. Just now this simple truth is somewhat obscured by the strange medley of opinions that crowd around the academic thesis of the relation of tubercle bacilli of the human type to those of the bovine type. The origin of pulmonary consumption is surely to be traced to the tubercle bacilli of the human type. Scientific investigations in Germany, America, England, France, Norway, and elsewhere have shown that in no single well-attested instance (*cave* Beitzke) has the tubercle bacillus of the bovine type ever been found in cases of ordinary consumption. In a singular manner the epidemiological evidence, which Kitasato has supplied to us from Japan, offers further convincing proof that consumption in man has its origin in man, spreads from man to man, and perpetuates itself through its microbe in the body of man. If this was not the proposition which Professor Koch threw like a bombshell among the members of the Tuberculosis Congress in London in 1902, it was something extremely like it; and Koch's opinion, that in man the essential cause and source of tuberculosis is pulmonary tuberculosis in man, is far nearer the truth than many will allow. A few words upon the prevalence of tuberculosis in Japan may be surely opportune, seeing that no other country can hope to furnish such striking

Man is Essential Source of Infection.

evidence. In Japan, as Kitasato tells us, the native cattle do not suffer from tuberculosis, and neither children nor adults drink milk to any extent, and yet in Japan there is just as much intestinal and meningeal tuberculosis among children, and just as much consumption and other forms of tuberculosis in adults, as in the chief countries of Europe. Even intestinal tuberculosis and miliary tuberculosis are as common in Japan as in England. These plain facts prove that in Japan tuberculosis in human beings has nothing to do with bovine tuberculosis, and on this and other evidence we may rest assured that at any rate pulmonary tuberculosis in England has nothing to do with tuberculosis in bovines. As I wrote more than ten years ago, "If tuberculosis were eradicated from cattle, the prevalence of tuberculosis among adult human beings would not be perceptibly affected." In Japan the antecedent is absent, and the consequent is not affected. Logically, the consequent is independent of the antecedent. No doubt, in young children localised forms of tuberculosis, especially tuberculous disease of the mesenteric glands, and sometimes even of the cervical glands, and occasionally generalised and fatal tuberculosis, may arise from infection conveyed by the milk of tuberculous cows, but these forms of tuberculosis are absolutely non-infectious and have nothing to do with the spread of pulmonary tuberculosis in man. The epidemiological evidence from Japan immensely strengthens the force of Koch's contention at the London Congress. Upon the vexed question of the relation of tubercle bacilli of the human type to those of the bovine type and *vice versa*, one may at least say that it is impossible to overrate the difficulties that beset the way of the investigator; but he who reads and inwardly digests the infinitely careful and strikingly convincing array of experimental details marshalled by Kossel, Weber, and Heuss, as the experts of the German Commission (*Tuberculose-Arbeiten*, Heft 1, u. 3), must admit that two types of mammalian tubercle bacilli exist. Most experts recognise this fact, but until this difference is even more widely accepted, most of us must remain mere partisans; and, after having carefully studied most of the contributions upon this stupendous question, I, for my part, unhesitatingly declare my allegiance to

the practical view, emphasised by Koch, that the prevention of pulmonary tuberculosis demands that we should vigorously deal with the sources of infection existing, *not in animals*, but *in man* affected with the open form of pulmonary tuberculosis, who scatters the seeds of consumption broadcast in his neighbourhood, day after day, week after week, year after year, unless he has learnt the hygienic method of coughing and the simple means of rendering his expectoration quite harmless to others. We must not confine our attention simply to the massive expectoration, which can be seen and avoided, but must remember also that even if the sufferer attends to the instructions with respect to this massive expectoration, there is also danger in the cough. The patient cannot help coughing, and with each cough he may be projecting into the air a regular fusillade of invisible germ-laden particles, which may float in the air and thence reach the lungs of the healthy, and there sow the seeds of a fresh case of the disease. The prevention of consumption is a far greater question than its treatment, and is and should be the business of everybody, The treatment must be left to the physician and his patient. Prevention concerns everyone; and it may be wise in the public interest even to administer without reference to the physician or patient. It is true that almost one-third of all the deaths that occur between the ages of 20 and 35 is due to tuberculosis, so that all those who love and cherish their sons and daughters in the bloom of youth and maturity should take a personal interest in preventing the awful sacrifice of life that must otherwise be laid to the charge of this murderous and pitiless disease. Calculated on a mere £ *s. d.* basis—not a very satisfactory basis—the money loss to England must be represented by millions of pounds every year. Every adult, whether as head of a household, as a mother, as a wage-earner, is worth on an average £50 a year to the State. On this low standard 50,000 deaths represents a loss of several millions. Nor can one forget not merely the energy wasted in the individual who suffers, but that also cast away by others, sometimes for years, upon one who is hopelessly doomed. But apart from this £ *s. d.* aspect, the ruthless destruction of fifty thousand lives every

year by a disease which can be, and should be, prevented, and can often be arrested by special methods too long despised, constitutes a problem of the gravest national importance. The problem is a stupendous one; it is very difficult, very complex and many-sided; but I believe we are now within striking distance of the enemy.

Nature of Consumption.

Consumption, as we see it, is rarely a simple process of uncomplicated tuberculosis. It is a process, variable in its nature, in its clinical manifestations, in its morbid anatomy, and in its course; and these variations depend upon the virulence and dose of the tubercle bacilli, the state of the tissue, and also on the number and character of the organisms that complicate the simple process due to the action of the tubercle bacilli. These variations, in their manifold clinical phases, have been analysed by the more exact methods of scientific inquiry, and, in consequence, method and clearness replace the confusion and vagueness that characterised our hitherto conception of the disease. Such an analysis, even for clinical purposes, may exhaust the methods of bacteriological research. More than a quarter of a century ago Koch's monumental work defined once for all time the nature of tuberculosis with marvellous accuracy and completeness. More than this; ever since the searching genius of Koch has been devoted to this subject, our knowledge has increased by leaps and bounds. Let us remember that in his earliest experiments, Koch set his mind to discover not merely the specific cause of tuberculosis, but rather a scientific method of treatment, based upon a careful study of the nature and effects of the specific cause isolated in a test tube. Practically all the sound and useful knowledge we possess with regard to the nature, the diagnosis, the prognosis, and even the treatment of this disease has but one source. It is to Koch that we owe those principles upon which we base our earnest advocacy of preventive measures. To him we owe the safe and sure means of diagnosis in all cases of doubtful tuberculosis. To him we owe the scientific methods by which we can at will analyse the complications of tuberculosis and also surely judge the course of the disease. To him

we shall not look—and we have not looked—in vain for scientific methods of treatment. Not only is his transcendent work in the past some guarantee of the truth of his scientific observations, but, as a disciple of this man of genius, I am able to assert, after nearly fifteen years of careful and laborious observations, that the main facts of Koch's original communication upon tuberculin are absolutely sound. Until the truth about tuberculin is recognised, the problem of the successful treatment of pulmonary tuberculosis must remain in abeyance. As Koch then maintained, (1) *tuberculin is an invaluable* and *indispensable* agent in the diagnosis of early tuberculosis, especially pulmonary tuberculosis; and, as I have seen in many scores of cases, (2) the early stage of *pulmonary tuberculosis can be cured with certainty by means of tuberculin.* It is my object to bring forward evidence that, in my judgment, establishes beyond reasonable doubt the truth of these two profoundly important propositions.

PART II

THE PROBLEM IN THE LABORATORY

PART II

THE PROBLEM IN THE LABORATORY

CONSUMPTION has been studied by physicians since the days of Hippocrates and Galen, but, although Lænnec and Virchow devoted their best energies to a description of the effects observed in the lungs and elsewhere, their elaborate descriptions threw no light upon the nature of this disease, and it is well known to most of us that Virchow and his followers were not the first to recognise that Koch, by finding life in the tuberculous masses, solved the mystery that was beyond the ken of the eye or the microscope. Sceptical they remained long after Professor Koch had convinced the man in the street. Villemin had convinced himself and others, notably Cohnheim, by experiments on animals that tuberculosis was the result of infection and therefore, in accordance with the germ theory of disease, was due to the action of a living micro-organism; but Koch, by his epoch-making labours, removed every vestige of doubt, and finally convinced even the sceptics by discovering the very cause of this widespread disease and exhibiting it "in splendid isolation" as a pure culture, consisting of millions of tiny organisms, upon the surface of solidified blood-serum. In his masterpiece, "Die Etiologie der Tuberculose," Professor Koch brought before our very eyes the tiny, slender, living germ called the tubercle bacillus, and convinced the scientific world, very much against its will, that this tiny germ "was the one and "only cause of tuberculosis, and without it there could be no "tuberculosis." Since then volumes have been written about

Experimental Investigations of Koch. Nature of Infection.

this tiny organism, which was first recognised through its property of being deeply stained by certain aniline dyes (methylene blue, gentian violet, fuchsin) and retaining this stain even after the addition of strong acids. The tubercle bacillus is acid-fast. Since that day other acid-fast bacilli have been discovered, some of them (Moeller's Timothee- and Gras-bacillus) being distant relatives of the tubercle bacillus.

More important still, experiments in the laboratory have shown that tuberculosis in different species of animals may be produced by several varieties of tubercle bacilli, each resembling one another in certain features, but differing from one another also in morphological, cultural, and pathogenetic characters. The analysis of these varieties has not yet reached finality, and it still needs the illuminating influence of more and more perfect knowledge leading unto truth before the chasm that divides the experts will diminish and disappear—to the credit of science and the benefit of mankind. It is generally acknowledged that there are two main kinds of tubercle bacilli—the mammalian, occurring in mammals, and the avian, occurring in birds; while the weight of evidence at present favours the view that there are two distinct types of the mammalian tubercle bacilli—the human and the bovine.

Source and Channels of Infection.

The human type occurs under natural conditions in man and some other animals. The bovine type occurs in bovines and in swine. Nevertheless, the bovine type may also occur in man, almost exclusively in children under the age of ten, who have been fed on unboiled cows' milk. *There is no evidence at all in favour of the view that the bovine type of bacillus has anything to do with pulmonary tuberculosis*, a point upon which too great emphasis cannot be laid—and it needs a great stretch of the imagination to suppose that the bovine type readily assumes the human type in the human body. In children the bovine type retains all the characters of this type; in adults even the bovine type may in rare instances occur in tissues, but does not cause a progressive form of tuberculosis. Naturally, if adults drink unboiled milk from a tuberculous cow, the bovine type of bacilli may mechanically reach the human tissues, especially in the vicinity of the intestinal tract,

but these bacilli seem to have no power of causing progressive tuberculosis. Adult human tissues enjoy natural immunity against this type of bacillus. There is no cause for wonder in this when we know that a certain breed of cattle, the Japanese, enjoys natural immunity against any and every kind of tubercle bacilli.

The question at issue is no mere academic question; it is a question of cardinal importance, because, if much tuberculosis in man has its source of infection in animals, prophylaxis demands the full recognition of this source. In children, certainly, there is an obvious risk of infection—though it is very difficult to measure the extent and intensity of the risk—and the prevention of disease from this source can be secured by either eradicating tuberculous disease from dairy cattle or by simply exposing the milk to a temperature sufficient to kill the bacilli (pasteurisation). If, however, as Behring persistently asserts, and would have us believe, the common form of tuberculosis in man—pulmonary tuberculosis—is also due to infection during infancy by means of unboiled cows' milk, not only does prophylaxis demand the precaution of treating all milk so as to kill the bacilli, but we are, according to Behring[1] in his truculent attack upon Flügge, relieved of the otherwise irksome necessity of compelling consumptives to be careful with their sputum and circumspect in their mode of coughing. Fortunately, very few authorities take His Excellency Prof. von Behring at his own valuation upon this phase of the question, for it seems to have settled down into the solid knowedge of the world that at any rate the commonest and most deadly form of tuberculosis—pulmonary tuberculosis—is due to infection conveyed from man suffering from the infectious form of this disease to his healthy relative, friend, or neighbour.

Infection requires certain conditions; it may be potential or actual. Actual infection requires that the infectious material should be carried from the sick to the healthy and should therein find suitable conditions for its growth and multiplication. So long as the infectious material is alive it is potential; but outside of living tissue it cannot multiply

[1] V. Behring recommends "Formalin-milk."

under natural conditions, and may even lose its virulence and its life. Preventive measures aim at preventing potential from becoming actual infection.

From all these considerations the great outstanding fact is that the commonest form of tuberculosis in man, pulmonary tuberculosis, the source of infection *par excellence* for man, has nothing to do with the tuberculosis of cattle.

It is in the laboratory that we seek to learn whether in pulmonary tuberculosis the initial site of infection is in the air-passages or elsewhere—in the small blood vessels of the lungs (Aufrecht, Orth); in the tonsils (faucial, lingual, pharyngeal), whence *via* the cervical lymphatics and lymphatic glands the tubercle bacilli reach the pleura and then lodge in the apex of the lung; in the mouth (Westenhoeffer); or in the loose meshes of the lymphatic system of the bowels of the infant (v. Behring). V. Behring tells us in poetic language that "consumption is the last verse of the song which is first muttered in the cradle." Most of us, for very many reasons, cling to Koch's original view, which Coruet especially, and more recently Pfeiffer and Friedenberger, and Flügge have sought to establish by experiments on animals, that in pulmonary tuberculosis the initial lesion is either in the air-passages or in the lymphatics that drain these areas. After the striking and convincing inhalation experiments, recorded by Kossel (*Tuberculose-Arbeiten*, Heft 1, u. 3), of the German Imperial Health Office, it will need a great deal of special pleading to dethrone or discredit this simple and reasonable explanation. Tubercle bacilli are tiny, solid particles, not always single, but often in clumps or masses, which may be readily carried hither and thither by themselves or in coarser particles of dust. As solid bodies they are subject to the same physical laws as other particles in the air, some of which, such as coal-dust, we can easily track to the lungs by the air-passages and through the lungs by the lymph spaces to distant parts and distant lymphatic glands. The deeply pigmented, coal-laden lymph spaces and lymphatic glands are the invariable accompaniment of life in cities. Moreover, anyone who had the good fortune to see for

Site of Infection.

himself the beautiful specimens exhibited by Birch-Hirschfeld at the Berlin Congress on tuberculosis knows that primary tuberculous lesions do occur on the surface of the air-passages, especially in those very branches that lead to that part of the lung—the posterior section of the apex—where tuberculous lesions so frequently begin. Further, we may track the course of other micro-organisms in the lungs, especially of streptococci, pneumococci, influenza bacilli, and staphylococci. These organisms enter the air-passages and may, like tubercle bacilli, be localised in the apices of the lung (Neufeld), though more often they infect the mucous membrane of the bronchial tubes widely and may thence be conveyed to the tissue-spaces and to the nearest lymphatic glands, and even finally enter the general circulation. All these migrations of tiny organisms and coal-dust may be literally followed with the eye by means of the microscope, and it seems a work of supererogation to look for any other explanation when this natural explanation actually stares us in the face. Ribbert's and Oehlecker's recent investigations clearly point the same way. Although in animals, especially rabbits and guinea pigs, tubercle bacilli may stray by circuitous routes from the intestinal tract or the mesenteric glands even to the thoracic glands (Oehlecker), the enormous preponderance of bronchial tuberculosis even in children (Barthet and Rilliet, Still, J. W. Carr, &c.) is most readily explained by infection *via* the air-passages (Koch, Cornet, Ribbert, Oehlecker, Pfeiffer), rather than *via* the alimentary canal (v. Behring, Orth). To children, especially very young children, crawling on the ground in the homes of the poor, where there may be a consumptive who knows nothing of the hygienic way of coughing or of disposing of his expectoration, the risk of infection through tubercle bacilli of the human type is *daily imminent*. One knows that these tubercle bacilli have been found in the finger-nails of these doomed infants.

Thus in the laboratory we may learn much of the nature of the infective agent and also of the usual channels of infection. Along its track, too, the bacillus often leaves behind its own peculiar mark or lesion, sometimes here, sometimes there, in

Latency of Primary Focus.

the air-passages (Birch-Hirschfeld), in the tissue spaces (Virchow), in the lung tissue (Virchow, Birch-Hirschfeld), or in the lymphatic glands. In any or all of these structures the slow decay (caseation) caused by the bacillus may go on, especially in the lymphatic glands (Petruschky), even in the lungs and bronchial tubes (Birch-Hirschfeld), *without arousing the suspicion of the individual, the parent, or even his physician.* This is truly one of the worst features of this treacherous little enemy. It may lie in ambush for months and years, and in this *latent stage of the disease* the health, the strength, and the appearance may suffer but little. The disease may become thus quiescent at any time; it may relapse after months or years; it may reach a serious stage, involving the greater part of a lobe, and end in perfect restoration of health. In spite of treatment of any and every kind, the disease may never relent, closely pursuing the victim till death. Without any treatment the disease may come to a stand-still, and never again disturb the health. The most affectionate regard for logic may not restrain us from attributing success to our own small efforts and failures to the vagaries of the disease. Our memories seize fast hold of successes; we leave others to record our failures, or leave them unrecorded. Let us adopt what measures we may, more or less failure is the rule, and yet we are apt to place a high value on many of our methods. As a rule the individual seeks advice when the disease is in the second stage, often in a late second stage; sometimes the disease reaches almost the third stage under the very eye of the physician before its real nature is recognised. Pulmonary tuberculosis is so dangerous and deadly because, in the majority of cases, it is well advanced before its presence is discovered.

Variable and Insidious Course.

It is very common for a physician to fail to recognise the disease in the first stage, and it is not uncommon also for a physician to discover disease in the first stage when the lungs are quite healthy. (See cases diagnosed by medical men as early phthisis, which I proved by a severe test to be free from tuberculosis.) This is a very important chapter. These facts must be

Detection of Disease.

stated and recognised before the inestimable value of tuberculin as a diagnostic agent can be fully appreciated. Further, nowadays, sanatoria are the fashion. All authorities upon sanatorium methods agree that it is only in the first stage that really satisfactory results can be obtained. Turban, whose authority no one disputes, tells us that of 419 cases, only 11 came for treatment within a month of the appearance of the first symptoms, 103 within six months, and the majority, 305, only after six months elapsed. In the first group (11) permanent results were obtained in 72·7 per cent., in the second group (103) in 67 per cent., and in the majority (305) only in 40·2 per cent.

After Koch's discovery in 1882, the test of tests was the demonstration of tubercle bacilli in the expectoration by certain stains. For many years this test dominated everything. Progress in knowledge has deprived even this test of most of its value. Valuable in certain cases in which the physical signs are absent or misleading, and in mild cases of mixed or concurrent infection, notably in influenza and pneumococcal and streptococcal infections, the discovery of tubercle bacilli in sputum by certain stains too often fails when it is most wanted. It fails as a test in early diagnosis, because the presence of tubercle bacilli in the sputum is itself a sign that disintegration of tissue has occurred. The great lesson of the last decade has been the paramount importance of detecting pulmonary tuberculosis in the first stage—before tubercle bacilli escape into the air-passages by the disintegration of the superficial tubercular lesion—while, in fact, the tuberculous formation is still closed, or, in other words, shut off from the external world, represented by the air-passages.

Moreover, Koch and his pupils have taught us that, in the great majority of cases, the tubercle bacilli play a relatively subordinate part in the severer forms characterised by fever, sweating, and wasting. By the light of Koch's methods, as applied by Kitasato and Pfeiffer, we have been guided to a clearer and truer understanding of the variable picture that pulmonary tuberculosis presents to us

Analysis of Processes. Mixed Infection.

in its manifold stages and phases. I admit that among English authorities there is still a strong tendency to look askance at, if not to disregard and belittle, the modern view of mixed infection, secondary infection, and concurrent infection. The term "mixed" infection—so full of light and meaning—was first used by Koch's distinguished colleagues, Ehrlich and Brieger, in 1882, in describing a case of typhoid fever complicated with malignant œdema, but Koch himself was the first to recognise the condition in pulmonary tuberculosis. In his own monumental work upon the Etiology of Tuberculosis, one reads thus: "The combination of bacilli and micrococci as they appear in this disease belongs to the mixed infections, the existence of which appears to be by no means rare." In this case the infection was rather a concurrent than a mixed infection. There was an invasion of cocci from an ulcer in the mouth, in a case of miliary tuberculosis, running a rapidly fatal course.

The role of infection independent of the tubercle bacillus—sometimes mixed, sometimes secondary, sometimes concurrent—occurring in any stage of tuberculosis of the lungs, may defy exact measurement by clinical methods; but nowadays it is hardly possible to overrate its importance when bacteriological methods are called to our assistance. Diagnosis, prognosis, and even treatment demand a careful differentiation of the elements constituting these other infections. A careful analysis of these elements by recent methods is an important, perhaps the most instructive, basis of classification. Physical signs may mislead us in diagnosis and prognosis; symptoms may help us to distinguish the acute, the sub-acute, and the chronic—the treatment varying accordingly—but a discrimination of cases by the resources of bacteriological science gives us a basis at once definite and trustworthy. The full meaning and importance of mixed infection did not develop until the genius of Koch, which had guided and inspired the investigations that culminated in the production of the diphtheria antitoxin, proposed a new method of treating tuberculosis on an equally scientific basis. The treatment of tuberculosis with tuberculin is specific. It is of no avail except for tuberculous diseases, and, if other

complicating conditions exist—if other micro-organisms are associated with tubercle bacilli in the morbid process—tuberculin may fail absolutely to assist or modify the morbid process; indeed, tuberculin may then do harm, and perhaps even hasten a fatal issue. I fear that many men have learnt to their cost and at their patients' expense, that tuberculin may be powerful for harm and powerless for good when the chief cause of the symptoms is not the tuberculous process at all, but the various infectious processes that may be quite beyond the physician's range of vision. Tuberculin treatment aims at a progressive process of active immunisation, radically different from the passive process, in which antitoxic serum is supplied ready made. The passive process of immunisation may be exploited with impunity and under any conditions; active immunisation, on the other hand, makes a larger demand upon the energy of the tissue cells, and requires that the cells and tissues should be in a relatively healthy state. In the presence of various micro-organisms, such as streptococci, the energy of the cells may be so depressed that active immunisation by means of tuberculin cannot be expected. Tuberculin treatment, therefore, has limits unknown in the antitoxic treatment of diphtheria. The failure of tuberculin in 1891 was due to a disregard of the limitations and restrictions laid down by Koch himself, and to a general ignorance of the *rôle* of mixed infection in pulmonary tuberculosis. It was not that Koch's announcement was premature; it was rather that medical men, by a bitter experience, discovered themselves exploiting a remedy without having had the training essential to success. Failure in their hands was a foregone conclusion, and tuberculin as a remedy for tuberculosis had a mere mockery of a trial. The key to successful treatment with tuberculin is a proper selection of cases by means of bacteriological methods, or, these failing, by a judicious use of tuberculin itself as a diagnostic agent. There is, indeed, no royal road to success in the treatment of pulmonary tuberculosis, either by specific, rational, or other methods. Each case requires skill, judgment, experience, and, in no small degree, patience; and after all the best and most conscientious efforts may be

baffled by the intervention of conditions that can be neither foreseen nor prevented. Indispensable to success is a thorough knowledge of the variable, complex, and treacherous condition, mixed infection. This mixed infection may even parade itself in the garb and trappings of health. As a rule, fever is the danger-signal, and strongly suggests a secondary or mixed infection. Yet, in spite of the existence of fever, there may be no mixed infection, as in acute tuberculosis and in those rare forms of acute gelatinous caseous pneumonia which Fraenkel and Troje have so well described. On the other hand, fever may be absent, and yet mixed infection, with all its evils and dangers, may be imminent. Nothing short of a careful and thorough examination of the sputum by the methods of Pfeiffer and Kitasato gives us unimpeachable evidence of the presence or absence of a mixed infection. Such an examination should and must go hand in hand with specific treatment. Other methods of treatment may be independent of this fundamental knowledge, but in tuberculin treatment an analysis of the infectious processes at work is indispensable. It is impossible to judge fairly the merits of tuberculin treatment, if we disregard the element of mixed infection. Mixed infection made shipwreck of the tuberculin treatment in 1891. By a fatal fortuitous coincidence, tuberculin as a remedy for tuberculosis was tried when influenza raged in Europe as a violent epidemic. Altogether apart from tuberculin treatment, the high rate of mortality from pulmonary tuberculosis in the influenza years proves that influenza was a very dangerous mixed infection in tuberculous disease of the lungs. If, perchance, tuberculin had been used, the tuberculin was blamed. Tuberculin has been condemned mainly, if not entirely, on the investigations of fifteen years ago. It was not known then that, although tuberculin might fail, and even do harm when influenza or other infection complicated the tuberculous process, tuberculin might do nothing but good when such complications were absent. I was fortunate enough to use tuberculin in some cases before influenza invaded Australia, and my limited experience seemed to prove the undoubted value of tuberculin if it were used under conditions prescribed by Koch him-

self. But, while tuberculin can and does act favourably upon the uncomplicated tuberculous process, it may do nothing but harm in the presence of a mixed infection. The phases and stages of pulmonary tuberculosis largely depend upon the manifold combinations embraced in mixed infection. Let me say, too, that certain infections of the lungs, that have nothing to do with the tubercle bacilli, may simulate tuberculosis of the lungs. This is especially true of influenza, and of infections due to streptococci or pneumonococci (Neufeld). Such cases cannot be benefited by tuberculin. In my own experience, two striking cases are worth recording: I made a *post-mortem* examination on a man who died of some obscure septic process. The sole obvious lesion was a small abscess of the size of a hazel nut in the apex of the lung. The pus yielded in the smear a streptococcus, and in the growth on agar a pure culture of streptococcus longissimus. From the spleen, which was swollen and engorged, I obtained a pure culture of the same organism. Is there room for doubt that the primary infection attacked the lung? Even more pertinent is the second case, of a brilliant young medical man who fell a victim to his excessive loyalty to duty. He consulted me once, and once only, for chest trouble with cough and expectoration. For some time he had had irregular fever—sometimes as high as 101° or 102°. Physical signs were practically negative; at most, there were some suspicious auscultatory signs at the right apex. The sputum contained no tubercle bacilli, but I obtained therefrom an absolutely pure culture of streptococcus longus. I was inclined to the view that he was suffering from a mixed infection of tubercle bacilli and streptococci. Cases enough are recorded in which the tubercle bacilli do not get into the expectoration in the early stages of these mixed infections. As the fever persisted, an injection of tuberculin could not be given. If it had been given, it might have been held responsible for the sad issue of the case. In spite of my advice, this man continued at his post for some weeks during as bad a bout of cold rainy weather as I have ever known in Sydney. Then he went to the country for some weeks. He returned to town, and was carefully attended by his regular

physician. I saw him but once, a month before he went to the country. He developed septic endocarditis, and died in the course of some months. Surely this was a case of primary infection of the lungs with streptococci, which spreads thence to the endocardium by the pulmonary veins. Is it not possible that in many cases of cryptogenetic septic endocarditis the infective agents enter the system by the air-passages? I know of no record of a similar history and nature. Thus, septic infection of the lungs may simulate pulmonary tuberculosis. Far more frequently these infections supervene upon the primary tuberculous process. Streptococci, diplococci, staphylococci, and micrococci are ever at hand to complicate the tuberculous process. Influenza bacilli and bacilli pyocyanei may also complicate. I have isolated pure cultures of the influenza bacilli from many cases of pulmonary tuberculosis. In the year 1905, on the other hand, I examined no less than nine cases of so-called influenza, and in eight cases obtained a pure cultivation of a streptococcus, in one case of ædiplococcus lanceolatus. There had been, according to my investigations, no true influenza in this epidemic. One may call it influenza from its clinical characters ; but the frequent complication of true croupous pneumonia is fair clinical proof that influenza bacilli had not complicated the process. Thus, acute, and especially chronic, bronchitis may be due to concurrent infection of the bronchial passages, causing apical catarrh, and this infection of the bronchial mucous membrane may end in a mixed infection. In mixed infection the streptococci are intimately and inseparably associated with tubercle bacilli in the tuberculous lesion, as Spengler and others have shown, both by special examination of the sputum and by examination of the diseased tissues, in fatal cases. The tuberculous lesion has, of course, disintegrated, and tubercle bacilli are also found in the sputum. A mixed infection may cause only mild symptoms, especially if there is much fibrosis of the lungs, or may suddenly cause the signs and symptoms of acute phthisis with bronchopneumonic exudation. This is important, and teaches a further lesson. Streptococci and pneumococci may attack a tuberculous as well as a healthy lung ; but in both, the fatal disease is the

same. Is it right merely because tubercle bacilli are found in the sputum, to make the diagnosis of acute tuberculosis? This is often done, especially during streptococcal and pneumonococcal epidemics. *Those who see only tubercle bacilli in the sputum may err egregiously in diagnosis.* Hæmorrhage may complicate a pure tuberculosis or a mixed infection, and may thus be either the first sign of a simple uncomplicated tuberculosis of the lungs in which the hæmorrhage may be even a favourable sign or the early symptom of a form of acute phthisis due to mixed infection, and running a rapidly fatal course. As a rule, the supervention of the mixed infection leads to wasting, loss of appetite, and sweating, accompanied by fever of varying degrees. The clinical picture changes when a mixed infection is superadded to the chronic tuberculous process. Then, the tuberculous lesion may extend rapidly and soften ; tubercle bacilli may be numerous or scanty in the sputum ; the expectoration is greatly increased, and swarms with other and various organisms. Kitasato's or Pfeiffer's method readily evolves order out of the chaos of this flora. In 1891 the effects of the mixed infection of various sorts—influenzal, streptococcal, diplococcal—were ascribed to tuberculin. The lesions to which Virchow specially directed attention in fatal cases that had been treated with tuberculin were actually the lesions that commonly occur in severe secondary infections when no tuberculin has been used. The simple, oft-recorded fact, that death occurred within a few days or weeks of the injections of tuberculin in 1891, proves incontestably that tuberculin was used in hopeless and totally unsuitable cases. Thus the brilliant success of the few was placed in total eclipse by the disasters that followed the rash and indiscriminate use of the remedy by the many. Ten years of patient work and careful observation have hardly succeeded in rehabilitating tuberculin among the useful remedies for tuberculosis. Professor v. Schroetter told the Congress in London in so many words, that the work upon tuberculin as a remedy in tuberculosis would have to be done all over again. Those who use bacteriological methods to their full extent will be convinced in less than a year of the supreme importance of mixed

infections. Those who disregard the lessons taught plainly by the best bacteriologists will hardly listen to any arguments of mine. "They have Moses and the prophets: let them hear them."

The Lesions in the Lungs and their Physical Signs.

In the laboratory also we may store up a knowledge of the endless variety presented by the tuberculous lesions in every stage of the disease, and nothing but a fairly accurate knowledge of these varieties can aid us in forming even rough and often inaccurate ideas of the possible conditions that may be compatible with the physical signs we may be skilful enough to record on our charts. Without a knowledge of these lesions, some causing consolidation, some softening, and some excavation of the lung tissue, our physical signs may mostly mislead us. The physician attempts with the aid of physical signs to picture for himself the gross changes produced in the tissues of the lungs and bronchial tubes by disease in its progressive stages. These physical signs, which we feel and see and hear, and not the conditions, which we can at best guess at, constitute the essential basis of all systems of classifying the stages of pulmonary tuberculosis; and unless we have some clear idea of the changes which tubercle bacilli and tuberculin, without and with the co-operation of various other organisms, may produce in the lungs, pleura, and bronchial tubes, we may go hopelessly astray in our diagnosis by means of physical signs. Mere degrees in the change of shape, in the impairment of movement, in vocal fremitus and resonance, in the change in the percussion note, in the altered quality of the breath sounds, and in the varying characters of adventitious sounds, signify much or little according to the skill with which we can translate the language of physical signs into the objective conditions in the underlying tissues themselves. Physical signs are always hard to interpret, and leave plenty of scope for erroneous conjecture.

Here, too, we may specially urge that it is far more important *not to misread the signs of early disease than it is to make the most accurate guesses as to the size and site and contents of cavities.* He who never misses an early stage, and never finds an early stage in a sound lung, should be the life-

long friend of many grateful patients; he who gives the most surprisingly accurate forecast upon the nature and extent of a cavity will in most cases soon be able to test the absolute accuracy of his diagnosis *post mortem*, and neither he nor anyone else can help the patient much. In the former case, if the physician knows the right thing and has the sense and courage to do it, he saves his patient's life. In the latter, his satisfaction begins and ends at the *post-mortem* table. Accordingly, it is well to seize every opportunity of studying the earlier phases of tuberculous disease of the lungs, which are found so often in those who die of other than tuberculous disease. A study of the early stage of this disease in the laboratory can alone teach us the great lesson that early lesions may hardly cause any physical signs of their own, and may cause no special symptoms. Most important is it to bear in mind this *latent* or early stage, because in a very short time, and sometimes suddenly without any warning, a severe hæmorrhage may supervene, or a severe mixed infection or both these catastrophes may occur, and the patient may rapidly pass into a parlous state. I have seen several instances in young girls in whom the latent stage was overlooked or ignored by the physician, and the quiescent form has ruptured into the air-passages; and the tubercle bacilli, thus liberated from the localised lesion, have been scattered through the air-passages by aspiration and caused acute tuberculous broncho-pneumonia, with all the symptoms of galloping consumption. Such forms may at first be called pneumonia broncho-pneumonia, acute influenza, or acute bronchitis. Nothing but the early recognition of these latent forms and prompt and effective treatment by means of tuberculin can prevent these awful catastrophes. The symptoms may be trifling and may abate and pass away, deceiving everybody, even the physician; there may be respite for a month, a year or much longer, but as a rule recurrence takes place sooner or later, and the last state of the patient is far worse than the first, for there is progressive destruction of the lungs. The disease may progress in the lung, as I have witnessed many times, while the general condition of the patient—his weight, his appetite, and his strength—improve. Often this improve-

ment is due to the abatement of the mixed infection. I have seen laryngeal complications arise, when both the patient and physician were well pleased because the patient gained 10 lbs.

But while Koch was studying the ways of this enemy of mankind in the laboratory, he was at the same time searching for weapons wherewith to fight and overcome this deadly enemy when once it had invaded the human body. These weapons, discovered by the genius of Koch, are laboratory products, called tuberculin. In my humble opinion, we hardly begin to study the clinical aspects and vagaries of pulmonary tuberculosis until we use tuberculin for diagnostic purposes, as a curative agent, and also as a means of checking our results after treatment. In treatment, tuberculin is eminently specific, and though its use may be compatible with mixed infections, it in no way affects these complications; but in the presence of mixed infection the cells and tissues may not be able to make use of the tuberculin in their own defence. My own experience may have been peculiarly lucky, for in no single instance in the space of sixteen years have I seen tuberculin do any real harm if used with skill and judgment in cases of mixed infections, and I have seen remarkably happy results from its use in apparently hopeless cases of pulmonary tuberculosis—even in cases of severe mixed infections.

The Products of the Tubercle Bacillus—Forms of Tuberculin.

Tuberculin is the general name for the toxic products of the tubercle bacillus grown upon the artificial media of the laboratory. It is not a serum. Its character and effects vary with the mode of preparation. The toxic products of the bacillus are the soluble, diffusible exo-toxins, and the endotoxins of the solid cell-body. Old tuberculin is merely a glycerine extract of virulent bacilli—either as a simple filtrate (T.O.A., T. original altes.) or reduced by evaporation to $\frac{1}{10}$ its original bulk (T.A.). This latter preparation is the old tuberculin of Koch, invaluable and often indispensable in diagnosis, and of great value as a remedy in pulmonary tuberculosis.

Besides this old tuberculin, Koch has introduced other preparations. New tuberculin (T.R.) is made by finely pulver-

ising masses of virulent tubercle bacilli, adding water and glycerine to the powdered mass, and then centrifugalising. The upper part (T.O.) is said to contain much of the fever-producing stuff of T.A. The lower part (T.R.) contains most of the endotoxins in the cell-bodies. T.R. or new tuberculin is a very valuable preparation, especially in the early stages of pulmonary tuberculosis. It is milder in character, less apt to cause severe reactions, but not so powerful as an immunising substance. Later still Koch dispensed with centrifugalisation and recommended the glycerine extract of the pulverised tubercle bacilli without any filtration—T.R. emulsion (T.E.)—as a more powerful immunising agent than any of the previous preparations.

Still other preparations of tuberculin are made in a similar way from bovine tubercle bacilli:

P.T.O. corresponding to T.O.A.
P.T. „ „ T.A.
and P.T. emulsion to T.R. emulsion.

Spengler speaks in high terms of P.T.O. In my opinion P.T. is a far more valuable preparation than P.T.O., and is particularly valuable in advanced forms of disease, and in cases in which over-sensitiveness is easily induced and persists. Often, indeed, a preliminary course with P.T.O. is valuable. In severe and advanced forms of pulmonary tuberculosis, P.T., with or without a previous course with P.T.O., is far less apt to cause much fever or much local reaction. I would certainly say that P.T. has far more powerful immunising properties than any other form of tuberculin. Koch's and Behring's work in immunising cattle by means of living bacilli of the human type first suggested to me that the converse of this principle might also be true, and subsequently Carl Spengler's work encouraged me. I have the same high opinion of P.T. that Spengler has of P.T.O., and in my experience the difference in effects of P.T. and P.T.O. respectively are so very marked that it is hard to believe that P.T. is merely P.T.O. evaporated by slow heat to $\frac{1}{10}$ its bulk. I should be very sorry to give any patient $\frac{1}{10}$ of a gramme of P.T. after a dose of 1 gramme of P.T.O. which had not had the slightest effect. After a dose of 1 gramme of P.T.O., $\frac{1}{50}$ of a gramme of P.T. may cause

a well marked reaction. Other forms of tuberculin may be made from tubercle bacilli of the avian type, and even from the saprophytic forms of tubercle bacilli which Moeller discovered in certain grasses and in dung.

These laboratory products constitute the chief and most powerful weapons we possess in dealing with the stupendous problem of the cure and prevention of tuberculosis in all its forms. By means of tuberculin properly used we can detect pulmonary tuberculosis and other forms of tuberculosis in the very earliest stage ; by means of tuberculin we can cure with certainty the early stage of pulmonary tuberculosis, as Koch told us seventeen years ago ; and by means of tuberculin we can measure the effect and permanence of a course of tuberculin treatment and learn whether the disease has been eradicated or is merely quiescent. In the latter case a further course of tuberculin, perhaps in larger doses or in a different form, may be needed. If the disease has been eradicated I am of opinion that the patient having been thus actively immunised is less liable to a second infection or re-infection than a normal person. It is hard to conceive what more is required of a remedy. In diagnosis, in treatment, and in prognosis tuberculin is the remedy *par excellence* in pulmonary tuberculosis.

The immense value of tuberculin as a diagnostic agent in animals has long since been admitted, and I can testify to its value in human beings (*see* tuberculin in diagnosis). In animals also the curative value of tuberculin in its various forms has been proved by Kitasato, Pfuhl, Spengler, and Beck. These experiments show that even the old tuberculin, which was hastily condemned not only as useless but harmful, has a decided effect in checking the progress of tuberculous formations, and in causing in them those very changes that are recognised as positive signs of the regression of the morbid process. In guinea-pigs, inoculated beneath the skin with virulent tubercle bacilli, an ulcer, that persists till death, forms in the second week at the seat of inoculation, and the animal dies in eight to eleven weeks. In such guinea-pigs treated with tuberculin soon after inoculation, the ulcer healed, and the animals lived five, six, seven, and nine months after infection, though the control animals died in six, seven, eight, and eleven

weeks (Kitasato). Kitasato writes, "I was able to convince "myself with the greatest certainty that even tuberculosis of "the lungs had been influenced in a decidedly favourable way "by tuberculin treatment." He adds, "In the case of those "guinea-pigs, five in number, that are still alive seven months "after infection, the inoculation ulcer is at length completely "healed, the lymphatic glands, previously swollen, are no "longer to be felt, the animals have steadily increased in "weight, and they have in all respects the appearance of "healthy animals." More interesting still, these animals were again inoculated by Koch. The second inoculation ran the following course:—Some days after inoculation the tissue round the seat of inoculation was swollen and indurated. In a week this indurated part was detached by spontaneous necrosis, and exposed a fresh-looking granulating surface. This ulcer was entirely healed within twelve days from the date of inoculation—an event that never occurs under ordinary conditions. The second infection had no further effect. These results were repeated by Spengler.

But the use of tuberculin as a remedy for tuberculosis has gone beyond the laboratory stage, and many observers (notably Rembold, Krause, Petruschky, Spengler, Turban, Camac Wilkinson, and later Moeller) not only speak well of the remedy, but do not record a single case of generalised tuberculosis during or after its use. I have long since come to the conclusion that so far from causing generalisation, tuberculin is the remedy *par excellence* to prevent generalisation. Rembold began as a sceptic, as did Moeller, but their own results soon converted them. Rembold set a good example in waiting for six years (I waited longer still) before he collected his evidence upon the value of the old tuberculin. Out of 70 cases 27 were cases of mixed infection. Of the remaining 43, 16 were in an early stage, 15 in the intermediate stage, and 12 in an advanced stage. Of the 12 advanced cases, two were improved at the end of six years; the rest died. Of the 15 in the intermediate stage, eight were alive at the end of six years; all of them improved; one case was cured. Of the 16 in the early stage, one died four years after treatment; the rest were alive, three greatly improved,

12 permanently healed—75 *per cent. cured.* In these cases judgment was withheld till six years had elapsed. What sanatorium, without tuberculin as a remedy, can show such a record? And yet I can show a still better record, because I have been able to use many forms of tuberculin, and I have not hesitated to use large doses. Krause, Petruschky, Turban, Spengler, Moeller, and others are able to furnish equally satisfactory results in pulmonary tuberculosis with tuberculin as a remedy.

Before we conclude this chapter on the "Problem in the Laboratory," we must needs describe the effects of tuberculin as studied in animals in the laboratory and in man.

"*Humanum est errare*" is not an adage that pretends to make a virtue of mistakes, and least of all should the physician, who is true to himself, manifest a facile tendency to condone mistakes; but, nevertheless, the history of medicine, as we see it in text-books, which sadly need revision, even before they reach the affectionate hands of students, in large part consists, if not of errors, at least of a correction of errors. In medicine mental inertia, tolerant of error, has been a frequent stumbling-block in the way of progress, and in medicine, as in politics, too often mental myopia restricts the vision to but one side—and that a small side—of a many-sided and complex problem. Where can we find, though we search the whole domain of medical literature, a better illustration of this inept mental complacency than in the common attitude of mind towards the many and difficult problems that have been created for us by the parasite which is the essential cause of tuberculosis. Years and centuries of labour, and piles of volumes devoted to the problem of the nature and origin of tuberculosis, helped us not at all until the genius of Robert Koch—just a quarter of a century ago—obtained his cultures of the tubercle bacillus, in splendid isolation, active and virulent, upon the surface of his newly-found medium of solid serum. Ever since that memorable gathering of the Physiological Society of Berlin on March 24, 1882, when Koch announced his brilliant discovery heralding a new era in the history of medicine, our knowledge has

The Nature and Significance of the Tuberculin Reaction.

increased by leaps and bounds; and even now in all that concerns tuberculosis—its origin, its nature, its prevention, its treatment—we do well to look for light and guidance to the genius who has devoted the best years of his life to the study of this protean disease. Let us never forget that the discovery of the specific bacillus of tuberculosis was the first step towards those fruitful investigations of Behring, Kitasato and Wernicke, which culminated in the discovery of a specific remedy for diphtheria and tetanus. Having discovered the specific cause of tuberculosis, Koch next set himself to search for an agent that would destroy the parasites in the living tissues. Years of incessant toil, during which he exhausted the vegetable and mineral kingdom in his quest, led him to the disappointing conclusion that all disinfectants that would kill the bacillus in tissues would kill the tissues also. Nevertheless, there are many amongst us who still claim that creasote *et hoc genus omne* are remedies for pulmonary tuberculosis. Proceeding then on absolutely new lines, Koch made the experiments upon guinea pigs with graduated doses of dead bacilli which have revolutionised our ideas upon infection and immunity. Therefrom he evolved the principle of specific treatment, discovering that the disease can be not only prevented, but cured, through the medium of its own cause. On November 13, 1890, Professor Koch gave an account of a substance which was able to "render animals immune against tubercle bacilli and to bring "to a standstill the tuberculous process in animals." This new remedy for tuberculosis, afterwards named tuberculin, consisted of a glycerine extract of tubercle bacilli, evaporated to one-tenth of its bulk in a water bath, and then filtered through a Chamberland filter. The method used by Koch in preparing tuberculin is in vogue at the present time. The effects of tuberculin were chiefly studied in guinea-pigs. Strange to say, guinea-pigs, that are so susceptible to living tubercle bacilli, are extraordinarily tolerant of tuberculin. Even a dose of 2 c.cm. of tuberculin, injected subcutaneously, has very little effect upon a healthy guinea-pig; but if the guinea-pig be already tuberculous, in the fifth week of the disease or later, 2 c.cm. will kill the guinea-pig with certainty

in 6–30 hours, and produces, invariably, in the tuberculous lesions, especially in the tuberculous foci in the liver, an absolutely typical picture. This in itself was a great discovery. Never before had any substance been known which acted at a distance with certainty upon diseased tissue, and diseased tissue only. Prof. Koch then gave himself a subcutaneous injection of ·25 c.cm., and found to his cost that man is far more sensitive to tuberculin than guinea-pigs. Calculated according to body weight, man is 1,500 times as sensitive as guinea-pigs, and yet in man there is far greater resistance to infection with living tubercle bacilli. In man tuberculous lesions tend to be localised—they may remain localised and inert for months, years, even a lifetime. Koch thus describes the effect of a dose of ·25 c.cm. of old tuberculin:—"Three or "four hours after the injection, pains in the limbs, lassitude, "inclination to cough, difficulty of breathing, which rapidly "increased; in five hours an unusually violent rigor, which "lasted an hour; at the same time nausea and vomiting and a "rise of temperature to 103·4; in 12 hours all the symptoms "abated, the temperature began to fall, and reached the normal "level next day. The pains in the limbs and lassitude "persisted for some days, during which also the site of the "injection remained painful and reddened." Many experiments showed that in man the limit of tolerance to tuberculin is reached with a dose of ·01 c.cm. (ten milligrammes). Such a dose has very little effect upon healthy persons, or even upon sick persons, provided there be no tuberculosis. But, if the individual be tuberculous, ·01 will produce a severe general and local reaction. "The "general reaction takes the form of an attack of fever, "ushered in with a rigor. The temperature rises to 103° "to 106°. The other symptoms are headache, pains in "the limbs, great lassitude, loss of appetite, nausea, vomiting; "sometimes even slight jaundice and a measly rash on the "chest and neck. (Herpes is also common.) A severe "reaction sets in early, in four or five, even in three hours after "the injection, and lasts 12–15 hours or longer. In milder "reactions the fever is delayed, occurring in 8–12 hours." The fever then may be of short duration, and, therefore,

unless the temperature be taken every two hours, it may be overlooked.

The local reaction is best studied in lupus patches, in which the tuberculin picks out the diseased tissues and spares entirely the healthy and healed parts. Koch says: "Even "before the rigor begins, the lupus tissue swells and becomes "red. The swelling and redness last while there is fever and "may reach such a degree that the lupus tissue becomes "brownish red in places, and necroses. This inflamed area is "surrounded by a whitish zone nearly $\frac{1}{4}$ inch in width, which "in its turn is surrounded by a broad, deeply red border. "When the fever passes away, the swelling gradually subsides "and disappears in 2–3 days. The lupus foci are then covered "with crusts of dried serum, which separate in two or three "weeks, leaving behind a smooth red surface. The pheno- "mena thus described have invariably developed with a dose "of ·01 c.cm., if any tuberculous focus exists in the body, and "I therefore believe that tuberculin in future will become an "indispensable diagnostic agent. One will thereby be in a "position to diagnose doubtful cases of incipient phthisis even "when on account of the absence of tubercle bacilli or elastic "fibres in the sputum, or through the absence of physical signs, "it is impossible to come to any conclusion concerning the "nature of the trouble. Thus, also, it will be easy to recog- "nise tuberculosis of glands, of bone, doubtful lesions of the "skin, &c. In apparently arrested cases of tuberculosis of "the lungs and joints it will be possible to decide whether the "morbid process has actually come to an end and whether or "not isolated foci exist, from which the disease might light "up afresh, as a fire from a spark that glows beneath the "lifeless ashes."

Surely we may here marvel at the precision which guides his clinical instinct to exploit this rich fruit of his laboratory experiments. May I presume to add that even the reaction that occurs at the site of injection often gives a fairly safe clue to the existence of tuberculosis in the body? In healthy persons there is no change at the site of the injection: in tuberculous persons there is much swelling, redness, œdema and

firm infiltration, sometimes very extensive; and often immediately around the puncture there is further a well-defined deeply red, raised and even slightly vesiculated area about an inch in diameter. This appearance develops best in the forearm, and therefore I make a practice of giving test doses in the forearm. Roux has lately suggested a per-cutaneous test with tuberculin, much after the manner which Spengler recommends in the specific treatment of those highly sensitive to tuberculin. Still later a 1 per cent. watery solution of dried tuberculin applied to the conjunctiva has been recommended as a test for tuberculosis by Calmette. This reaction, general and local, to tuberculin, is specific, and occurs only when tuberculous lesions exist in the body. Certainly, reactions, even with fever, have been recorded in cases of actinomycosis, syphilis, leprosy, cancer, etc., but in none of these recorded cases has tuberculosis been *excluded with certainty;* and it is easier for one who has seen many hundreds of such reactions with tuberculin to imagine that tuberculosis was somewhere present than to explain how the reaction *can* be other than specific. Certainly, in large enough doses, as professor Koch proved in his own body, tuberculin is a *poison*, capable, like other bacterial products, of causing fever and severe symptoms; but, besides these ordinary bacterial elements, tuberculin contains a substance which has a selective action upon tuberculous tissue, and thereby causes the specific reaction. In doses of ·005 or ·01 c.cm. tuberculin does not cause fever in a healthy person; but in far smaller doses (·0001 c.cm.) tuberculin acts upon tuberculous tissue in such a way as to release an active fever-producing substance. The action upon the tuberculous tissue is essentially inflammatory. An enormous literature has grown around this subject from which we learn that, under the influence of tuberculin, the tuberculous tissue becomes drenched with tissue juices and infiltrated with the amœboid cells of the blood. This inflammatory œdema may lead to softening of the tuberculous tissue. Healthy tissue is rigidly respected. Now, while it is easy enough to describe accurately this extraordinary effect of tuberculin upon tuberculous tissue, as yet its intimate nature is a hidden mystery. Long ago von Babes

maintained that the local reaction was the direct result of the addition of tuberculin to the tuberculin already existing in the tuberculous focus—a simple process of summation or cumulation. As yet we do not understand what is termed the cumulative action of digitalis ; so that this explanation is not satisfactory. The reaction seems rather to be akin to the phenomenon of over-sensitiveness to specific poisons, which was first described by von Behring in his experiments with diphtheria and tetanus toxins. He found that animals immunised by the isopathic method (by graduated doses of the specific toxins) exhibit in the tissues obnoxious to the toxins a marked over-sensitiveness, which he termed histogenic over-sensitiveness, although the hæmatogenic immunity, due to the presence of abundant antitoxin in the blood, protects the over-sensitive tissues against the toxin. This view is certainly strengthened by the paradox reaction which Kretz discovered in immunised animals further treated with an equilibrated mixture of toxin and antitoxin. A normal animal does not react at all to an equilibrated mixture of toxin and antitoxin —there is no production of antitoxin. On the other hand, an animal already treated with toxins and thereby immunised, responds to the same dose of equilibrated mixture with an abundant production of antitoxin. This discovery of Kretz has been utilised for the production of antitoxins of high potency. Further evidence in the same direction is yielded by Behring's observation that the total amount of toxin necessary to kill a guinea-pig diminishes if the toxin be given in divided doses. Hyper-sensitiveness is established the more readily by small doses. In like manner Loewenstein and Rappoport, working under Moeller, claim that in a tuberculous man or animal the specific toxins cause the tissues to respond by this hyper-sensitiveness ; and they argue also that this very over-sensitiveness to the specific poisons is an indispensable step towards artificial immunity. All who have used tuberculin have very early in their experience become very familiar with the fact that at the outset there is this over-sensitiveness to tuberculin ; and even, if you like to call it so, a temporary fall of immunity. Wright has called this the negative phase, but in my opinion has misinterpreted it and

been misled by it. He considers that this over-sensitiveness is the result of overdoses or too frequent doses. Nevertheless, if small doses be repeated the period of this over-sensitiveness may be prolonged indefinitely. On the other hand, if the doses be gradually increased the over-sensitiveness rapidly disappears. Moreover, if the degree of immunity may be expressed in terms of the highest dose of tuberculin that can be given without causing a reaction, one cannot expect any high degree of immunity from the method of small doses at long intervals, which is the fashion of those who follow Wright. With my experience to guide me, I cannot conceive that small doses of tuberculin not increased to any degree can produce immunity in either early or late stages of pulmonary tuberculosis: indeed, both in early and more advanced forms of tuberculosis, when attempts have been made for various reasons, on account of heart disease, or pregnancy, or hæmorrhage, to carry out the system of small doses, or by gradual increases, the result has been just the reverse of what was expected. Either no immunity has been produced or a prolonged phase of over-sensitiveness appears, which postpones the development of immunity. The natural course of pulmonary tuberculosis, in which no doubt infinitesimal doses of toxin or other immunising factors enter the blood, without any advantage to the organism, supplies the experiment under natural conditions, which sooner or later ends in failure. The stage of hyper-sensitiveness is prolonged and the disease gets the upper hand. By increasing the doses and overcoming the over-sensitiveness, according to Koch's original plan, immunity is produced, as I have proved actually in scores of cases of pulmonary tuberculosis in every stage. I do not exaggerate when I say that I have seen this over-sensitiveness again and again, and I can hardly remember any cases, except advanced and hopeless cases, in which I was not able to beat down the over-sensitiveness, and, by proceeding to large doses, have not only caused all symptoms to disappear, but months and years afterwards I have tested these cases under most exacting conditions, and have obtained no reaction at all. What Wright and those who follow his plan can do remains to be proved. If his method also succeeds, I can but say that still another mystery has

been discovered in the vagaries exhibited by tuberculous tissue towards small and large doses of tuberculin. Moreover, as I have never yet failed by the use of large doses in early cases of pulmonary tuberculosis to bring the patient to that condition, which would at least be described as a case of complete arrest of the disease, I am not disposed to adopt a method which, after all, has not been shown to be even of value in tuberculous disease of the lungs.

On the other hand, for diagnostic purposes there is reason in exploiting this phenomenon of over-sensitiveness. Even Koch's own method recognises this tendency to increased sensitiveness towards repeated doses of the same strength. He gives the following directions :—" In suitable cases, a dose " of ·1—1 mg. of old tuberculin is given. In weakly persons " one begins with ·1 mg. ; in robust persons, if the lesions are " not extensive, one may begin with 1 mg. If no reaction " occurs after the first injection, one gives double the dose in " two days. If a rise of temperature, even of only 1 deg. F. " (·5 deg. C.) occur, then the second dose should be repeated " as soon as the temperature has become normal. It often " happens that this second dose causes a much greater reaction " than the previous dose of the same strength. This is a very " characteristic effect of tuberculin, and may be looked upon " as unmistakable evidence of the presence of tuberculosis. If " 2 mg. fails to cause a reaction, the dose must be increased " till the limit of 10 mg. is reached." Koch elsewhere says that two doses of 10 mg. given at proper intervals and causing no reaction prove the absence of tuberculosis. Other authorities are content with a maximum dose of 5 mg., others with 3 mg. Loewenstein brought forward at the Tuberculosis Congress in Paris a special method which aims at producing over-sensitiveness by repeated small doses of the same amount. In children he gives $\frac{1}{20}$ of a mg. every three days for four doses, so that the test occupies ten days or so—an objection in itself. In adults $\frac{1}{10}$ of a mg. is injected four times, if no reaction occurs, at intervals of three days. Loewenstein claims that a reaction is produced in the great majority of cases. If no reaction occurs he proceeds to 2 mg.—6 mg. and 10 mg. I can see no special advantage in this method. Three injections are

nearly always needed, and by virtue of the over-sensitiveness caused by this repetition of the same dose, temperatures of 102 deg. and 103 deg. may occur. There is no virtue in this over-sensitiveness; and if the tuberculin test is merely the prelude to tuberculin treatment, this method of repeated small doses is distinctly objectionable because the over-sensitiveness delays the process of immunity. I, therefore, follow Koch's method. The symptoms of the reaction are in proportion to the fever. A temperature of 99·5 may be sufficient if there are general symptoms of mild degree and a local reaction; and especially if there is pronounced œdema and infiltration at the site of injection. If there be merely a slight rise of temperature—not reaching 100 deg. F., I repeat the dose as Koch recommends. If the temperature rises to 101 deg. F. the symptoms are more marked, headache, drowsiness, lassitude, loss of appetite, and often pains in the limbs. If the temperature be 102 deg. to 103 deg. F., the symptoms usually set in earlier, last longer, and are more severe. Severe headache, great lassitude, drowsiness, nausea, even vomiting. Herpes is also common in severe reactions, and then also there may be delirium, albuminuria and even hæmaturia. Even a temperature of 104 deg. F. may follow such a small dose as 1 mg. Always, too, symptoms related to the tuberculous part show themselves. In pulmonary tuberculosis, cough, increase of expectoration, in which for the first time tubercle bacilli may be found, difficulty of breathing, and pains in the chest. Rales, too, may be heard for the first time. Swelling and tenderness develop in superficial glands, and, no doubt, in those also beyond the range of vision, if they contain tuberculous deposits. It has long been known that tuberculin causes an increase of the eosinophiles and a temporary decrease of the neutrophiles.

Wassermann's Experiments and Views.

An account of the tuberculin reaction, which is, after all, a reaction of immunity, would not be complete without a reference to the most recent work of Wassermann and Bruck upon the nature of this reaction; although a somewhat severe criticism by Morgenroth and Rabinowitch clearly indicates some faulty links in the chain of evidence which is brought

forward by these authors to prove that the tuberculin reaction depends upon the action and interaction of tuberculin as an antigen, and its antibody, styled by Wassermann and Bruck antituberculin, existing in the tuberculous focus. This latest development in the biological and biochemical diagnosis of disease, by means of the products of immunity, has been applied with success to such widely different diseases as syphilis, gonorrhœa, leprosy, and small-pox. The biological principles which lie at the foundation of such functions of specific sera as precipitation and agglutination, of hæmolysis and of certain processes of immunity, have been applied by Wassermann and Bruck to the phenomenon of the tuberculin reaction.

The essence of their method of sero-diagnosis consists in the proof of the presence of specific antibodies—by the absorption and disappearance of the complement. Specially prepared sera acquire specific functions by means of specific products, such as immune bodies (factors in the mechanism of immunity), precipitins, agglutinins, all of them of the nature of antibodies. These specific antibodies, mixed in definite proportions with the antigens, are able to appropriate the complement and thus render it inert.

The test of the disappearance of the complement is by means of the simple process of hæmolysis. The phenomenon of hæmolysis takes place in the presence of the complement, when the blood of an animal—*e.g.*, a sheep—is added to the serum of a rabbit, which has been injected from time to time with sheep's blood. If the complement be appropriated or in other ways rendered inert, hæmolysis does not occur. Accordingly the failure of hæmolysis indicates the appropriation or disappearance of the complement. This method of investigation depends upon a variation at will of the three main factors concerned in the well-known reaction of Pfeiffer. In this reaction the three necessary factors are the cholera vibrios, as antigens; the immune body as antibody or amboceptor, and the complement which normally exists in the organism. If any one factor fails, the specific bactericidal effect fails also. Accordingly, if in an experiment two of the factors are known to be present and the specific

phenomenon (in this case dissolution of the vibrios) takes place, it is certain that the three factors were present. Thus, cholera vibrios may be at once distinguished from other vibrios by this specific reaction of immunity. It was later discovered that not only bacteria but bacterial products or extracts may also act as antigens. Making use of this development in serodiagnosis, it has been demonstrated by mixing together in certain proportions and under certain physical conditions of temperature, etc., the syphilitic tissues of man or infected monkey, a complement and suspected tissue, such as the lumbar fluid of a general paralytic or tabetic individual, that the complement has been absorbed, proving the presence of a substance in the lumbar fluid which has a specific affinity for the syphilitic antigen. This experiment yields further proof of syphilis as an etiological factor in the causation of general paralysis and tabes dorsalis. Wassermann and Bruck have exploited this method of biological diagnosis in order to solve the mystery of the tuberculin reaction. The tuberculin reaction is a reaction of immunity, and the scientific analysis of this reaction is of importance, since it should surely help us the better to use the various forms of tuberculin in the treatment of tuberculosis.

In the tuberculin reaction two obvious facts present themselves. In the first place there is some selective action exhibited by the tuberculous tissue, in consequence of which tuberculin is drawn from the blood and concentrated in the tuberculous focus. The other event is the inflammatory softening of the tuberculous tissues. The tuberculous tissue attracts the tuberculin atom by atom to itself. Even so small a dose as 1 mg. injected at a distance profoundly affects the lupus patch. In a man of average weight the tuberculin is diluted in the blood at least 5,000,000 times. Tuberculin so diluted injected into the lupus patch would have no effect. Accordingly the tuberculin does not produce this profound effect by merely bathing the tissues in this diluted state. Specific affinity between the tuberculous tissues and tuberculin can alone explain the striking fact. Such specific affinities are nowadays known to be constant phases of cell activities, and, if we translate these affinities into the language of

Ehrlich's masterly hypothesis, we should say that the substance tuberculin is withdrawn from the blood and concentrated in the tuberculous tissue by means of a haptophore atom group—a specific antibody, which Wassermann has called antituberculin.

This antituberculin in lupus tissue attracts and binds to itself the tuberculin circulating in the blood. We need not look far afield for the *raison d'être* of antituberculin in the tuberculous tissue. Long since Ehrlich, in order to explain the tuberculin reaction, suggested the existence of three distinct zones in a tuberculous mass. The innermost zone lying nearest the bacilli is saturated with tuberculin and mostly dying or dead—the outermost zone is healthy and unaffected —while the intermediate zone is damaged by the bacterial products and over-sensitive. When tuberculin is present in the blood it reaches the tuberculous focus and, affecting neither the central nor marginal zone, concentrates its energy upon the middle zone, where it causes the reaction through the agency of the affected but still active cell elements. In this reaction probably the young cells of infiltration and the emigrated white blood cells play the chief *rôle*, and in this tissue the antibody antituberculin is formed by living cell elements in response to the irritation caused by the tuberculin secreted from the active tubercle bacilli. The antibody is ready to seize with avidity its antigen, the tuberculin, artificially introduced into the blood. Of course, a complement is necessary, but the complement is likely to be ready at hand in the presence of active leucocytes to complete this biochemical process. Thus far the process is relatively simple and the explanation is not far fetched or unreasonable. Tuberculin must exist in and around the tuberculous focus, either through the living or dead bacilli *in situ*; and in accordance with the general principles of local immunity may well form its antibody, probably in the intermediate zone, in harmony with Ehrlich's explanation of the local reaction. Now, while it may be relatively easy thus to explain the first great fact disclosed in the tuberculin reaction, it is far from easy to explain thereby the severe changes that affect tuberculous tissue.

These preliminary considerations will pave the way to our appreciation of the delicate and difficult experiments which have been devised by Wassermann and Bruck to explain the nature of the tuberculin reaction. These experiments were designed to prove (1) the existence of tuberculin in tuberculous tissue, and (2) the presence of an antibody, antituberculin, as the reaction product of the antigen tuberculin ; and Wassermann and Bruck claim to have proved both points by means of biological experiments, based upon the fixation of the complement. In their original investigations Gengou and Bordet showed that if emulsions of bacteria and the homologous inactivated immune body (or amboceptor) were mixed together, under certain conditions, and the complement added, the complement was fixed or appropriated by the antigens and antibodies. The proof of the fixation of the complement was furnished by the failure of hæmolysis, even when red corpuscles and their specific hæmolytic amboceptors are added to the mixture. There is no free complement to complete the process of hæmolysis. The complement has disappeared. Wassermann and Bruck went a step further, and showed that if soluble extracts of bacteria were used instead of bacteria, the same fixation of complement took place. Similar fixation of the complement occurs in the phenomena of specific agglutination and precipitation. It was by a converse process that Neisser and Sachs used this method to demonstrate the presence, not of immune bodies or amboceptors, but rather of antigens. The great advantage of this modification is that in such diseases as syphilis, in which the specific organism has not been isolated, the presence of specific antibodies can be demonstrated in the serum of monkeys infected with syphilis and the presence of antigens in syphilitic organs. In this way also syphilitic antibodies have been discovered in the lumbar fluid of those suffering from general paralysis and tabes dorsalis.

In mixtures, supposed to contain antigens and antibodies, under proper conditions the disappearance of the complement proves that both these elements are present. Only in the presence of the two elements specifically related can the

complement disappear. Thus, by varying the experiment at will, it is possible to prove the presence of antigens on the one hand or antibodies on the other. By this method Wassermann and Bruck have proved the presence of anti-tuberculin and tuberculin in tuberculous organs. The method consists in mixing together in definite quantities the antigen and antibody, and then adding the complement (guinea-pig's serum). This mixture is placed in an incubator at 37 deg. for an hour. In that time the complement is fixed. That the complement has been fixed is proved by adding to the mixture the elements necessary for hæmolysis, *minus* the complement, that is to say (1) the inactivated serum of rabbit which has been previously treated with the blood of a sheep, and (2) the blood of the sheep (5 per cent.). The complement used is guinea-pig's serum, fresh and normal. The prepared serum of the rabbit is the hæmolytic amboceptor, and the sheep's blood is the antigen. After the complement (guinea-pig's serum) has been added to the original mixture of the antigen and antibody to be examined and the whole has been incubated for an hour, inactivated rabbit's serum and the sheep's blood are added.

If, then, hæmolysis fails, both antigen and antibody must have been present, because the complement is no longer able to cause hæmolysis. Nothing but antigens and antibodies specifically related to one another can appropriate the complement. Accordingly, if it is known that antibodies are present in the mixture, failure of hæmolysis proves the presence of antigens, and, in like manner, if antigens are known to be present, the failure of hæmolysis proves the presence of antibodies. If hæmolysis occurs, the homologous body is absent. We are now in a position to understand the method of proving the presence of tuberculin (antigen) and its antibody (antituberculin) in tuberculous tissues. In this way the tuberculous organs and tissues and the serum of man, cattle, and guinea-pigs have been examined by Wassermann and Bruck. Extensive control experiments must also be carried out.

Organs were removed with aseptic precautions and ground up in a mortar in salt solution containing 5 per cent. carbolic

acid, then well shaken, and finally centrifugalised so as to get rid of solid particles. Next measured quantities of these extracts were added to diminished amounts of Old T. and T.R. Em. Fresh normal guinea-pig's serum was added as complement, and the mixture placed in an incubator at 37 deg. for an hour. At the end of that time the hæmolytic amboceptor for the sheep's blood and, lastly, the sheep's blood itself was added. The mixture was again placed in incubator for an hour at 37 deg., and then kept in ice over night. Table proves the presence of antituberculin in tuberculous organs.

PROOF OF ANTITUBERCULIN IN TUBERCULOUS ORGANS.

(*a*) Tuberculous Organs of Man.

1. Tuberculous Lung.

	Extract of T Lung.	Old T.	T.R. Em.	Guinea-pig's Serum Complement.	Hæmolytic Amboceptor.	Sheep's Blood.	Result.
Experiments	·1	·05		·1	·002	5 %	Arrest of hæmolysis (Antituberculin in Lung).
	·1	·01		·1	·002	,,	Complete hæmolysis.
	·1		·05	·1	·002	,,	Arrest.
	·1		·01	·1	·002	,,	Complete hæmolysis.
Controls	·1	—	—	·1	·002	5 %	Complete hæmolysis.
	—	·05	—	·1	·002	,,	
	—	—	·05	·1	·002	,,	
	—	—		·1	·002	,,	
	—	—		·1	—	,,	0
	—	—		—	·002	,,	0
	—	—		—	—	,,	0

Again—

Lung extract + ·1 T.—arrest
+ ·05 T.—arrest
+ + ·1 T.R.Em.—arrest
− ·1 T.—hæmolysis
− ·1 T.R.Em.—hæmolysis.

Explanatory.—T.R.Em. = Emulsion of dead tubercle bacilli. Old T. = Old tuberculin. T. = Tuberculous.

Numerous experiments of a similar kind show that *antituberculin* is present in the tuberculous tissues both in man and animals. On the other hand, in thirteen cases of pulmonary tuberculosis not treated with tuberculin, no antituberculin was found in the blood—although in animals sometimes antituberculin was found in the serum. Similarly it was proved that *tuberculin* was present in the tuberculous formations.

Wassermann next examined the blood of some persons treated with tuberculin and found antituberculin in the serum of persons who had tuberculous disease, though antituberculin failed to show its presence in the serum, when there were no tuberculous lesions. Hence we may conclude that injections of tuberculin in tuberculous persons cause specific antibodies to appear in the blood. Professor Ostertag found the same result after injecting tuberculin into tuberculous cows. Moreover, on mixing together tuberculin and antituberculin, no trace of precipitins appears, showing that the formation of antibodies is independent of precipitins.

We may deduce certain general conclusions from these observations. In the first place antibodies against the products of the tubercle bacilli are present in the tuberculous tissues, but not in the blood, and the tuberculin seeks out its antibody in the tuberculous tissues and thus produces the local reaction of tuberculin. The local reaction, ending in severe inflammation and softening of the tuberculous tissue, seems to depend upon this union of the antigen, antibody, and complement ; but there are some difficulties in the way of attributing to the combination of antigens, antibodies, and complement the severe inflammatory changes of a local tuberculin reaction. Wassermann and Bruck take for granted that, whenever by means of amboceptors complements are concentrated and are active, albuminous substances in the organism are dissolved by a process of digestion. Hence they attribute the softening of the tubercular tissue to the concentration of complements brought about by the specific union of the products of the tubercle bacilli with their antibodies in the tuberculous areas, and there is some reason for supposing that not merely free enzymes in the blood, but

substances formed from the leucocytes which crowd around the tuberculous centre, play an important *rôle* in this reaction. Arneth's great work upon the effect of doses of tuberculin upon the leucocytes of the blood in those suffering from pulmonary tuberculosis proves that many leucocytes are destroyed by doses of tuberculin, and in their death liberate substances which play an important part in the production of immunity in pulmonary tuberculosis.

Again, in tuberculous persons who have been treated with tuberculin, antituberculin is formed in the blood, and, combining with the tuberculin given in treatment, protects the tissues from the action of tuberculin, and reactions fail. Nevertheless, the presence of antituberculin in the blood probably shows that the tuberculous process is not yet healed. In other cases treated with tuberculin, the doses of tuberculin do not cause antituberculin to appear in the blood. Such cases, which react to tuberculin as in health, may be considered to have been cured. It is very likely that these methods for determining the presence of antituberculin in the serum may influence one's methods of specific treatment. As yet we are only upon the threshold of inquiry. But in the light of their investigations, we may think with Wassermann that the specific reaction of tuberculous tissue shows itself because the products of the tubercle bacilli are drawn into the tissues by their antibodies, and by this process the vitality of the cells of the organism in digesting the tissue is concentrated at this particular place.

The value of this method has also been shown in cases of acute miliary tuberculosis. By the use of a tubercle bacillus immune serum and the serum of the patient, products of tubercle bacilli were discovered; and similarly, by using Old T. instead of the immune serum, antituberculin was found in the serum. This analysis of processes at work in the tuberculous foci further indicates the specific nature of the reaction. It teaches us clearly that a tuberculin reaction can only occur when the tuberculin penetrates the tuberculous foci, and there combines with its antibody. Accordingly, a tuberculin reaction cannot occur if there is no tuberculous lesion in the body, and fails to occur if an old tuberculous lesion has healed, or is so securely

surrounded by fibrous tissue that no active process is possible. I have been thoroughly convinced for many years that one hardly begins to understand the nature of tuberculosis until one has studied it by means of tuberculin. In diagnosis we cannot do without tuberculin, in treatment we mostly fail unless we use tuberculin, and even in prognosis tuberculin guides us aright in practice. I assert boldly that no one has a right to speak of arrest of pulmonary tuberculosis unless he has shown that the patient no longer reacts to tuberculin. Tuberculin controls our knowledge in a most exact way. Even with the best of treatment—of course I mean specific treatment—it is our bounden duty to control our results year by year by means of tuberculin. I am not satisfied with any treatment unless a year afterwards the patient is not only apparently well but fails to react to tuberculin. That is, in my judgment, the best guide to the degree of improvement and the value of treatment—*immunity against the toxic product of the specific cause;* and the larger the dose tolerated, the higher the degree of immunity. Using tuberculin in this way to check and control one's work, one may expect to succeed where others fail. Early diagnosis, direct and radical treatment by specific methods, and a trustworthy prognosis all depend upon the proper use of those various forms of tuberculin which have been freely offered to us in the cause of suffering humanity by the supreme authority in the world upon tuberculosis—Dr. Koch.

PART III

THE PROBLEM IN MAN, AS THE MOST FREQUENT HOST OF THE PARASITE.

PART III

THE PROBLEM IN MAN, AS THE MOST FREQUENT HOST OF THE PARASITE

It is obvious that the bacillus is not the disease ; nor is even its presence in the human body equivalent to tuberculous disease. Tubercle bacilli may of course be found on any surfaces, exposed to the outside world, in the skin and beneath the nails, notably in children crawling about a dirty floor, in the nose (Strauss, 40 per cent.), throat, air-passages, alimentary tract, etc., without causing disease. Possibly in some men, as in some animals, *e.g.* Japanese cattle, there is natural immunity, or no disposition to the disease. If it be so, we have no means of discovering it or proving it. In most human beings there is a disposition to tuberculosis, if the tubercle bacillus but gets its own opportunity. This disposition depends upon an affinity between certain tissues or organs and the bacillus or its poison, or both, which leads to certain changes, called the reaction, in the tissues. This disposition may be general or local. In young children the disposition is general, and acute generalised tuberculosis is the result. In adults especially, in pulmonary tuberculosis, there is a local disposition, a tendency in the lungs, especially at the apex, to undergo these changes or this reaction in the presence of tubercle bacilli. The form of this reaction, its rapidity and progress, depend upon many factors, not merely on the tubercle bacilli, their number and virulence, and other organisms, but also on the site of the lesion and the state of the tissues invaded. No doubt, too, the age of the tissue (Behring), metabolic energy (Mitulescu), and even states of

the circulation (Bier) and blood (anæmia, chlorosis) are subsidiary, and may be determining factors in the infective process. If the affinity exists, there is irritation in response to the toxins of the tubercle bacilli, with the formation of new cells and positive chemiotaxis (leucocytal accumulation), followed by slow necrosis, due to endotoxins, called caseation. Inflammatory reaction is an essential part of the process, varying in degree, due sometimes to the action of tubercle bacilli, but sometimes to mixed infection, or even to both infective processes. Caseation is the characteristic lesion of tuberculosis; and softening of this caseous area may have disastrous results, even when the lesion is small. It may lead to hæmorrhage; it may convert a closed into an open form of tuberculosis, with all its risks; it may lead to fresh foci of tuberculous disease at a distance from the primary focus; it specially favours mixed or secondary infection. I repeat that acute galloping consumption in young girls, acute tuberculous pneumonia, or broncho-pneumonia may thus arise, and may be called acute influenza, or acute bronchitis, until the careless or misguided physician rectifies his mistaken diagnosis. Worst of all, the caseous area may burst into a blood vessel, especially into a vein, and lead to acute miliary tuberculosis, resembling in its features the acute miliary tuberculosis produced in animals at will by an intravenous injection of small doses of tubercle bacilli.

These dreadful catastrophes will and must occur, until we learn to detect the disease in the earliest stage—a consummation devoutly to be wished, but quite beyond our reach unless in the interest of the unfortunate host we use tuberculin without stint and without hesitation for diagnostic purposes. It will at all times be hard to save the host's life in such cases, because not only the specialist in lung diseases must be ever on the alert, but also other specialists, and, above all, the general practitioner, who must be keenly alive to the danger of overlooking an early case of pulmonary tuberculosis. Pulmonary tuberculosis may masquerade in various guises and deceive the very elect. I have known several instances in which throat specialists have been content with the superficial diagnosis of laryngeal catarrh, or a hæmorrhage from the

throat; gynæcologists have made the diagnosis of amenorrhœa and chlorosis; and other physicians have diagnosed gastritis, gastric ulcer, rheumatism, hysteria, neurasthenia, when a few small doses of tuberculin would have revealed pulmonary tuberculosis in an early stage to be the efficient cause of *these symptoms of pulmonary disease in distant organs.* We learn often enough that throat symptoms, blood disturbances, digestive troubles, ovarian troubles, and nervous symptoms, *without any lung symptoms at all, may give the first clue* to the presence of pulmonary disease. It may be added that tuberculin alone can settle the diagnosis in early tuberculosis of the genito-urinary tract, of bones, of joints, and even of serous membranes, including the meninges. Once this great fact is realised and remembered at the psychological moment by every practitioner of medicine, and tuberculin is called into requisition to help the diagnosis, nothing but good can come to the infected host and honour to the physician, who is well worthy of his hire. In such cases which have come under my own notice, and there have been many, the rapid and extraordinary improvement of the patient under tuberculin would convince the most hardened sceptic. The bold use of tuberculin in these very suspicious cases settles the question whether there is or is not any tuberculosis, and one cannot express in words the enormous gain to the host of this insidious parasite if the disease should be discovered early and treated promptly with tuberculin.

Every individual has an interest in tuberculosis, especially pulmonary tuberculosis, because the disease is so common and so capricious in its incidence that infection may be the lot of any one. A knowledge of the nature of infection may give us the power of warding off infection, and a knowledge of the nature and early symptoms of the disease may in some cases prevent that postponement of the visit to the physician which is one of the chief causes of disaster. After all, under our present system, it is the unfortunate victim who first discovers that there is something wrong, and he is wise to seek medical advice early. Too often the visit to the physician is postponed, and the disease is allowed to drift into the second or even the third stage. In my experience

it is quite rare for the patient, in the early stage of the disease, to seek medical advice of his own free will. I have a large array of early cases simply because I am convinced that infection occurs from person to person, and acting upon this conviction I proceed to test all cases that have been exposed to infection, especially in the family circle. The symptoms in the early stage are altogether too trifling to suggest serious disease of the lungs. Moreover, when disease in an early stage may or may not exist, even if the physician be consulted, two mistakes are possible. The physician may discover tuberculous disease (in his own opinion) when the lungs are free from disease, or he may fail to discover tuberculous trouble when it certainly exists. In my own experience I have discovered tuberculous disease of the lungs in scores of cases in which the disease has been overlooked by other physicians (or more often surgeons), and, on the other hand, I have proved it not to exist when the diagnosis of pulmonary tuberculosis (in various terms, "weak lungs," "tendency to consumption," "incipient phthisis," "a spot or patch in the lungs," "going into a decline") has been made. Such mistakes were inevitable and excusable in the olden days, when the diagnosis of early pulmonary tuberculosis was largely based upon vague guesses at truth. Nowadays, with tuberculin in our hands, whether we use it in the old or modified method of subcutaneous injection, by Pirquet's or Calmette's (Wolff-Eissner's) method, there is little or no excuse for these mistakes. Of course one has to admit that even with the most exact methods of clinical diagnosis human fallibility is responsible for some mistakes, and therefore at all times wrong diagnoses are possible, but such errors can be reduced to a minimum. Even with care, and after long experience I plead guilty to one or two mistakes, but in every such case but one I should not have made the mistake if I had been allowed to conduct the test in my own way. In the one case (Miss L.) in which I gave a wrong opinion, I allowed myself to be influenced by the nervous irritability of the brother, and did not give the full dose as advised by Koch. In an experience of fifteen years, this is the only case of pulmonary tuberculosis in which I know that I made a mistake. Still,

in order to establish confidence in this method of diagnosis introduced by Koch, I am ready to confess that if I had not used this test as a matter of routine in my daily practice, I should have been guilty, like other medical men, of many scores of mistakes. The test, of course, demands that the patient should also carry out his part of the contract. The temperature must be recorded properly every two hours after the injection, and all conditions, such as going to concerts, etc., liable to disturb the temperature, should be eschewed. In order to diminish the risk of errors, it may be well to conduct the test in a sanatorium under a nurse's eye. Calmette's ophthalmic reaction makes the diagnosis perhaps easier, but in my experience the reaction in the eye may be severe, and therefore compels the patient to stay indoors during the test. Personally, I should, after my experience with Calmette's test, prefer a subcutaneous injection to an ophthalmic instillation. The old bogey of the danger of mobilisation after a proper dose of tuberculin seems to have vanished into unsubstantial nothingness whence it arose. From my experience I am bold enough to assert that far from tuberculin causing mobilisation, tuberculin must prevent it, because though I have seen many cases of generalised tuberculosis, chiefly tuberculous meningitis in the later stages of pulmonary tuberculosis, I have not had a single instance of tuberculous meningitis in the cases I have treated with tuberculin, although I have used enormous doses of tuberculin in every stage of pulmonary tuberculosis. I quite admit that in hysterical and neurasthenic subjects the subjective sensations may be extremely severe, and even the injection (*injectio vacua*) may cause an attack of hysteria ; still, by patience and firmness, it is not difficult to carry through a course of tuberculin up to large doses, in spite of hysterical outbreaks, and to beat back the disease. Once the real disease has been checked or cured, the functional disease also gradually withdraws. There is some justification for the view of those French physicians of a past generation who called pulmonary tuberculosis a nervous disease. The nervous symptoms may be largely the result of tuberculous disease of the lungs, obviously a toxic effect, although Koch, Wassermann and

others hold that the toxins do not circulate in the blood to any extent.

Once pulmonary tuberculosis in the infectious stage appears in a family, the host must be made to obey strictly the regulations which are designed to prevent the infection spreading to other members of the family. The source of the infection is known, and provided proper measures are adopted by the patient, the risk of the disease spreading to the healthy is small; but otherwise there is a daily risk. It is in the homes of the poor that this risk is greatly intensified, especially in the later stages of the disease, when the victim becomes careless and often helpless, and then little children, crawling and playing about the floor, may easily become the innocent victims of a father's, or a brother's, or a mother's careless and even filthy habits. By coughing, or through the sputum, whether visible and massive or invisible and in minute droplets, such a patient may scatter broadcast the agents of infection, and often the injury he does to his nearest and dearest relatives is not traced to its proper cause. *The disease does not begin to show itself for one or more years, and after such a lapse of time the true source and cause of infection is quite forgotten.* It is the custom to disinfect the rooms occupied by such patients dying of the disease. Unfortunately, the damage is already done, and disinfection of this sort has but a partial effect. The door is bolted after the horse has gone. In the future it will come to be a *general practice to test those who for months and years have been exposed to the risks of infection.* Nothing has helped me in the early recognition of pulmonary tuberculosis so much as the routine use of tuberculin in these very cases. The disease is always in an early stage, and, in my opinion, can be completely cured by means of tuberculin in the great majority of cases.

The duty of the host towards his own family is obvious, as soon as the nature of the disease is established. Further, it is of the highest importance to determine whether the disease is so advanced that tubercle bacilli escape from the lungs during the act of coughing or sneezing, or even loud speaking or in the massive expectoration. If the bacilli escape, special precautions must be adopted and enforced. I

regret to say that even now, chiefly because heredity or a constitutional taint is held to be the dominant factor in causing the disease, medical men do not instruct their patients how to act so as to avoid the awful risk of giving a brother, or a sister, or a friend the dreadful disease. *How can it be otherwise when physicians acquiesce in the conspiracy to keep their patients in ignorance of the nature of their trouble.* I can see no sensible reason for this *conspiracy of silence.* It is rarely fair to the sick, it is never fair to the healthy. Such silence can do no good, and may do great harm. I have always told patients the nature of their trouble, if there was any risk of infection, and in no single instance has the patient been depressed or injured by the simple statement of the truth. It is quite easy to gain the confidence of the patient by encouraging him to believe that he must get much better, and if tuberculin be used early and properly, this hope will be fulfilled. Besides, one must treat the patient as an intelligent being, not as a child, when one urgently needs his co-operation in any system of treatment or prophylaxis. The host of the parasite of pulmonary tuberculosis in an infectious form, commonly called consumption, has clear duties towards society that must be respected if he wishes to remain within that society, but it is necessary to restrict his freedom as little as possible consistent with safety to others. Beyond a doubt we have to face an awkward dilemma when we advise upon the question of marriage. The risk of marriage is greater for the woman than the man. The increase of duties and therewith anxieties in the household may depress vitality, but for the woman pregnancy is the chief source of danger. If not pregnancy itself, certainly the normal duties of maternity inevitably increase the tendency to recrudescence of a latent focus and quicken the activity of an already active tuberculosis. Certainly if the mother be suffering from pulmonary tuberculosis in an infectious form, safety for the child requires that a healthy wet nurse should be provided, for the child should not be exposed to risk of infection by its mother after its birth. How to secure safety to the child in such circumstances may tax the resources and judgment of the physician. The relief from lactation may save the mother's life, especially

if at the same time tuberculin be used for curative purposes. In the man, the chief danger lies in the possibility of infection spreading to the wife. There are those who think that this danger is not great, because unless the wife or husband, as the case may be, is predisposed, infection will not occur, and if either be so disposed, the risk from other sources is so great that tuberculosis in the husband or wife does not greatly add to the risk. My own impression is that the risk is distinctly and seriously increased. A serious difficulty in the way of a sound conclusion is the fact that infection may occur and not manifest itself for some years later. Further, one must not look for infection until the case has become one of open tuberculosis. Accordingly, all cases in the first stage are useless for the purpose of investigation. If tuberculin be freely used for diagnostic purposes, and Calmette's test may be used widely in order to throw light upon this phase of the question, we may have more definite facts upon which to come to a logical conclusion. Even if a wife or husband be expelling bacilli in the sputum, precautions may be adopted and diminish greatly the risk, or the dose may be so small that the evidence of infection is greatly delayed. Still, if one or other has suffered from open tuberculosis for two years or more, infection should be manifested in greater degree among these than among other married persons, who have not been thus exposed to infection in the family. Naturally, if a husband be consumptive, and others suffering from consumption be in the household, infection of the wife may take place, although the husband has not been the source of infection. In all such cases tuberculin as a diagnostic agent should be used, either in Koch's way or in Calmette's way, since infection may have taken place, although there be no evidence of disease in the form either of symptoms or physical signs.

Once a case of pulmonary tuberculosis—especially of an infectious character—has appeared in a family, the sufferer must be informed of the nature of his trouble, and very clear and specific instructions must be given to him not only as to the best way of disposing of his expectoration, but also with respect to his manner of coughing, etc. Coughing,

sneezing, and even loud talking may be the chief source of danger, especially when the cough is severe and frequent and the visible expectoration of small amount.

In such cases thousands of bacilli may be expelled into the air, and, yet being invisible and unseen, the imminent risk is not recognised. Whatever be the cause of the cough, *the handkerchief and not the hand should cover the mouth during the act of coughing*. Else the hand itself becomes infected, and in various ways may pass the infection on to others. How is it possible to put this reasonable system of prophylaxis into practice unless we take our patient into our confidence and tell him plainly the nature of his disease, with the double object of getting his assistance in every detail that pertains to his own good and the good of his fellow creatures?

PART IV

THE PROBLEM FOR THE PHYSICIAN

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CHAPTER I

HEREDITY IN TUBERCULOSIS

THE discovery of the tubercle bacillus as the cause of tuberculosis has profoundly modified our ideas upon this phase of the problem. Heredity implies the transmission from parent to offspring of certain definite features, qualities, tendencies or potentialities, but no influence that comes into play after the union of the male and female elements, *i.e.* after conception, has anything to do with heredity. No one assumes that the male or female element may carry with it a tubercle bacillus. In fact, a disease due to a parasite cannot be inherited. The parasite belongs to the environment, which is absolutely outside of, and unrelated to, those vital qualities in cell-life which determine inheritance. A tuberculous mother may conceive, and the tubercle bacilli in her blood may pass through the placenta after it has formed and infect the fœtus *in utero*, but this presupposes that the mother is suffering from a late stage of tuberculosis, when the tubercle bacilli are circulating in her blood. In such a case the fœtus may be born with tubercle bacilli in its tissues, or even rarely with definite tuberculous lesions. This is not hereditary but congenital tuberculosis. Such forms of tuberculosis have been observed in animals and man, but they are rare curiosities. It is physiologically inconceivable, if not impossible, that a tuberculous father can infect his offspring before birth in the act of conception. Long ago, Gärtner exhaustively discussed this phase of the question,

and little further need be said upon the subject. It is very rare for the tubercle bacillus or its disease to exist in the tissues of the fœtus at birth, and in all cases the mother has been the subject of tuberculous disease during the period of pregnancy. In any case the hereditary transmission of an extraneous foreign substance can have no meaning, and it would be well if the term heredity in relation to tuberculosis were abandoned. At most one may imagine that tissues possess a greater or less affinity for certain germs or their poisons in accordance with Ehrlich's brilliant hypothesis. This tendency does depend upon inborn and transmitted qualities of cells and tissues, but similar tendencies no doubt exist in relation to many other, if not all, infectious organisms and merely implies vulnerability. No doubt certain qualities in the mother's blood, especially in the fluid part, may be transmitted to the fœtus *in utero*, mostly increasing, rarely diminishing, resistance, but again these changes are acquired and not inherited. In the same way, the milk of the mother may convey to the suckling offspring elements that protect against infection, but then only if the mother herself has become protected by an attack of the special disease. These facts are merely the ordinary phenomena of natural and acquired immunity, and acquired immunity in itself implies disposition to disease. For the most part, acquired immunity is the direct result of a post-natal infection. Too often disposition to disease is a term which implies little else than exposure to infection. Von Behring goes further still, and is the author of the view that disposition to tuberculosis is nothing less than a condition depending upon the penetration of the intestinal mucous membrane of the infant by tubercle bacilli after its birth—is, in fact, tantamount to actual infection. Thus Behring holds the inheritance of tuberculosis to be merely a wrong name for infection in early infancy. Behring and others, notably Cornet, greatly discount the popular conception of predisposition. Many authorities still believe that a disposition to tuberculosis exists in certain races, and even in certain isolated families of the same race, quite apart from infection. Disposition is merely a relative term, indicating a bias towards infection just as natural immunity

indicates a bias against infection. In tuberculosis it would seem that, in spite of so-called predisposition, there may be a distinct bias towards acquired or artificial immunity. Thus many high authorities, Weicker, Turban and others, seem to prove that treatment in sanatoria yields better results in the predisposed. The predisposed resist the disease longer and resent it less than the normal individual. Tuberculosis, having its origin in an extraneous vegetable parasite, cannot be an hereditary disease, and, in default of the old doctrine, the disposition is variously interpreted. Some attribute it to an inherited taint, some to an acquired constitutional defect (Spengler, inherited syphilis), neither of which can be estimated or defined nor yet eliminated ; while others again find the embodiment of this disposition in certain local anatomical features. Thus Birch-Hirschfeld traces the disposition to certain developmental defects in the apices of the lungs, W. H. Freund lays great stress upon certain anomalies in the thoracic wall, especially defects in the growth of the first rib, and Rothschild considers that abnormalities in the setting of the manubrium upon the sternum is a factor in disposition.

All these views (Behring, Birch-Hirschfeld, W. H. Freund, Rothschild) illustrate the progress of thought and increased knowledge from the vague and indeterminate to the definite and determinate. Thus dissatisfaction with the older explanations has led to a revolt against views that are as old, and should be as dead as Hippocrates. These iconoclasts, notably Behring and Cornet, will hear of no disposition to tuberculosis other than that which applies equally to diseases of a like kind, such as diphtheria, tetanus, glanders, leprosy or syphilis. The tendency of the old school is to labour the question of predisposition, the tendency of the modern school is rather to give a right value to the definite, tangible and measurable factor—the infective agent in all its variations and vicissitudes.

When we have mastered the specific affinities of the different kinds of tubercle bacilli in all their grades of virulence for different animal tissues, we may begin to understand the meaning of predisposition. Gradually our

present conceptions of disposition as a factor peculiar to tuberculosis will be brushed aside, like many another conception, in the clearer light of scientific investigation. Meanwhile there is nothing illogical in craving some consideration for the view that liability to the disease may be mainly the result of the opportunity for, and the intensity of, infection, and variations in the disease itself may depend rather upon the kind and virulence of the specific cause than upon the nature of tissue as a soil. The disease is the result of a specific affinity between the specific cause and the tissues, and this specific affinity manifests itself in the lesion. Whenever, then, a lesion occurs, there is evidence of specific affinity. The investigations of Schlenker, Naegeli and Burkhardt have shown that lesions held to be tuberculous are found in the majority of *post-mortem* examinations (Schlenker, 66 per cent.; Naegeli, 93 per cent.; Burkhardt, 91 per cent.). "Jedermann ist ein bischen Tuberculös." Such latent lesions are found in the delicate (predisposed) and in the robust. But though these observers have recorded a great variety of lesions, many of them merely interesting as *post mortem* curiosities, but having no interest for the clinical physician, they have ignored the tubercle bacillus. It is therefore not by any means certain how far these lesions have been misread and misinterpreted. Nor should we forget that these facts and deductions were drawn from observations upon the poorest section of society, living in a community (Zürich) where tuberculosis is very prevalent (Naegeli).

Further, it has to be remembered that more often than not, the rapid course of tuberculosis, which is relatively common in growing girls, is the result of other organisms (mixed infections) than tubercle bacilli; thus the clinical picture is further blurred by these extraneous factors. Are we ready, then, to extend our ideas of predisposition also to the infections caused by streptococci, pneumococci, influenza bacilli, etc. These cases of galloping consumption arrest attention, and the acute course of the process readily suggests to the layman that there must be predisposition to account for this riotous behaviour. Since it has been proved that this tumultuous and galloping form very often owes its origin to factors

outside of the tissues and independent of the tubercle bacilli, it is evident that the logic of those who affect the hypothesis of predisposition must at times be of a sinuous or circuitous variety. The less we think and talk about predisposition, which we cannot define or explain, the sooner we shall find the path that will lead us to a practical solution of the momentous problem of the prevention and treatment of consumption.

Surely, too, it is rather absurd to speak of predisposition, on the one hand, as though it were a sort of predestination, and, on the other hand, affect to believe that residence in a sanatorium or in some lovely climate, even for a year, will effectually neutralise the action of this mysterious predisposition. Those who cling to this mysterious idea are the very people who applaud the virtues of climate, of high altitudes, of open-air methods, and add mystery to mystery when they attempt to explain the rationale of their "little systems, which have their day and cease to be."

It is quite conceivable that an injurious or toxic influence might be transmitted from the patient to the offspring, either through the male or female element, but such an influence might have no relation to the action of tubercle bacilli in the parents or ancestors. A specific toxic influence can only act when one or other parent is actually tuberculous—harbours in a living and active form the tubercle bacillus. But that which is designated predisposition to tuberculosis may have no relation to tuberculosis in ancestors. It would be hard for the theorists to explain how tuberculosis in a cousin or an aunt could thus affect collateral members of a family. Accordingly, I have systematically disregarded the history of tuberculosis in any but those in the direct line—parents, grandparents and their direct progenitors. If neither parents nor grandparents suffer from tuberculosis it is straining the conception of a constitutional tendency to breaking point to search for evidence among collaterals. Again, one may argue that this so-called constitutional bias towards infection may have nothing to do with the existence of tuberculous disease in parents or ancestors, and the occurrence of disease in the "cousins and the aunts" is the visible expression of this

bias. This is not the idea of predisposition that presents itself to most laymen and others who argue in a circle. The common idea is that a father or mother of tuberculous stock (whatever that may mean) can imprint upon their offspring a stamp or mark characteristic of tuberculosis, and as an illustration, the "habitus phthisicus" is the outward and visible sign of this tuberculous heritage. It has to be proved that this "habitus phthisicus" is in any way related to the existence of tuberculous disease in previous generations or parents. I have seen more than one case in which this "habitus phthisicus" was extreme in the child, yet both father and mother were quite healthy. Moreover, I have seen it well marked in individuals showing no obvious tuberculous disease. The external picture of the "habitus phthisicus" is well known. This "habitus phthisicus" manifests itself in the thin, weakly, weedy, long, lanky individual with stooping gait and drooping shoulders, a narrow, flat chest, wide, oblique, intercostal spaces moving but little (paralytic chest), and wing-like shoulder-blades projecting outwards. This type of individual is very thin, the skin is thin, and the vessels of the skin thin and weak. Often, too, the skin is cold and clammy, and cold drops of sweat often trickle down from the axilla; the hair of the skin is long, downy, light in colour, and poor in quality. Vasomotor irritability is marked, and flat-footedness is common. Such are the external signs of the "habitus phthisicus." This type, said to predispose to tuberculosis, may be independent of tuberculosis in parents or ancestors, may be absent in tuberculous subjects, and may certainly be, and perhaps is, the result of tuberculous infection in the individual. Naturally, such a form of chest, by interfering with movement (paralytic chest), eminently favours infection and the progress of pulmonary tuberculosis.

Because there is a liability to disease, it in no sense follows that the same disease in parents or ancestors has helped to create this liability. On the contrary, it is a general rule in infectious diseases in man and animals that the occurrence of infectious diseases in the immediately previous generations tends to create some degree of immunity. This general rule

may or may not apply to tuberculosis, but the simple fact, attested by the best authorities, that pulmonary tuberculosis runs a more favourable course and does not necessarily impair the health of the victim so seriously or so rapidly when there is a distinct family history of tuberculosis, seems to indicate a certain degree of inherited immunity. Thus the offspring of these families, in which tuberculosis has appeared in the previous generation, should be rather protected than predisposed. According to Turban, those with a family history gave better results than those without any such history in the proportion of 49·6 per cent. : 44·8 per cent. According to Weicker, in the proportion of 46·6 per cent.: 44·1 per cent. Thus the constitutional tendency does not add to the gravity of the prognosis in individual cases. The predisposed, in fact, stand the disease better, enjoy life better in spite of weakness and illness, and fight the disease better by living longer. In my own experience the fine, healthy, active athlete, if he fall a prey to the disease, makes a very poor fight for life if he depends solely upon the vigour and vitality of his organisation.

From a sociological point of view, the question arises whether this disposition to tuberculosis may be related to a general weakness or loss of resisting power akin to degeneration. The physical deterioration of the European races has been a subject of anxious observation and inquiry of late years, but it can hardly be urged that the prevalence of tuberculosis is a measure of this physical degeneracy. In England, Norway, Sweden, Belgium, and even in Italy, the nation seems to suffer less loss from tuberculosis than in Germany, France, Austria, and Russia ; yet the ugly facts disclosed by the recruiting officers of the English Army, before and during the Boer war, offer us material rather for serious reflection than for congratulation. Tuberculosis is the special curse of cities, causing many more deaths in the city than in the country. Predisposition utterly fails to explain this. Opportunity for infection is the obvious explanation. At any rate, if the environment of cities be the potent cause, the disposition is acquired and not transmitted. Prisons, asylums, factories—even convents and

schools—favour infection, and the striking success that followed in the wake of preventive measures in such institutions in Germany shows that facilities for infection were the effective cause of the prevalence of the disease. The city population is recruited and reinforced from the country. Country life is the ideal life for the maintenance of a high standard of health in the nation, but if 60 per cent. of the male population of Great Britain shows signs of degeneracy, smallness of stature, narrowness of chest, and bad teeth, the country must also supply a certain contingent of degenerates. The colonies of Great Britain are trying to teach that health and contentment are the happy lot of the man who can live on the land. No doubt to the healthy instinct, country life with its abundant opportunities for physical exertion and life in the open air appeals with a force of its own, and perhaps when health and the faculty of enjoying these simple pleasures are on the wane, man lives but in a paradise lost, and restlessness and discontent may urge him to regain his paradise in the turmoil of the city. Thus the individual of weakly constitution may tend to gravitate towards the city, so that a certain number of degenerates found in cities may come from the country. Probably, too, by a process of natural selection, the weaklings and wastrels both of town and country seek those very occupations in factories, etc., that favour pulmonary tuberculosis. Thus far individual disposition may also discount the influence of occupation as a predisposing factor in the origin of tuberculosis. Now if tuberculosis mainly attacked weaklings and degenerates, some sociologists might question the wisdom of prophylaxis, but every medical man and very many laymen must know that, like other infections, such as pneumonia, smallpox, or typhoid fever, tuberculosis itself may and often does attack the best men and women in the race. There may be perhaps some grim satisfaction for the unfortunate victim of tuberculosis to bear this in mind. Nevertheless, not the most intense disposition can cause the disease unless the germ of consumption—the tubercle bacillus—has entered the tissues and found a lodgment. Even after their entrance, the germs may remain latent, or may cause sufficient reaction in the tissues to lead to their imprisonment

and render themselves harmless, at least for a time. These latent foci are a frequent source of trouble in the future. How long the tubercle bacilli may remain quiescent but still virulent cannot be determined. Lydia Rabinowitch records a case in which the nodule had become calcified and yet living, and virulent tubercle bacilli had survived therein. It is certain that the bacilli, though quiescent, may retain their life and virulence in joints, in bones, in glands, and in the lungs for many years, until some accident, a strain, a blow, a bruise, or an infection, especially measles,[1] or whooping cough, or influenza, or a simple cold, or a succession of colds, sets the spark to the infection, and active tuberculosis, even of an acute type, may suddenly assert itself. Then certainly the condition may be grave, and baffle the best efforts of the physician. Even tuberculin may entirely fail, because tuberculin must be given slowly and cautiously, and it may need many months to introduce a sufficiency of tuberculin to overtake the disease. Before these months have elapsed, even in the course of a few weeks, an acute process may run its course to a fatal issue. On the other hand, if tuberculin had been administered in the latent stage of the disease, such catastrophies might be prevented. Even general diseases, such as diabetes and chlorosis, may give the sleeping enemy its opportunity. My own experience further convinces me that alcohol helps the disease to overpower the resistance of the tissues, no doubt by depressing vitality. If a tuberculous patient be addicted to alcohol, sometimes through the unwise recommendation of alcohol as a weapon for fighting the disease, I refuse to attempt treatment until total abstinence is ensured.

Hansemann relates a case in which tuberculous meningitis supervened in a child soon after a serious fall on its head.

[1] In a very striking way I have seen the effect of measles in increasing the sensitiveness to tuberculin, and this hyper-sensitiveness to tuberculin is the manifest expression of increased disposition to tuberculosis. Case I. Woman in third or fourth month of pregnancy, suffering from extensive disease of right lung, and moderate disease in left. Treated with tuberculin ; no reactions ; had severe attack of measles. After measles, severe reactions, even with much reduced doses. Case II. Young girl, twenty, affected in just the same way. The attack of measles profoundly altered tissues in their behaviour towards tuberculin.

At the *post-mortem* examination, the *fons et origo* of the tuberculosis was found in a caseous bronchial gland at the hilus of the lung. It is a matter of everyday knowledge that an acute inflammation of the lungs, due to streptococci, pneumococci, the bacilli of influenza, and whooping cough, may convert a latent into an active tuberculosis of the lung. Until the true import of these latent foci of tuberculosis as frequent sources of active pulmonary tuberculosis be recognised, the full value of tuberculin in early diagnosis and treatment cannot be appreciated.

The enemy may lie in ambush for many years, but it is nevertheless a source of constant danger. A wise man will design to destroy the enemy before he has the chance of doing mischief.

CHAPTER II

EARLY DIAGNOSIS

THE early diagnosis of pulmonary tuberculosis is quite the most important chapter in the problem before us. Like many other diseases, tuberculosis may be easily and completely arrested in its very early stage by proper methods. Nowadays, in infectious diseases at least, there is no room for "happy guessing." In diphtheria, in plague, and in other like diseases, diagnosis is something more than "happy "guessing." There are means at our disposal in the diagnosis of infectious diseases which rarely play us false, but before we consider these special methods of detecting the disease in its first beginnings, it will be well to have a clear idea of the early stage of pulmonary tuberculosis. But a few months before his death, Birch-Hirschfeld demonstrated at the Berlin Congress upon tuberculosis that pulmonary tuberculosis most often begins as a local deposit in the mucous membrane of the medium-sized bronchus. In 32 cases of latent pulmonary tuberculosis occurring in 196 cases of sudden death examined by Birch-Hirschfeld, not one case presented the anatomical lesion of tuberculous broncho-pneumonia starting as a caseous inflammation of the finer bronchial tubes. In four cases the early changes consisted of an intestinal miliary tuberculosis, while in 28 cases "the primary bronchial tuberculosis is by preference "(*vorwiegend*) localised in the posterior half of the apex of "the lung (more often in the right than in the left) and "in the part of the upper lobe lying immediately over "this"—an area that corresponds to the distribution of "the *bronchus apicalis posterior*. This part of the bronchial "branch corresponds to that part of the chest wall which

"participates least in the respiratory excursions and presents "also in adults, by virtue of its position, unfavourable con- "ditions for overcoming mechanical hindrances to the "respiratory air-currents. This disposition to disease is "further favoured by certain defects in the development "of these very bronchial passages, that may probably be "largely the result of unfavourable conditions of life during "the period when the growth of the lungs is most active— "in puberty." If primary tuberculosis of the lungs is due to the invasion of tubercle bacilli in the inhaled dust of the air, this anatomical disposition favours and determines the settling of the infective agents as foreign bodies. Conversely, these facts strongly favour the view that the infectious material is first conveyed to the lungs by the air-passages. Moreover, it is difficult to connect a disposition of the lungs, depending on anatomical defects or peculiarities, with any special constitutional tendency. Such defects may also be reinforced by secondary conditions in the lungs, catarrhal and other inflammatory conditions causing injury to the protecting ciliated epithelium. Once the tubercle bacilli have invaded the mucous membrane of the bronchus further changes of a tuberculous character ensue, causing swelling of the mucous membrane and narrowing of the lumen of the bronchus, followed in time, maybe, by obliteration of the bronchial tube. Thereupon the smaller bronchial tubes beyond the lesion shrink and collapse and produce the well-known signs of early pulmonary tuberculosis—depression above and below the clavicle, impaired movement in this area, increased vocal fremitus, impaired resonance, altered breath-sounds, and maybe the adventitious signs of catarrh. Herein we have presented to us by the hand of a master not only the anatomical substratum, which produces the early physical signs of pulmonary tuberculosis in all their fine gradations, but also a rational explanation of the frequent incidence of tuberculous disease in this very situation. Such an anatomical lesion in its first beginning cannot fail to teach us why in the early stage of the disease physical signs may fail—*one of the primary lessons to be learnt by those who desire to detect the disease in the very early stage.* If we accept this series of

anatomical pictures, based on careful and exact observation, as the condition obtaining in most cases of early pulmonary tuberculosis, a view in complete harmony with the less direct evidence of physical signs as observed by clinicians, we hold in our hands the key to the proper understanding of the meaning of the early stage of the disease and of the difficulties that may involve its detection at the bed-side.

Subsequent changes and extension of the disease to other parts of the same lung and to the lung of the opposite side are due to a variety of causes. (I know no better account of the usual course and progress of the lesion in the lungs than that given by Dr. Kingston Fowler in "Diseases of the Lungs, by Kingston Fowler and Godlee," but I absolutely join issue with him upon the meaning and influence of mixed infections. It is quite evident that Dr. Kingston Fowler has not himself investigated this important phase of pulmonary tuberculosis). Once the primary focus has progressed to caseation, softening and ulceration, the tuberculous process may extend on the distal side of the lesion, and tubercle bacilli, liberated from the original focus, may be drawn by the aspiratory action of the chest into neighbouring and distant parts of the lung and even into the lung of the other side. Cough especially helps to scatter the germs in all directions, although at the same time it may get rid of much infectious material. The diseased area, especially if there be disintegration of tissue, however slight, indicated by the presence of tubercle bacilli in the expectoration, and obviously if there be much ulceration of tissue, offers a nidus pre-eminently suitable for the growth of other organisms, especially of streptococci, staphylococci, pneumococci, and influenza bacilli. Then begins the sad eventful clinical picture of the mixed or secondary infections. It may be at this stage when severe symptoms develop that the victim is first conscious that he is really ill. Not seldom even in this stage the disease is misunderstood and misnamed "influenza," "a severe cold," "bronchitis," "pleurisy," sometimes even "pneumonia" and "typhoid fever," and further disaster may follow in the wake of slip-shod methods of diagnosis. Any of these conditions may be present, but an etiological diagnosis alone can bring the possibility of succour

within reach of the patient and credit to the physician. At this critical stage in the patient's history there is no margin for mistakes. The urgent problem to be settled is whether there is infection with tubercle bacilli alone or in association with other organisms. This embraces the etiological diagnosis, a subject quite important enough to deserve a chapter to itself, for herein lies the great secret of success. While the tuberculous focus is closed, tubercle bacilli do not escape into and by the sputum, and in this stage the risks of secondary or mixed infections are at a minimum.

Pleurisy is merely a phase and form of pulmonary tuberculosis—in most cases an integral part of the tuberculous lesion—rarely the initial or sole lesion. Such at least is the nature of the chronic pleurisy that occurs so frequently in tuberculous lesions at the apex of the lung. Elsewhere tuberculous disease of the pleura may occur and cause localised areas of dry pleurisy, or a chronic, acute, and sub-acute form of pleurisy with effusion. For years past I have tested with tuberculin every case of pleurisy that has come under my observation—cases of chronic latent pleurisy with abundant effusion, with moderate effusion, and other acute cases with high fever and severe symptoms, and so far every case tested (in fever cases after abatement of the fever) there has been a positive reaction. A few years ago Professor Osler admitted to a percentage of 30 in these cases. I believe that now he fixes the percentage much higher. On the other hand, I have seen many cases called pleurisy in which I could find no physical signs and the tuberculin test was negative. One of these cases was treated at the out-patient department of a Hospital for a year by two different medical men. Getting no relief, he came to me. He had no cough, no physical signs—he did not react to tuberculin—pain he certainly had, but worse on moving, especially twisting, not on coughing, and affecting both sides. This was rheumatism, not pleurisy.

Thickened pleura at the apex is a very common accompaniment of pulmonary tuberculosis and modifies the physical signs.

These tissue changes at the apex constitute the chief basis of classification, especially in the cases in which

there is no fever. If fever exists, the cause of the fever must be determined if possible. In grave cases fever—even high fever—may be due to the activity of the tubercle bacilli in the tissues, but in the vast majority of cases the fever occurring in pulmonary tuberculosis appears at a later period of the disease and signifies mixed, or secondary or concurrent infection. Weicker speaks of a temperature within normal limits but showing marked subnormal depressions in the morning as suggestive of tuberculosis. The absence of fever does not exclude the presence of secondary organisms, for "passive mixed infections" are recognised. Further, since localised catarrhs, even at the apex, may be due to streptococci, pneumococci, and influenza bacilli (Neufeld), a confusion of non-tuberculous and tuberculous infections is inevitable, unless tuberculin be used for diagnostic purposes. In the presence of fever, Calmette's ophthalmic reaction recommends itself. It is especially in cases accompanied by fever that the etiological diagnosis is indispensable, and instead of waiting for the fever to abate, for the purpose of giving a test dose of tuberculin subcutaneously, one may proceed at once to use Calmette's method.

My own experience and the records of sanatoria show that within a year of the first appearance of symptoms the disease may reach any stage. Hence the duration of the disease may deceive, for patients may allow the disease to reach the third stage before they consult a physician. Cases of this sort are acute cases, and always accompanied by fever and severe symptoms. Most often the acute symptoms, such as progressive weakness, emaciation, sweating, anorexia, and even diarrhœa, are due to a mixed infection. The fever is generally intermittent, normal, or subnormal, 96–97° in the morning, 102°, 103°, even 104° in the evening (streptococcal curve). Less often the fever is more or less continuous, 102°, 103°, 104°, especially in the pneumonic form. I have not yet seen a case quite like those described by Fränkel and Troje, and later by Spengler. Of acute cases I have had several, though even they are fortunately not very common. In these forms some localised lesion sets free a large number of bacilli, which scatter themselves through the lungs and cause numerous foci which coalesce to

form large areas of tuberculous infiltration and consolidation, followed by extensive softening, maybe a puriform softening of lung tissue. The tubercle bacilli are then specially virulent, and cause the symptoms of an acute toxæmia.

It will be useful to give a few illustrations of cases running an acute and rapid course.

M. M., *æt.* 19, seen in consultation with Dr. F. Physical signs of lobar pneumonia, involving whole of lower left lobe, dullness, tubular breathing but crackling râles. Sputum contained numerous tubercle bacilli, and also yielded on plates abundant growth of pneumococci. Continuous fever, 103—104°. Crisis failed to appear. I advised intravenous injections of collargol, and three injections of 10 c.c. of a 1 per cent. solution were given at a few days' interval. The fever abated and disapppeared, and the temperature remained normal for many months. In the lungs the signs of extension and disintegration appeared, and after six months from the time I first saw the patient a sudden and fatal hæmorrhage caused the death of this poor boy, without his ever suspecting the real nature of his illness. Naturally in such a case there is every reason for keeping the patient in ignorance of the truth.

Two similar cases in young girls—one 13 years old, the other 18—with intermittent temperatures of 102°, yielded in the same way to intravenous injections of collargol, and although both these cases were in the third stage when they came to me, one of them pronounced by three leading doctors in consultation to be "dying," they are still alive—improved beyond recognition—by carefully graduated doses of bovine tuberculin (P.T.).

A fourth case of mixed infection in a young married woman, who had a temperature of 104° every evening for ten days with a normal morning temperature, received a single intravenous injection of collargol. The effect was dramatic. The temperature fell to normal next morning and remained normal afterwards. Treated with tuberculin this woman also is to all appearances well. It is more than three years since she had any treatment.

Another very extraordinary case is worth recording. The

young girl, *æt.* 17, served in a medical man's house. She had an attack of pneumonia, with its symptoms and physical signs and even sputum. The sputum contained a little blood, but, though examined by the doctor himself several times, and also by me, and afterwards in the hospital, no tubercle bacilli were found in it. However, crisis failed, and irregular temperature persisted. Being interested in this case, which had been examined by at least seven other doctors, I undertook to take her into the hospital and test her. At my first visit I told the doctor, her master, that I thought there must be tuberculosis. It was a case of apical pneumonia, involving the whole of the upper lobe. She was 4 st. 5 lb. on entering the hospital. After a few days in hospital the temperature became normal. The first dose of tuberculin raised the temperature to 103°, and for some days the temperature was above normal. She was treated with P.T.O. and P.T. In two months she gained a stone. She went up the country and in 1907, two years and a half later, was back in her master's house, weighing 8 st. and apparently well. This doctor is convinced of the value of tuberculin. All the other doctors who saw this patient agreed with my verdict that it was acute tuberculosis.

Another acute case came into the hospital and was treated for pneumonia. It appears that, as he did not seem much distressed, several medical students had been allowed to examine him. When I approached to examine him, he took me also for a student and resented further examination. As he seemed to be fairly well, I did not examine him, but passed on. These facts were all brought to my mind vividly when, in the course of a few months, this man came to me with the history that he had never got over the attack he had in the hospital, and I found on examination that he was in the third stage of consumption. I treated him also with tuberculin emulsion for pity's sake, and he gained nearly a stone in weight. Temperature, for the first week 100–100·5°, gradually fell to normal, and improvement was obvious.

Acute cases generally run their course in months rather than weeks, but under tuberculin treatment I have seen acute

cases showing remarkable improvement and living years after treatment. There is no difficulty in classifying such cases. The diagnosis from pneumonia depends on a careful discrimination of symptoms and physical signs, and the failure of crisis, followed by signs of destructive processes instead of resolution in the lung. The sputum in pneumonia should always be stained, and if Ziehl's stain for tubercle bacilli be used with methylene blue as a counter stain, pneumococci will be detected, and also tubercle bacilli, if they should be present.

Chronic cases, on the other hand, may progress so slowly that the duration of the disease cannot be accurately determined. Indeed, chronic cases may cause no special symptoms and may not be recognised till the *post-mortem* examination reveals their real nature.

There may be no pulmonary symptoms—no cough and no expectoration—even in relatively late stages of the disease.

Symptoms of the Disease.

In one case there was extensive consolidation of the right lung, especially of the whole upper lobe, with no cough and no sputum. General symptoms—loss of strength and energy and a state of invalidism due to the toxæmia—were pronounced. Cases may even reach a late second and third stage although tubercle bacilli cannot be found in the expectoration. Although tubercle bacilli do often appear in the sputum early, it is wise to consider the stage relatively late, since there must be an area of softening with ulceration, and a *closed* tuberculosis has passed into the *open* form with all its danger to the patient and risks to the relations and friends. On the other hand, the tuberculosis may long remain closed chiefly during the early stages of the disease. Hence we must unlearn the teaching of some years ago (von Ziemssen, Strümpell Grancher, Kingston Fowler) that the presence of tubercle bacilli in sputum is a very important aid in diagnosis. As a rule, when tubercle bacilli are present, there is no difficulty in recognising the nature of the disease by other evidence, but too often *tubercle bacilli fail to appear in the sputum* when evidence of this kind is sadly wanted. Still there is no excuse for failing to examine sputum,

and if the result be negative, an expert's opinion may further ensure a correct diagnosis. The chief advantage of discovering tubercle bacilli in the sputum is that it *ipso facto* dispenses with the need of testing with tuberculin, and proves the necessity for precautions to prevent the infection of the healthy, especially in the family. In my judgment and in my experience, failure to find tubercle bacilli in the sputum is a positive indication for tuberculin as a diagnostic agent. Those who neither examine the sputum nor use tuberculin are in a hopeless state with regard to the diagnosis of early pulmonary tuberculosis, and it follows therefore that, when the sputum fails or fails to contain tubercle bacilli, those who do not use tuberculin for diagnosis may either raise unnecessary alarm or encourage their patients to live in a fool's paradise. They are perched upon the horns of an awkward dilemma from which they cannot escape. If they trust to guessing, they might as well leave that to the patient. I have known so many wrong guesses even by men who examine for admission to our Sanatorium in New South Wales.

Certainly symptoms may avail in diagnosis; they may be pulmonary or local and general. The general symptoms refer to (1) the blood and circulation, (2) digestive apparatus, (3) nervous system, or (4) in women to the sexual apparatus (amenorrhœa, etc.).

Anæmia or, in young girls, chlorosis may be a part of pulmonary tuberculosis. Such girls are easily tired or easily excited, and function of any sort easily exhausted. There is a state of aglobulism with diminished energy, poor appetite, distaste for food, wasting, irritable weakness of the heart and vasomotor apparatus, irritability and increased frequency of pulse, especially on slight exertion, shortness of breath, palpitation, flushing, especially after meals, and marked sweating, especially in the axillæ. Persistent frequency of pulse (tachycardia) is a serious symptom, and sanatorium authorities reject those manifesting this ominous symptom; with a rapid pulse, arterial tension is low. Indeed, any or all the symptoms of a chronic form of anæmia or chlorosis are common. I have seen cases in which pernicious anæmia was suspected. Nevertheless, there may be no such signs of

anæmia or of circulatory disturbances, and increased frequency of the pulse is rarely a symptom in the early stage of pulmonary tuberculosis.

The temperature varies greatly, and must be taken every two hours for trustworthy records. The temperature may be normal in any stage, even in the third stage; it may be variable and unstable, easily disturbed by slight causes, *e.g.*, menstruation, and is not usually high, but rather sub-febrile (99·4°, 100°, 100·4°). A rise of temperature after meals and exertion suggests early tuberculosis. At sanatoria the regulation of the exercise depends on the temperature. Fever after exertion indicates that the exercise has been too great. An inverse temperature is not common, but specially signifies tuberculosis. Severe fever may suggest typhoid fever, rheumatism, even malaria.

Tuberculosis may cause symptoms simulating rheumatic fever and even swellings in joints (in one case erythema nodosum as well and in another erythemata), like those of acute rheumatism. In one such case the diagnosis of rheumatic fever had been made. I found also definite signs of induration at the left apex. This patient reacted in a most typical way (within a year her brother died of Addison's disease due to tuberculous disease of the suprarenals). (Case 6, Stage I.) This rheumatic type of tuberculosis is merely a manifestation of tuberculin-intoxication. Those who have used tuberculin must be very familiar with the pains in joints and muscles that in certain individuals appear regularly after injections, especially in the earlier stages of treatment, often time after time in the same joint—in the hip, in the knee, in the thumb, in the little finger, in the heel—or in many joints, or in the back or bones, or in muscles (cramps in the calves of the leg, even agonising and paralysing); pains, too, in and about the thorax, quite apart from pleuritic pains, at any rate without any signs of pleurisy. Less often pains in the neck and even in the eyes. There may also be severe pains in the epigastrium, doubling up the patient after each dose; perhaps related to diaphragmatic pleurisy, or they may be rather of the nature of visceral pains (Head), or dragging, boring, and even acute

lancinating or cutting pains in the shoulder, radiating into the arm, or deep pain in the chest. There are strange individual idiosyncrasies, because in the same patient the pains after injections are apt to affect the same part. I forbear to suggest that there may sometimes be a local deposit to explain these pains. One has great opportunities for studying these pains—their varieties and situations—after tuberculin injections.

The commonest local or pulmonary symptom is a cough, without or with expectoration. Cough is a common symptom apart from tuberculosis, but is one of the commonest and most significant symptoms of pulmonary tuberculosis in every stage. It is too often under-valued by the patient and his friends, and should never be attributed to other causes by the doctor until he has absolutely excluded pulmonary tuberculosis by a searching examination. In the insidious form of pulmonary tuberculosis the cough may be the first symptom to attract attention. An early cough may give the physician the all important clue to the real cause of the trouble. To call such a cough a "stomach" cough is folly; even among doctors the meaning and nature of this cough is often misunderstood. A slight cough, especially in the morning or after effort—maybe a mere dry, hacking cough—occurring in a growing girl or young man, should not be lightly passed over. There are many causes exciting cough as a reflex phenomenon, but in early life, especially in young girls, when aneurism or tumour is not a likely cause, the wise physician should have no peace of mind until he has proved—not merely guessed—that the cough is not due to pulmonary tuberculosis. Quite apart from pulmonary tuberculosis, tuberculous glands in the neck or mediastinum may press on the endings of the vagus or on the trunk of the nerve and excite cough. Detecting tuberculosis in the glands, the physician may prevent pulmonary tuberculosis by promptly treating with tuberculin. I have had several cases of this nature. Signs and symptoms apart from cough may be entirely absent. To the specialist in diseases of the nose, throat and ear, there are, of course, numerous familiar causes of cough, because the nerve endings of the vagus supply these organs, but he

must beware lest he also misplaces the origin of the irritation. The specialist is somewhat disposed to trace every irritation to the area in which he "lives and moves and has his being." I have seen so many cases in which first-class specialists, forgetting that sight cannot penetrate beyond the surface, have seen nothing to justify the diagnosis of tuberculosis, and yet, a year or two later, these patients have presented themselves to me in the second or even third stage of pulmonary tuberculosis. If nothing can be seen by the specialist, and the lungs have not been examined by an expert, this foolish guesswork may lead to a sad and fatal tragedy. The specialist in diseases of the throat must make these fatal blunders until he realises the enormous value of tuberculin in the early diagnosis of pulmonary tuberculosis manifesting laryngeal symptoms without any typical tuberculous lesions in the larynx. The hacking cough of chlorosis also cannot by its nature be distinguished from the cough of pulmonary tuberculosis, and, as chlorosis and pulmonary tuberculosis are often associated, nothing short of the etiological diagnosis by means of tuberculin can settle the real nature and origin of the cough. The expectoration, or phlegm, which comes from the chest (while the spit—a term in vogue in some sanatoria—comes from the mouth), varies in character and amount, and yields invaluable information of a very direct kind. Quite apart from tubercle bacilli, or the organisms of mixed infections, the mere quantity and gross appearance may help in diagnosis and prognosis. During a course of treatment under tuberculin it is very interesting to observe how rapidly the amount diminishes and the qualities alter till there may be little or no phlegm, or it may be so scanty that one has to wait a month or two before one obtains enough for a severe examination. In one case I waited two months for the expectoration, and although there was hardly more than enough for one cover-glass preparation, there were a dozen tubercle bacilli in the field. In another interesting case, which was being treated for pulmonary tuberculosis by a medical man who did not examine the sputum, I was surprised to find that the sputum contained a regular menagerie of bacteria, but no tubercle bacilli. I at once suspected trouble outside of the lungs, prob-

ably in the nose. There was no trouble in the nose itself, and no discharge ; yet there was a tiny pellet of pus in the middle fossa of one nostril, and on exploring the antrum with a cannula I washed out the most filthy putrid cloudy material. To make quite sure that there was no trouble in the lungs, I also tested with tuberculin, and the result to ·01 gr. Old T. was absolutely negative. Yet this woman had been treated for a whole year for lung trouble. The progressive changes in the sputum, with which we are all familiar, give most useful information concerning the state of the bronchial tubes. In the early stages the sputum may be watery mucus containing some charcoal pigment, and sometimes small opaque yellowish streaks or masses, which offer far the best field for the search of tubercle bacilli. Later, as the pyogenic organisms come upon the scene, the sputum becomes opaque and yellow or creamy, or of a dirty greenish colour. In rare cases this greenish or bluish colour may be due to the presence of the bacillus pyocyaneus. The number and character of the tubercle bacilli may not help much in the prognosis. Still, a large number of bacilli, distributed or in clumps, lying free in the mucus and not within leucocytes, is not favourable. On the whole, if leucocytes contain many of the tubercle bacilli, the prognosis may be better. The chain-like appearance of tubercle bacilli, in which the bacilli are generally wasted, notched and irregularly stained, is most often found in a late stage when cavities exist. It is a fact testified by many records, that even in the third stage tubercle bacilli may not be found in the sputum. In my experience it is exceptional not to find them. Of course, if there is bronchiectasis, as in Case 14, Stage III., it may be hard to find the few tubercle bacilli in the abundant mass of expectoration. In such cases again a test dose of tuberculin may be necessary to establish the diagnosis. However, it is chiefly in the early stages of pulmonary tuberculosis, when prompt treatment by tuberculin can work such wonders, that the absence of tubercle bacilli from the sputum misleads the unwary physician, and he loses the golden opportunity of showing his wisdom and saving his patient from an imminent tragedy. It cannot be insisted upon with sufficient vehemence

that in the great majority of early cases of pulmonary tuberculosis tubercle bacilli are not found in the sputum, and, therefore, an etiological diagnosis can only be achieved by means of tuberculin. Once this great truth, taught us so long ago by Professor Koch, is thoroughly recognised, the treatment of pulmonary tuberculosis becomes a relatively simple and satisfactory matter.

Hæmorrhage of the lungs manifests itself as blood in the sputum in every degree. Hæmorrhage may not occur at all in pulmonary tuberculosis, but the one predominant cause of blood in the sputum or hæmorrhage from the lung is pulmonary tuberculosis. Some specialists in diseases of the throat may think that bleeding from the throat is not uncommon. My experience tells me that bleeding from the throat can hardly be mistaken for hæmorrhage from the lung. If the blood comes from the throat, a specialist can easily settle its source by examining the nose from the front, or the nasopharynx, by means of the rhinoscopic mirror, and fixing the site of the bleeding point. If he cannot find or see the bleeding point, and especially if the nasopharynx is clean and dry, this source is absolutely excluded. In the pharyngeal wall or lingual tonsils and surrounding parts, bleeding points may rarely be discovered. In a hospital experience of nearly fifteen years as a specialist in diseases of the throat, I cannot call to mind a single case of bleeding from these parts which suggested hæmorrhage from the lungs, or in other words pulmonary tuberculosis. Always, too, after a hæmorrhage the sputum shows more or less marked traces of blood, and in time becomes rusty and brown. This does not occur in bleeding from the throat or simple blood-spitting from injury to the mouth or gums. Hæmorrhage, even small recurring hæmorrhages, but especially when the blood is an ounce or more, is always a serious symptom, and in itself signifies pulmonary tuberculosis until this disease has been excluded. I had two interesting cases of recurring hæmorrhage with moderate loss of blood amounting to one or two ounces a day. The physical signs at the apex were absent, but in both cases there were crepitations, not at the apex, but over the lower lobe of the

lung. In both cases the tuberculin reaction was absolutely negative. Hydatid disease was, therefore, diagnosed, and the X-rays applied. In both cases a shadow was observed. In one case the lung was explored without locating any tumour, but two months after the operation, the patient coughed up the cyst and was soon on the road to recovery. In the other case I sent the patient back to the surgeon, who had sent him to me, and, I believe, he advised the patient to wait till the X-ray picture was more definite. Hydatid disease of the lung is the only condition in Australia which must be remembered as a not infrequent cause of pulmonary hæmorrhage. Bronchiectasis and tropical abscess of the liver invading the thorax are less common causes. In the clinical forms of acute pneumonia with much blood in the sputum it is well to look for tubercle bacilli.

Hæmorrhage, as a symptom of pulmonary tuberculosis, may be trifling and transient, and yet be the important clue to a correct diagnosis. It may be the first sign of a mixed infection, when the occurrence of hæmorrhage is followed and may be preceded by fever in various degrees. This fever (103°, 102°, 101°) may persist for some time with remissions and intermissions. Such a hæmorrhage is a further sign that the lesion, perhaps hitherto closed, has become open. When hæmorrhage occurs early it often happens that then for the first time tubercle bacilli appear in the sputum, but tubercle bacilli may not be found in this bloody sputum. Of course the presence of elastic fibres in the sputum is not related to the early stage of pulmonary tuberculosis.

Laryngeal catarrh, often with a tendency to colds in the head (nasopharynx), may be an early symptom of pulmonary tuberculosis. Such catarrh is not tuberculous in character, and has, therefore, been termed pretuberculoüs. It causes hoarseness, which may be easily misinterpreted. I have found again and again in the history of my cases that in the early stages of their disease they had consulted a specialist for the throat, and he had told them that they had catarrh, but it was not serious. The terrible sequel in at least a score of cases that consulted me a year or more afterwards should be an everlasting warning to those specialists who on a

haphazard diagnosis called the early stage of pulmonary tuberculosis by the false name of laryngeal catarrh. Undoubtedly many conditions of the nose and nasopharynx favour the development of tuberculosis, and the throat specialist should always bear this in mind. At any rate, he should avoid a diagnosis, which may lead to a tragedy, by excluding tuberculosis in cases of laryngeal catarrh. If he fails to use tuberculin as a diagnostic agent in such cases, he may be risking the life of his patient.

My own experience compels me also to direct special attention to the careless and often flippant way of dealing with pleurisy. In my own experience pleurisy has rare relations to any other disease than tuberculosis, and its relations to tuberculosis are both frequent and intimate. Many and many of the tragedies due to pulmonary tuberculosis may begin with a pleurisy, which will perhaps keep the patient in bed for a few days or weeks, but then grant him a long respite, even for many years. The recovery is so complete to all outward appearance, and the respite may be so long, that only those who know the vagaries of this disease will trace the subsequent tragedy of consumption to the trifling and transient attack of pleurisy. I admit that pleurisy is often diagnosed when it is not present, but more often these moderate, mild and insidious forms of pleurisy are discovered and treated as minor ailments, hardly worth the doctor's attention. I must protest against such a foolish conception of this serious disease. In the last few years I have taken the trouble to investigate by means of tuberculin every case of pleurisy that has passed under my notice either in hospital or private practice, and I seriously assert that so far I have not found a single case of pleurisy of this character that did not react to tuberculin. Moreover, every case of pleurisy without a single exception was accompanied by other evidence of tuberculosis, and responded by way of improvement to tuberculin treatment in the most remarkable way. As soon as I meet with a case of mild or moderate pleurisy which is not tuberculous, I shall consider it well worth recording. Of course I am not speaking of pleurisy as a mere incident in other diseases, such as pneumonia, typhoid fever, influenza, or

of purulent pleurisies, but of those mild or moderate forms of pleurisy which so far have not received the attention they deserve and yet constitute an important contingent of the tuberculous diseases of the lungs. As soon as we begin to speak of pleurisy as a complication of pulmonary tuberculosis, we fail to understand the nature of pleurisy. A case of well marked pulmonary tuberculosis without pleurisy would be well worth recording, and the invasion of the pleura, whether early or late in the progress of the disease, is the simple and inevitable result of the progress and extension of the disease. On the other hand, the pleura being near the surface sensitive in nature, at any rate if inflamed, and having special physical signs (friction), may just contribute the facts necessary for the diagnosis of tuberculosis, the prompt and proper recognition of which may save the patient from the later tragedy which too often terminates the clinical histories of "simple" pleurisy. In my opinion, when the signs and symptoms of these pleurisies are definite there is no need to test with tuberculosis, because these simple and mild forms of pleurisy are always tuberculous. The acute form also, apart from the pleurisy that may accompany many infectious diseases, the so-called acute idiopathic pleurisy, is always merely a manifestation of tuberculosis. Accordingly, if we use the term "pleurisy" as a name for a definite disease, we should mean nothing but a localised tuberculosis in the pleura. According to this view, our statisticians should further revise their tables, and medical men certifying to deaths from pleurisy should by inference know "pleurisy" only as a lesion of tuberculosis. If then the diagnosis of "Pleurisy" is made, *ipso facto* there is tuberculosis, and treatment should be just the same as the treatment for pulmonary tuberculosis. Indeed, in most cases there is pulmonary tuberculosis with the pleurisy. Is it not strangely inconsistent to treat pleurisy as a slight ailment and to make such a to-do with open-air treatment and the like for pulmonary tuberculosis when they are virtually one and the same disease? This is my view of the matter, and it is based upon my own observation, that what is commonly called pleurisy is virtually tuberculosis of the lungs.

A long chapter might be devoted to the aberrant symptoms

of pulmonary tuberculosis, symptoms related not to the lungs at all, but to other organs. Chlorosis, anæmia, and amenorrhœa may be the striking symptoms which bring the patient to the doctor, and yet the physician or gynæcologist may rest satisfied with an examination of the blood or reproductive organs, while the source of the trouble lies undiscovered in the lungs. Again, any form of dyspepsia, gastric insufficiency, gastric irritation or nervous dyspepsia, chronic ulcer of the stomach, gastritis, may be simulated by pulmonary tuberculosis. The immediate and permanent improvement obtained by means of tuberculin in many such cases which have come under my observation constitutes a very satisfactory chapter in diagnosis and treatment. In all tuberculous forms of dyspepsia and gastric troubles, especially in young girls, tuberculin does wonders, both in diagnosis and treatment.

Nervous symptoms may also be obtrusive in pulmonary tuberculosis. Such symptoms may be due to metastasis causing actual tuberculous lesions in the nervous system, especially in the meninges. Let me give two striking illustrations. A young girl was sent from the sanatorium to the hospital and came under me. She was in the third stage of consumption, but nevertheless was a perfect model for a clinical lecture upon hysteria, exhibiting curable paraplegia or paraparesis, and the typical sensory phenomena in the lower limbs with the well-defined horizontal line of demarcation above the knees. I gave a clinical lecture upon this case of hysteria. Not only hysteria in its typical and intense form, but many other nervous conditions may be a part of the clinical picture in pulmonary tuberculosis. The sequel of this case is also interesting. A few months later this girl's mother died suddenly, and the girl then said she would die also. She was brought again to the hospital in a state of coma, and then I made the diagnosis of tuberculous meningitis, though the resident medical officer thought her condition was still hysteria. She died within two days, and at the *post-mortem* there were typical tubercles in the meninges, especially at the vertex. I had already seen another of these cases of tuberculous deposits in the vertex, in which the progress of the disease was very rapid, death occurring in less than three weeks.

The other case came into my ward during my absence in Tasmania, and Dr. R. diagnosed peripheral neuritis. When I saw the case the next day, I said to my resident that there was certainly myelitis (bladder trouble), but on examining the lungs I found pulmonary tuberculosis (Stage II.) in the left lung. At once I made the diagnosis of tuberculous meningitis, which within a few weeks was verified at the *post-mortem* examination.

On the other hand, another eminently instructive case proved the enormous value of tuberculin as a means of excluding tuberculous disease of the spine and meninges in children. This little girl of 13 had been bedridden for one and a-half years. She lived in the country, and was sent to a very sound doctor in Sydney, who treated the girl for a month, and then sent her to the hospital where I saw her. The girl was wasted to a shadow—literally skin and bone—pale-faced, and with the legs in a state of extreme flexion upon the abdomen, and both legs flexed upon the thighs. At first sight one was compelled to accept the diagnosis of the other doctors. It was quite impossible to straighten the limbs, and any attempt to do so caused pain and crying. Before proceeding to any radical measures, I told the students that we should first test her with tuberculin. I also told my house physician that I did not think there was tuberculosis. Even with a large dose of ·01 Old T. there was not the slightest reaction, nor was there any swelling or redness in the arm. At once I acted on the assumption that structural disease was out of the question. We fed the little girl by the nose, which she resented, and in self-defence she was ready to take food by the mouth; massage was exploited, her limbs were straightened under chloroform and bandaged down to two Thomas' splints. Within three months of that time this wretched specimen of humanity walked into the hospital to see me—a straight, robust, rosy-cheeked, and cheerful maiden of fourteen summers. After having spent a year and a-half curled up in bed, the transformation was extraordinary. Without tuberculin one would have long hesitated—and already the doctors had hesitated for a year and a-half—to do the right thing.

CHAPTER III

PROGNOSIS

IT is extremely difficult to estimate the value of treatment, because pulmonary tuberculosis runs such a variable course. I inquired into the history of about 80 cases in the third stage. In 21 cases the duration of the illness was less than a year, in 14 more than a year and less than 18 months, in 24 more than 1½ years and less than 2½ years, in 7 more than 2 years and less than 4 years—no cases of 4 years' duration—3 cases of 5 years', 4 cases of 6 years' duration, and isolated cases from 6 to 20 years'. Thus in these cases the duration was on an average from 2 to 3 years. In many of these cases doctors had been consulted in the early stages and had informed the patients that there was no trouble. What a different story might have been told if tuberculin had been used in diagnosis and treatment! All these cases were hopeless when they came to me. Many of them had open-air treatment, creasote treatment, etc., with the result that in less than three years they were dying. I might in justice call all these cases "*control*" cases, illustrating the failure of other methods of treatment. *Is it a mere chance that while in the hands of other men scores of cases passed from the first to the third stage in 3 years, not a single case can be recorded against me in which under tuberculin treatment the disease had passed from the first into the second stage?* I read thus in my notes: "Saw Dr. S. S. 1½ years ago, who said 'nothing "the matter with lungs.' Then saw Dr. B., who 'snipped off' "uvula for cough—sent to country by several doctors."

Again, "Seen nine doctors. A year ago Dr. B. (a good "doctor but no believer in tuberculin) said 'lungs were sound.' "Had open-air treatment. Weight has been good, but disease "has advanced to Stage III."

Again, "Nine months in a sanatorium—sent out 'lungs dry "and quiet.'—yet now Stage III."

Again, "Treated for indigestion for 9 months; then sent up "country. Stage III. in less than one year after."

Again, "Patient in Stage III. Milks cows."

Again, "Saw Dr. S. who said it was 'all in the throat'—a "year after in Stage III."

Again, "Came from New Zealand 3 months ago and gained "14 lbs.—yet now (3 months later) in Stage III. Pulse 140."

Again, "Pleurisy 12 months ago—sent to country—died in "another 6 months."

Again, "Dancer, 9 months ago weighed 9 st. 4 lbs. Had "pleurisy. Dr. S. said 'nothing the matter with lungs'—less "than a year Stage III."

Again, "Pleurisy 15 months ago—now in Stage III."

Again, "Saw Dr. R. 1½ years ago. He said 'chest was "weak'—open-air treatment. Now hopeless in Stage III. c. "Tuberculous laryngitis."

Again, "Saw Dr. C. a year ago—sent to Bathurst, gained "weight and lost cough—in Stage III. and died 3 months "after."

I have records of at least 115–120 cases in which ordinary methods have utterly failed within 2 or 3 years for the most part. With tuberculin I have had no single failure of this kind. Almost all these cases had had ordinary treatment—had been sent to the mountains and well fed—yet in less than three years the greater number are dead. These numerous cases show the uselessness of haphazard trips to the country. Many of these patients had the early warnings—pleurisy, throat symptoms, cough, and loss of weight, and if tuberculin had been used their fate would have been very different. By ordinary methods these cases in Stage III. can hardly live one year—by means of tuberculin they may live in apparent health for many years.

With improvement the cough lessens and ceases, phlegm ceases, tubercle bacilli are not found, energy increases, and often there is also gain in weight and the appearance of health. But lest the enemy may awaken it is best to test all such cases from time to time, once in six months or once a

year, so as to be able to give further treatment with tuberculin if there is any indication of the enemy becoming active again. A reaction with tuberculin in doses 5 mgs., 15 mgs., 30 mgs., and 50 mgs. is such an indication. If the treatment has not been by means of tuberculin, tuberculin may also be used for the same purpose, but in the smaller doses used for the original test. In Stage II. the prognosis is quite good when tuberculin is used for treatment and at intervals afterwards to control the result. The danger is that the patient, after being well for several years, may think he can dispense with the testing by means of tuberculin. That over-confidence may be fatal. *It is a remarkable record that of all the cases in Stage II. treated with tuberculin since* 1902, *there has been but one death*, and that was due to this over-confidence. He relapsed nearly six years after treatment. My statistics show successes to the extent of over 90 per cent. Allowing for possible luck, I have little doubt that in Stage II. 70–75 per cent. of successful results can be obtained and maintained by means of tuberculin at least for many years. *In Stage II.–III. I have secured as good results with tuberculin as can be obtained through sanatoria in Stage I.* My records show quite 60 per cent. of permanent results at the end of 1907. Of course, when one knows the extent of the disease, it is little short of marvellous that these victims should now be enjoying apparently good health.

In Stage I., I agree with Professor Koch that pulmonary tuberculosis "can be cured with certainty by means of tuberculin." If I gave only the records of cases, numbering at least a hundred, which I am able to control after a full course of treatment, I should have to give a record of 100% successes. At least, I feel sure that by patience, courage, and the intelligent use of tuberculin in diagnosis, prognosis, and treatment infinitely better results can be obtained than by any of the so-called rational methods that are now in fashion. I am convinced that the degree of immunity depends upon the amount of tuberculin that can be given without causing any reaction, and accordingly I utterly repudiated Wright's views and methods when he published them, and clearly proved that in pulmonary tuberculosis the opsonic index could

not help one and might easily lead one astray. Medical men in Sydney imagined that I attacked Wright's system for mere perversity, but the point at issue for me was a very simple one, as I had an experience that left me no option in the matter. I had used tuberculin in the treatment of pulmonary tuberculosis with great success, and I found that the larger the doses were, the more permanent the results. When, then, Wright appeared as the advocate of tuberculin in minute doses *without even trying it in a single case of pulmonary tuberculosis* and his own enthusiastic disciples entered the field as the new-born advocates of tuberculin treatment in *pulmonary tuberculosis*, it was time for me to step into the arena and tell these mesmerised disciples that tuberculin in homœpathic doses, according to Wright's opsonic system, had not been proved to be of any value at all, and that, in fact, I had proved that tuberculin in large doses was an extremely valuable remedy in pulmonary tuberculosis. The logic of Wright's disciples in Australia left them open to attack, and, lest the failure of Wright's system might again put back the clock and cause tuberculin as a remedy to be again discredited, it was my bounden duty to express the conviction that Wright's system applied to pulmonary tuberculosis must inevitably lead to failure. My own successful results were due to large doses of tuberculin, and I had proved the value of large doses in hundreds of cases; Wright's system was essentially theoretical and problematical as applied to pulmonary tuberculosis and entirely incompatible with my practice, which had been very successful. At a demonstration of Wright's extremely ingenious technique for determining the opsonic index, the author failed to estimate the index, and afterwards I merely asked the question, "Is it worth while?"

CHAPTER IV

TREATMENT

(A) *Climate as a Factor, with special reference to the Climate of New South Wales.*

THE problem of selecting a suitable climate for various cases of pulmonary tuberculosis is constantly presenting itself to us in our daily practice, and its proper solution may after all defy all the enquiry and thought and discrimination that we bestow upon it. Each one has, in a sense, to work out the problem for himself, and as the x, y, z's have a varying value, exact comparisons are hardly possible. My own conclusions are so completely at variance with the current opinions of the hour--opinions firmly rooted in the minds of some of our leading practitioners, and more or less clearly reflected in the mind of the laity—that the time has come for me to give reasons for the faith that is in me; if so be we may thus at least gain a clearer insight into the complexity of the problem and realise that the vision of truth is still very far off.

In Europe, climate as a factor in the treatment of disease has been studied on more or less haphazard lines in the numerous sanatoria, watering places, and institutions that are scattered in all directions, but even at these places vested interests may militate against the discovery of truth; yet gradually, with the progress of time and knowledge, special climates have, by implication, been stripped of the great virtue that was once thought to be their exclusive possession. Accessory conditions, such as fresh air, sunshine, careful diet, graduated exercises, massage, and the continued use of mild drugs acting on different organs, are now claimed to possess the royal virtue originally credited to climate.

In Europe one observes a marked change of opinion as to the value of climate in the treatment of pulmonary tuberculosis. In Australia we have not even the experiences of sanatoria to help us. In spite of the existence of Mr. Goodlet's noble work at Thirlmere, a sanatorium at Echuca and at Mount Macedon, and one at Roma, we have very little positive relevant evidence on which we dare express any but a haphazard opinion as to the value of climate in the treatment of pulmonary tuberculosis. The cautious will therefore hold their opinions in reserve, and shun that dangerous form of dogmatism that is often rampant when truth is entirely beyond our reach, and logical methods are not equal to the task of proving our errors. On the nowadays all-important problem of the relation of climate of any or every sort to the treatment of pulmonary tuberculosis in the early stages, dogmatism cannot help us, and will probably lead us astray. As yet it is hardly possible to define precisely what we mean by the early stage of lung tuberculosis. I should like to restrict this term to the stage of the disease before tubercle bacilli are present in the sputum—when the tuberculous lesion is still closed or shut off from the external world, represented by the air passages. This definition would have this advantage, that medical men and laity alike would understand the form of tuberculosis under discussion, and would also have it firmly impressed on their minds that the disease may have possession of its victim months, and even years, before the obvious symptoms of the disease develop. At present many medical men and the laity consider that pulmonary tuberculosis of necessity causes fever, cough, and wasting. On the contrary, it is true that tuberculous disease of the lung may never show its existence by any symptoms, as *post-mortem* examinations continually testify, and in other cases may exist for a very long time without even attracting the attention of patient or doctor. It is not impossible that medical men often raise groundless fears about weak lungs, and on the other hand overlook the existence of disease that can be recognised by proper methods. If tubercle bacilli are found in the sputum, the stage is relatively late and the golden opportunity for treatment in the early

stage may be lost. In this early stage there may be no fever, no expectoration, even no cough. I have seen a case of closed tuberculosis involving a large part of the upper lobe of the right lung in a man who for years searched the globe in vain for the climate that would make him well. Physical signs were altogether out of proportion to his local symptoms. On the other hand there may be but slight physical signs till a hæmorrhage unexpectedly occurs, and too often the first stage rapidly passes into the second stage, usually with the signs and symptoms of secondary infection. Such cases are totally unlike in every respect, and yet they are both cases of closed tuberculosis till the hæmorrhage blots and blurs, and may ruin for ever, the pure type of the process. Otherwise such cases are cases of pure tuberculosis. Again, in tuberculosis involving the pleura, the first positive evidence of the disease may be acute pleurisy suddenly attacking a person apparently in sound health, and soon developing into pleurisy with rapid effusion and all the accompanying trouble and danger; or, on the other hand, there may be extensive disease of the pleura without any acute process, but ending in adhesions and thickening, sometimes running a latent course for a long time. This state, then, in which expectoration hardly occurs—and tubercle bacilli are absent from the expectoration—is the basis of classification for many, especially in the most modern sanatoria for the cure of tuberculosis among the poorer classes. If this stage is past the case is rejected (Weicker). As soon as tubercle bacilli enter the air passages and escape in the expectoration, there must be destruction and disintegration of the lung tissue at the surface, and then the risk of secondary infection is imminent. Till then the process is tuberculosis pure and simple. Then it is that the flood-gates are open, and organisms of various kinds, streptococci and diplococci mainly, may find a suitable nidus for growth and development in this disintegrated tissue, whence the tubercle bacilli escape. As tubercle bacilli leave, the organisms of septic processes enter by the door, and the whole character of the process is profoundly changed. No doubt this secondary infection that brings the process to the second stage may be

long delayed, especially in pure air, by conditions that we little understand, but once it is established fever shows itself, and on the chart this infection asserts its origin by the zig-zag course of the temperature curve. Anæmia, wasting, sweating, diarrhœa, and other symptoms may in the course of a few weeks demonstrate the severity of a twofold toxæmia. There may even be a degree of septicæmia. But even at this stage the case may not be hopeless. Change to a pure air in the mountains or plains—or at the sea-side—with other measures, may get rid of the secondary infection, and the tuberculosis making a pretence of slumbering for a while, may progress so slowly that it causes hardly any symptoms. The expectoration diminishes—even tubercle bacilli may disappear, and in favourable cases the lung heals. Thus the disease may pass from the second to the first stage. More often the expectoration is scanty, and some few tubercle bacilli can be found. The disease may seem to be in the first stage though it is in a latent second stage—ready at any moment to become aggravated. It may require all the bacteriological skill of the physician to recognise this latent second stage. Often this *latent second stage* is nothing else than the passive secondary infection of other authors. Even after considerable destruction of lung tissue with the formation of irregular cavities the process may regress, and in rare cases end in healing of the ulcerated surfaces. Again, the process may return to the phase either of a latent second stage or even of closed tuberculosis. For many, then, this state is the criterion. When the tuberculosis is thus closed, the first stage of the disease exists. I am convinced that if we can only provide a sanatorium for the treatment of these cases by the most modern methods[1] the public will have no reason to withhold its support from the philanthropic scheme that has been inaugurated for the treatment of tuberculosis among the poorer classes. I hope that I have given some precision to the otherwise loose and indefinite condition known as the early stage of pulmonary tuberculosis.

If my definition is accepted, I may state at once that we have no evidence for or against the value of this or that

[1] Tuberculin.

climate in pulmonary tuberculosis in the early stages. This basis of classification has not existed, and it is idle to suppose that any physicians in Sydney or Australia have investigated the effect of different climates on this stage of tuberculosis. But even interpreting the early stage of pulmonary tuberculosis in the ordinary vague and varying sense, we can hardly get nearer the truth, for no physicians in Australia have attempted to discriminate between the effect of climate on these cases as distinct from other cases. At present we are not in a position to form any opinion of our own, much less is there any ground for dogmatism. Most physicians diagnose tuberculosis of lungs by fever. That symptom alone, if of moderate severity, is generally sufficient to show that the case does not belong to the early stage of simple tuberculosis. In judging of the effect of climate upon the course of *pure tuberculosis* of the lungs; in other words, of pulmonary tuberculosis in the early stages, we must carefully put out of view the effect that is produced by climate, mountain or other, upon the second stage of tuberculosis, when secondary infection with fever, is the dominant and grave condition. Any line of reasoning I use, leaves out of consideration the disease in the second stage when secondary infection is so common. On this phase of the question I have already explained myself. (See *Australasian Medical Gazette*, August 20th, 1897). How then are we to form an opinion concerning the effect of any climate upon the first stage of tuberculosis of the lungs? As we have no trustworthy evidence of our own, we may learn something from other sources, and especially from the views of recognised authorities. Thus we may be in a position to form some general conclusions that will guide us in condemning or favouring the sea coast of New South Wales in dealing with the early stages of tuberculosis of the lungs. It is well to say at the outset that we have no evidence to show how the climate of the sea coast affects early tuberculous disease of the lungs. Those of the older physicians who may be inclined to dispute this assertion, hardly resort to the systematic examination of sputum for tubercle bacilli in their everyday practice. If they use this method, they use it when

it is hardly necessary. *Negative evidence*, unless obtained by the methods of the skilled bacteriologist, has no great value. Tuberculin as a diagnostic agent is not used at all, and the physicians, who cling to the old views, have neither the experience nor the knowledge necessary to determine the diagnosis of secondary infection. But my main objection is of a more serious kind. Whatever may be said to the contrary, it is certain that no observations have ever been made discriminating between the effects of the climate of the sea coast and the effect of the density of population in Sydney and Newcastle. The physicians in Sydney have attributed to the climate of Sydney and the coast effects that distinguish, and are a necessary accompaniment of all crowded populations irrespective of site or climate. It needs no argument to prove that Sydney, as a dense centre of population, will not show as good a record as other districts with sparse and scattered populations, but the effect is not at all the effect of climate, good or bad, but the effect of that crowding of masses and accompanying conditions of life that militate against health and specially favour the spread of infectious diseases, even of tuberculosis of the lungs. A fairer comparison may be drawn between Sydney and cities in the world of similar population. Such a comparison shows beyond doubt that so far as the incidence of tuberculosis of the lungs in the community is concerned, Sydney is one of the most favoured cities in the world, the incidence of tuberculosis of the lungs being less than 1 per 1,000, according to latest returns. It is at least a fair argument to use that the relatively low incidence of tuberculosis in Sydney must be due to some special favouring local conditions, and one might even state that the climate of the sea coast, mild and equable throughout the year, is a factor in this relatively fortunate result. But soil and situation are factors. Until it is proved to be otherwise, I see no other logical conclusion than that, were it not for the density of the population, Port Jackson itself would form an admirable site for a sanatorium. And this conclusion is strengthened when we examine the incidence of tuberculosis in districts that possess a climate like that of Sydney, but lack the crowded population. The

North Coast and South Coast districts will serve for such comparisons. These districts contain many small scattered towns, such as Grafton, Lismore, Kiama, Wollongong, Nowra, and others, and have an aggregate population of about 140,000. They show an incidence of one death from tuberculosis in every 2,730 living persons. New England, with a population of 63,300, shows an incidence of one death from tuberculosis in every 3,720; Namoi and Gwydir, one per 2,360; Young and Gundagai, one per 2,470; Bathurst, one per 2,180; Hunter, one per 1,830; Mudgee and Argyle, one per 1,800; Murrumbidgee, one per 1,300; Murray, one per 1,490; Western districts, including Broken Hill, one per 470. In England, which is *facile princeps* in the application of hygiene to the needs of the nation, and enjoys also the honour of presenting the lowest death-rate from tuberculosis among all European nations, in spite of the fact that it is sea-bound and has no mountains worthy of the name, in spite of its rain and wind and fog, its want of sunshine and its uncertain climate, in England the death-rate from tuberculosis is one per 737 living persons, in United States (28 larger towns) one per 400 living persons, in New South Wales, one in 1,200.

At the great German Congress on Tuberculosis it was stated that Buenos Ayres showed the lowest death-rate of all the large states of civilisation—1·62 per 1,000 inhabitants—one death per 630 inhabitants. Sydney is better than Buenos Ayres—one per 1,080 (1900)—and, therefore, in regard to its mortality from tuberculosis shows the lowest record of all large cities. The learned German knew nothing of Sydney's fortunate best on record. Buenos Ayres, with a population of 567,542, lost 1,876 from pulmonary tuberculosis in the two years 1892–3. The metropolis of Sydney in 1899–1900, with a population of 480,000, lost 926. Roughly speaking, there are three deaths in Buenos Ayres from tuberculosis of the lungs for every two deaths in Sydney from the same cause. Sydney alone of all the great cities shows a rate of less than one per 1000. It is worth while mentioning too that, comparing the largest cities in the world in which population exceeds 500,000, the towns on the sea-board show the lowest mortality from tuberculosis,

Buenos Ayres, Naples, Amsterdam, London, and Hamburg. Accordingly sea air in the northern hemisphere—even in the warm Mediterranean—in no way favours the tuberculous process. Even in the southern hemisphere Buenos Ayres is no exception. If, therefore, it is asserted that sea air in our region of the southern hemisphere brings disaster to cases of tuberculosis, we may surely ask for something more than assertion—we want proof. So far as I am able to sift evidence the proof is quite the other way. A simple analysis of the death-rate from tuberculosis in the districts of Sydney, which I take from the valuable and laborious commentary of Dr. W. G. Armstrong, Medical Officer of Health, confirms my views in the most startling manner. The combined districts of Vaucluse, Mosman, Hunter's Hill, and Lane Cove, with a total population of 10,575, had not one single death from tuberculosis to its account in 1899—a very remarkable record, showing at least that the sea air on the coast does not favour tuberculosis. Randwick, with a population of 8,225, shows only a rate of 0·24 per 1,000 in 1898, *i.e.*, less than 1 in 4,000, a far more favourable rate than exists in any country district. Manly also has a low rate.

If, then, we compare, not the districts of the wealthy, but the districts of the poor, we find that Balmain last year, with a population of 28,675 distributed with a density of 32 to the acre, has only a rate of 1·05 per 1,000. This favourable rate is a strange commentary upon the present views of the profession with regard to the injurious effects of the sea air and sea coast upon tuberculosis of the lungs.

Let us now turn to the coastal districts of New South Wales. In what way a climate, that presents a low general mortality, affects tuberculosis of the lungs it is difficult to say, but probably, if a climate is very favourable to general health, it will not be unfavourable to those suffering from early tuberculosis. The North and South Coasts present incomparably the lowest death-rates of all the districts in New South Wales—North, per 1,000 in 1897–9—7·1, 6·9, 7·9. South Coast, 6·8, 6·8, 7·7. No other districts approach these districts in the low death-rate. New England comes next with a rate of 8·2, 9·8, 10·1 per 1,000.

There is another thought worthy of attention. If there is one firmly-rooted conviction in the minds of all who have considered the question it is that predisposition plays an important part in the origin of pulmonary tuberculosis. Some exaggerate the importance of this factor, hardly anyone refuses to recognise it. We can hardly explain the nature of this disposition of tissue to invasion by the tubercle bacillus, nor have we a right to say that this or that nation exhibits a greater disposition to the disease. Yet, if we hold that predisposition shows itself in families or groups, we should be able to apply this view on a larger scale to nations. In such a case we might well argue that the British race exhibits less predisposition than any other race, because the British nation suffers less than any other European nation. The French nation suffers twice as much from tuberculosis, and Russia three times as much as Great Britain, at any rate in the large centres of population. As soon as we begin to argue that other conditions account for this difference we are at once knocking the bottom out of the view of predisposition. If, however, we accept the doctrine of predisposition as the first factor of importance, as some still maintain, our nation—the grand old British race—shows collectively a far lower degree of predisposition than other nations. Switzerland with its mountains and their vaunted power against tuberculosis shows an incidence of tuberculosis in the population of 2,031 per 1,000,000; England, 1,358 per 1,000,000; New South Wales shows an incidence of 806 per 1,000,000 souls. The conditions in New South Wales modify the incidence of disease among Englishmen and their descendants. In New South Wales every year there are 500 fewer deaths per 1,000,000 from tuberculosis than there are in England. Now if predisposition means anything it means some definite and permanent character of tissue. It can have no particular meaning if it can disappear in a generation. Experience seems to tell us that the disposition to tuberculosis is consistent with robust health and fine physical development. We are therefore not justified in believing that the conditions in New South Wales suddenly modify the predisposition of Europeans to tuberculosis. For no reason at all the idea

gained currency that the Australian born were more liable to tuberculosis than their English born parents. This is just a fair example of the readiness with which people indulge in reckless theories that rest on no foundation at all either of observation or fact, and may live as long as careful observations on the point at issue are wanting. There is hardly any more substantial basis for the view that the sea coast of New South Wales is not suited for cases of pulmonary tuberculosis. On the other hand we must not flatter ourselves with the notion that the mortality statistics from tuberculosis in New South Wales are vitiated by the intrusion of a number of imported cases of tuberculosis. In 1899 there were 1,069 deaths from tuberculosis. Of these no fewer than 600 were born in Australasia; 220 more had lived in Australasia twenty years or more; 165 more had resided in Australia ten to fifteen years; while only 58 had been less than five years in Australia. We are justified in saying that ninety per cent. of the cases had their origin in Australia, while sixty per cent. were among the native born. It would require a careful analysis of living persons who are native born and importations to show whether the native born show a greater or less disposition than the European. Now, if the predisposition exists in the same degree in the British race, whether imported or native born, we must hold that there is still a large contingent of persons prone to the disease. The disease has not yet attacked the full complement of predisposed. The soil is ready, but the seed has not been sown. In our community above all others, every effort should be made to prevent the disease extending its ravages to the full complement of predisposed. In short, prophylaxis will be of even greater benefit in Australia than in the older countries. Yet we are behind the world in measures of prophylaxis. There is not even a general hospital for consumption, which educated public opinion should urge the Government of the State[1] to establish, in order to diminish in some measure the numerous foci of infection that are a source of danger to those who live on terms of close intimacy with those suffering from the disease.

It is interesting also to observe the mortality rates in

[1] 1907. At last Government has awakened to the necessity for such institutions.

Victoria and Melbourne respectively, and in Queensland and Brisbane and compare them with those of our own State. In 1899 the death-rate in Queensland was 12·07; in Victoria 14·28; in New South Wales, 11·82. In Melbourne, with a population of 470,000 the rate of mortality from tuberculosis of the lungs has varied from 948 to 654, the average being above 700. In Sydney with the same population, the annual death rate from the same cause is 481 (1899). This excludes ninety cases that died in the Liverpool Asylum, of which the majority belong to the metropolis. Even allowing the rate in Sydney thus corrected to be 560, we find a difference greatly in favour of Sydney. The conditions of life in Melbourne kill, by tuberculosis, 180 to 200 more persons every year than the condition of life in Sydney. Is this an argument for protection? The same advantage is evident also in the conditions of life in the colony generally. Victoria loses every year from tuberculosis, 300 to 600 more than New South Wales, even though the population of New South Wales is greater. In Queensland and New South Wales the death-rate from tuberculosis is about the same, while Brisbane with its smaller and far more scattered population, shows a better record than Sydney, 1 in 1,490. Indeed relative to the population, the death-rate is better than that of the colony taken as a whole; if indeed the area of the city is the same for the collecting of mortality and population statistics. There may be some local conditions, such as the situation of institutions outside the city area, that would place these deductions from statistics in a different light. If, however, these statistics are correct, *Brisbane has an enviable position with regard to tuberculosis.* I know no other country in which the rate of deaths from tuberculosis is relatively less in its capital than in the remainder of its population from the same cause. So far as these figures serve us, we find that a low general mortality means a low mortality from tuberculosis; indeed, that the mortality from tuberculosis is an index of the general health of the community, and conversely we may say that when the general death-rate is low, then the rate from tuberculosis will be low. No country in the world can show a lower death-rate from tuberculosis than New

South Wales, and in New South Wales the coastal districts show a better record in this respect than any other district with the single exception of the New England district. *Even the New England district shows a rather worse record than the South Coast in respect to the incidence of tuberculosis of all forms.* But no other districts approach these districts in their relative immunity from tubercular disease. The coastal districts further show a far lower death-rate from pneumonia, bronchitis, and other diseases of the lungs. In 1899 there were ninety-six deaths from pneumonia and bronchitis in New England (population 63,290), but only thirty-four deaths in the South Coast (population 62,740), and forty-nine deaths in the North Coast (population 78,300). Diseases of the lungs are a good index of the variability of the climate. In no other district is the incidence of diseases of the lungs so small as in the coastal districts. These statistics undoubtedly seem to establish the fact that the climate of the North and South coasts is unusually propitious, and inasmuch as the incidence of tuberculosis in these districts is lower than in any other district I personally shall require further evidence before I give up the attitude I have long adopted in recommending strongly the coastal districts of New South Wales for the treatment of tuberculosis.

I wish, therefore, to put forward the following proposition : " That the air of the sea coast of New South Wales, far from being injurious to those suffering from pulmonary tuberculosis in the early stages, is beneficial, and may have as much curative virtue as any other climate in the world." This proposition is well worthy of discussion and of a collective investigation by the local branch of the British Medical Association.

I have shown that in this coastal climate there are fewer deaths from tuberculosis per 1,000 than in any other part of New South Wales. In 1897 there were only twelve deaths from tuberculosis in the population of 78,300 in the North Coast district. The conditions, therefore, cannot favour the disease nor hasten its course. The mild equable climate, free from great variations in temperature, and with a cool, refreshing north-east wind from the sea supplying relatively germ-free air during the hotter months, from January to April, and

during the cooler months with winds that are less severe and more propitious than the cold, piercing winds of the mountains, with a mean summer temperature of 70° to 75°F., a mean winter temperature of 53·5°F., and a mean annual temperature of 63·2°F.—such meteorological conditions would *a priori* lead one to anticipate a favourable effect, while experience tells us in plain and unmistakeable language that these conditions cannot be bettered anywhere in Australia, if in the world. No doubt we must be ready to meet and dispose of special pleading. My views are heretical, and, as happened in the days of the Inquisition and Gallileo, such views can only win the day on the transparent basis of truth. I court inquiry; I seek for full investigation. Even if my views should prove in the end to be exaggerated or unsound, the investigation will give us a more excellent view of truth. I have no other interest in this matter than to seek and find the truth. *Prima facie*, my views, supported by the evidence of statistics, bear the semblance of truth. Until numerous facts and observations are collated and given their proper place and order, truth cannot be established. At present we have no evidence to the contrary that is worthy of the name, no evidence that would pass muster in a court of law, or even of reason, and I throw down the gauntlet as the champion of the new view that the sea coast of New South Wales is not unsuitable for the treatment of pulmonary tuberculosis in the early stages or even in the later stages. I have suggested that the low mortality from tuberculosis in Sydney and the coastal districts is evidence in itself that the conditions obtaining in these parts are of advantage in the treatment of tuberculosis, and should be exploited. There are those who even now hold the opinion that the low rate of mortality from the tuberculosis in Sydney and the coastal districts *is*—not may be—in part the effect of medical and lay opinion in condemning these districts. As a consequence of this condemnation, so these special pleaders argue, those who suffer from tuberculosis in Sydney and near the coast pack up their portmanteaux and hie to the mountain districts or the plains, where they die. Thus Sydney presents a more favourable record, and the districts of immigration present a

less favourable record than the conditions of the respective areas would justify. Such emigration and immigration, however, should have the result of showing a greater number of deaths from tuberculosis in the country districts than would be found in other places and other countries of similar populations. On the contrary we find that the death-rate from tuberculosis in the country districts of New South Wales is not only not high but exceptionally low. I have also the support of Dr. W. G. Armstrong is saying that so far from the death-rate in the country districts being increased by this exodus of tuberculous cases into the country, there is on the contrary an exodus from the country districts into the town. Dr. Armstrong writes: "Very many persons who died in Sydney hospitals came from country districts. Some few deaths of Sydney residents have occurred in country hospitals, but," he adds, "the number of deaths occurring in the country which ought to be credited to the metropolis is, however, certainly insignificant." This may or may not apply to tuberculosis, as the statistics are purely the statistics of the Sydney hospitals. One may say that many go, or are sent to the country in the earlier stages, but few remain there to die. They seek the country, too often in vain, for a cure, but that failing, they return home to die. Certainly my own experience tells me that many of the worst cases I have seen have lived all their lives in the districts with vaunted climates. From the west I have had many cases; from the North Coast, none; and but a single case from the South Coast. Now it is to work out accurately the problem in this direction that we should have a collective investigation. The investigation could be easily carried out by drawing up forms to be filled up by medical men in the various districts. The forms should be drawn up in such a way that the country practitioners should be able to state the number of cases that have *died* under their care, the birth-place, length of residence in the district, and such other information as will show the incidence of the disease among those who belong to the district and those who have come from other parts, especially from Sydney and other coastal towns or districts. It is very remarkable that the districts in

the immediate neighbourhood of Sydney should present such favourable records. One would expect that residents in these districts would be more often exposed to the risk of infection by the mere proximity which facilitates intercourse with the population of the dusty and crowded city. Yet in one year the North Coast, including Grafton, Lismore, Ballina, Maclean, Bellingen, Port Macquarie, Kempsey, Casino, Taree, and other places, had but twelve deaths from tuberculosis in the whole district. There is at present not much to support such special pleading. However, until we have the results of a collective investigation such as I propose, I am absolutely justified in holding the opinion that the district of the sea coast is as suitable as any other for the treatment of pulmonary tuberculosis. A sanatorium for the poorer classes should be in situation within reasonable access of Sydney for many reasons. It can be built and maintained at the lowest possible cost. It enables the friends and relations of the inmates to visit the sanatorium without much cost. This will minimise the sense of isolation that may otherwise be a bar to the prolonged stay in the institution that is indispensable for satisfactory results. A happy state of mind contributes to the success. Most important of all, there can be thorough supervision, even from Sydney. Of course a resident medical officer is indispensable, but I should hope that as in Vienna and elsewhere a central committee of medical men, of special knowledge and experience, should control and guide the principles of treatment in the institution. These are immense advantages that cannot be lightly set aside, because certain individuals, medical and lay, have the notion, formed from their own personal experience or from a limited and indiscriminate experience, that the mountain air has specific virtues. I grant that there is abundant testimony from the older physicians in favour of treatment at higher altitudes. But the pendulum of opinion swings now in this now in that direction. Not very long ago the older physicians firmly believed and taught that consumption was more easily cured in a warm climate than in a cold one. There was no foundation for this view, and accordingly cold climates in time came into fashion. Patients were ordered to go to the Andes

instead of Madeira or Egypt. Yet Koch tells us that tuberculosis plays no part at all in the mortality among inhabitants on the east coast of Africa—in the heat of the tropics where malaria reigns supreme. The great pioneer of the rational method, Dr. Brehmer, of Goerbersdorf, strongly advocates mountain air on account of the diminished atmospheric pressure, of the lower degree of moisture, of the great warmth of the sun, of the intense sunlight, of the rarity of fogs, and the purity of the air, especially its freedom from microbes on account of the sunlight. Sir Herman Weber admits that it is difficult to prove therapeutic results by statistics. He has seen 36 out of 144 cured—a misleading term—by mountain air. In 52 of the cases no improvement or downward progress to death. Better reports have appeared, especially those of Dr. Turban, but Dr. Turban and others have also adopted the rational method, based on dietetics, physical exercise, and plenty of fresh, pure air. Dr. Turban, too, has spoken favourably of tuberculin treatment in his institution. No authority of any eminence claiming the specific virtue of mountain air, has ever attempted to differentiate between the effect of the mountain air and the effect of the numerous measures that constitute the rational system of Dettweiler, Dr. Brehmer's pupil and successor. Sir Herman Weber, who is the special advocate for mountain air, says that in cases of circumscribed lesions almost all climates can be exploited with good effect, though (he adds) in robust constitutions especially mountain climates and sea voyages. After pointing out indications for special climates, in which one observes that he highly commends warm climates with plenty of sunlight, he adds "The cure of tuberculosis in the early stages is possible in all healthy climates." Listen to this peroration: "But climate alone, without careful medical supervision, is for the most part unsatisfactory. The blind faith of patients in climate leads often to failure, to progress of the disease and death. Therefore, for the majority of patients, treatment in sanatoria is to be preferred, but for the poorer classes is a necessity. The erection of sanatoria for the people is a national necessity for

the cure, the prevention, and the eradication of tuberculosis." Even Sir Herman Weber places greater virtue in the methods of the sanatorium than in any climate.

In the latest text-book on medicine we read: "It is only during the winter, however, that the climate of Davos or of the Engadine has special advantages." In the still dry air patients can bear the great cold. Appetite is stimulated, and much food can be taken. The climate of Nordrach is by no means a climate that has any special advantages, if we accept the account of a physician who was cured at this famous sanatorium.

Dettweiler, disciple of Brehmer, is the great apostle of sanatorium treatment on hygienic, dietetic, and physical principles. The disciples of this, the latest school, in the treatment of consumption, set but little store on the effect of climate, whether of mountain, sea, or plain. To use Dettweiler's own words, "This new method consists in curing "the disease by the method of rest and open air carried out in "its smallest details according to the strength of the patient "and the stage of the disease, even during the night." This systems renders it possible to treat consumption in all climates free from extremes and even allows patients to remain at home. In detail, it consists of hardening the skin by proper use of air, light, water (baths, douches), moist and dry friction, massage, in gymnastic exercises of the chest and whole body, by climbing, &c., in a systematic application of all physical remedies, in exercise or the reverse, according to the state of the patient—a system gradually and carefully adapted to circumstances in a manner that only a special institution can apply and control. This constitutes sanatorium treatment on a rational basis, that claims superiority over all other methods. Thus, "the disease can be healed in all good climates with equally good results." This is the open-air treatment. It would not be right to call it open-air treatment if certain climates were necessary. Even some of our physicians loudly talk of open-air treatment and recommend it, and yet cling to the older view that the air of the mountains is necessary. Worse still, they argue that the climate of our coast is not suitable for this method. Those

who preach this gospel have but imperfectly understood the new teaching. If one advocates the open-air treatment, one must follow those whose experience has demonstrated its value, independent altogether of special climates. We entirely misunderstand the experience and teaching of the new school if we say that a sanatorium for the people should be erected in the mountains rather than near the coast. In my limited experience I should hesitate to send patients to our mountains, with their variable climate, their mists, and their cold winds. A few robust men and women might do well. I doubt if the mountain climate would suit the majority as well as the mild climate of the coast. Turban, whose sanatorium is at Davos, writes that Dettweiler has especially accomplished the great task of demonstrating that the carrying out of the open-air method is not bound up in any special climate. Even the increase of the red corpuscles and of the hæmoglobin value of the blood—with increase of volume—such as may be the result of high altitudes, is after all a compensating process with a physiologicai explanation. The most modern sanatoria have not been erected at high altitudes. Falkenstein, Hohenhonnef, Nordrach, Alland, near Vienna, the sanatoria near London, Edinburgh, and Liverpool, in effect disregard the value of mountain air. Dr. Sydney Jones has himself mentioned statistics from open-air treatment near London, which we may strive to imitate but can never excel. Dr. Sydney Jones stated that 75 *per cent. had been cured.* They will never record such statistics again. Yet the climate of the sea coast of New South Wales is as favourable as that of London ; I should say far more favourable. I could quote authority after authority, emphasising the great value of sanatorium treatment, and by implication paying little attention to mountain air as an appreciable factor in the new treatment of pulmonary tuberculosis. The new treatment consists essentially in open air, dietetic regulations, and physical training. Of quite subsidiary importance is the air of the mountain, of the sea, or of the plain.

I might even, with reason, venture to discuss a second proposition, viz., that the climate of the mountains near Sydney, Katoomba, Mount Victoria, &c., is not as suitable as

the climate of the coast for sanatorium treatment, but I wish chiefly to test the truth of the firmly rooted opinion that the climate of the sea coast is not suitable. If we arrive at the conclusion that this climate is eminently suitable for a sanatorium for consumption, there are very great obvious disadvantages in erecting a sanatorium on the mountains. The want of a good water supply, the difficulty if not impossibility of keeping cows for the all important milk supply throughout the year, the difficulty of growing vegetables and fruit trees, which would in other places offer good and pleasant occupation to the inmates of the institution, are among the most obvious disadvantages. Hence the selection of a site in the mountains can only be justified when it can be shown that these disadvantages are more than counterbalanced by the virtues that have not yet been proved to exist in the air of the Blue Mountains.

Long established error is often a vigorous weed that blights every healthy growth, and can only be uprooted by dint of much labour and patience. If error is closely intertwined with vested interests or personal reputations, it may be difficult to kill the parasite, but the demands of truth and the public interest far transcend in importance either vested interests or personal reputations. Otherwise, the fallacy, if, indeed, it be such, that now possesses the public mind, might well be left to die without a murmur—without a regret. If my views should ultimately win the day on the eternal basis of truth, the public gain will be great. Many an unfortunate victim may still have to bear his disease, but he need not suffer exile in strange and distant places, where he is bereft of the pleasures and comforts of society, even of the necessaries of a healthy and happy life. Surely, in the interests of the whole community, we should lose no time and spare no energy in discovering, by a collective investigation, whether I am right or wrong in saying that neither experience nor facts justify the profession and laity in perpetuating a gross libel upon the beautiful and health-giving climate of the sunny coast of New South Wales.

1908. *The Aftermath of this Article, written in* 1901.

The preceding article was published in 1901 and aroused a violent storm of indignation and opposition. The leader of this opposition promised to deal with the subject at length in order to show that my statistics were faulty and my conclusions utterly unsound. I have waited in vain for seven long years for a rebuttal of my evidence. In those seven years the majority of the profession have been converted to my views, and many doctors, who used to send their patients to the mountains and into the country, now send them to the district near Sydney where I established my own private sanatorium. Moreover at this sanatorium I have had results which place my original contention beyond any doubt. In 1905 I had forty-five cases of pulmonary tuberculosis under treatment in this climate from November, 1904 to April, 1905. I specially selected the very worst months of the year for my observations. During these months the atmosphere is apt to be close and enervating, and sudden changes of temperature are frequent; favouring chills, and depressing vitality. Of the forty-five cases under treatment, three lost a few pounds. One of these was in the third stage, but it was a loss of but 3 lbs. The other two, who lost weight, lost their disease also—to all appearance—so that no great harm was done. In the remainder—forty-two cases in all—there was an *average* gain in weight of *eight pounds*. This extraordinary gain in weight in spite of disease at a time of year when most people lose both weight and energy in a climate heartily condemned by the medical profession of Sydney, was no doubt largely due to tuberculin, but at least we may say that the conditions of climate were not adverse. However, if the medical profession admit that tuberculin was the effective cause of the great improvement, I am quite content. If they have been converted to my views regarding the healing virtues of tuberculin they will in time be converted to my views upon the health-giving virtues of the sunny coast of New South Wales.

(B) *Treatment by means of Sanatoria without Tuberculin.*

Nowadays the air rings with the praise of the sanatorium in the treatment of pulmonary tuberculosis. The great German physician Brehmer suffered from consumption, and cured himself at Goerbersdorf. He introduced a new system without entirely breaking away from the teachings of Hippocrates, Galen, and Celsius. He wrote in 1854, "Pulmonary "consumption is curable; it is curable under suitable climatic "conditions (mountain air) by a hygienic-dietetic system of "treatment carried out in an institution." His new system was ridiculed as a fad, but his masterful energy and supreme confidence won slowly, and in spite of much opposition, the consideration and approbation of all. His chief disciple, Dettweiler, elaborated the system of Brehmer, and added to it "Rest" as an essential factor. Thus rest, abundance of pure fresh air, energetic feeding, strict management of the daily life of the patient under the watchful eye of a specialist, hardening of the system by living in the open air, and hydropathy constitute the essentials of Brehmer's system.

The theories upon which Brehmer based his methods are forgotten, but his practice showed that pulmonary tuberculosis might be cured, not by climate *per se*, but by sanatorium methods carried out in any good climate free from extremes. Many a consumptive may therefore be treated with success in his own home. It is equally certain that this method of treatment cannot be applied to the poor in their own homes. The poor therefore cannot be treated except at the sanatorium. Moreover, it is daily becoming apparent that a residence of at least six months in the sanatorium is necessary. Thus the system is in its essence costly and therefore beyond the reach of the majority of the poor. If we consider the prevalence of the disease among the poor, none but a visionary can suppose that such a costly method can help the majority. As a means of bringing respite, and occasionally cure, to those who have money and time at their disposal, the system may commend itself, but I am persuaded that hundreds and thousands have learnt to their disappointment and sorrow

that even under favourable circumstances the sanatorium has its conditions and limitations. Sanatorium methods generally effect immediate improvement, and if the patient can spend some months each year under these special conditions, his life will be prolonged by some years, and in some cases the disease will be even completely cured.

Sanatorium treatment is not open-air treatment. Sanatorium treatment is certainly not the treatment recommended by the mass of medical men in Sydney. There is a strange unreasonable and unwarrantable prejudice against the sea air of New South Wales, which leads every doctor in Sydney to think that he has solved the problem of appropriate treatment for consumptives if he despatches them into the country to get away from the sea air. I have already dealt with this current prejudice, and have but to add that, in my own experience, I have found the air of the sea coast of New South Wales to be no unfavourable factor in the treatment of consumption. This haphazard system of sending into exile all who suffer from consumption is not sanatorium treatment. Sanatorium methods can be carried out quite as well within twenty miles of Sydney as in the Blue Mountains, or at Hay or Wellington or Orange or Bathurst or Armidale. I have had very serious cases of consumption occurring among the native born of these localities, and if the climate will not prevent the disease, it is not likely to be a powerful factor in curing it. (See Appendix.) Medical men in Sydney often pack off their patients to the country without any instructions. They do not even tell them to take their temperatures lest they should get too fond of taking it, as if it were some fatal, fascinating beverage. The importance of "rest" is ignored. They are merely told to live in the open air—in a tent—on a verandah. Few patients, however, have the luck to escape drugging, especially with creasote *et hoc genus omne*, and many of them afterwards suffer from indigestion and a ruined digestive apparatus. "Stick to creasote" is the reiterated advice of the physician who has not tried the effect of the drug upon himself. "Creasote" drugging was no part of Brehmer's system, and it is hard to understand on what evidence creasote has become so popular. Creasote in

sufficient doses may be a germicide, but on no mathematical calculation can it be shown that creasote taken into the stomach in drop doses and absorbed can in any way injure a single tubercle bacillus. Yet those who do use it seriously imagine that in some extraordinary way these few drops distributed through the body in a state of enormous dilution may lessen the activity and toxicity of millions of tubercle bacilli often lodged in tissues and passages far removed from the remote chance of even momentary contact with the drug. As an antiseptic or germicide, creasote given by the stomach must be inert when it reaches the lungs. If thus diluted it can affect a tubercle bacillus, I forbear to think how, in a pure state, it may affect the mucous membrane of the stomach. I have records showing that the use of creasote has been disastrous; yet some physicians have no scruples in applying this strong caustic directly to the delicate structures of the stomach wall for months and years. Thus Brehmer's system means something more than open-air treatment, and certainly stops short of creasote treatment. What a shocking burlesque of Brehmer's system our local physicians practice. They bustle off their patients into the country, so that they may escape the pernicious influence of the health-giving climate of the coast, and enjoin them to suffer the heat and dust and discomfort of the Australian bush, as well as the unsuitable food and bad cooking inseparable from it—provided only they take enough creasote to ruin their digestion for all time. A few may survive the horrors of such exile—many revolt—and all would do far better in a decent climate, with decent food and cooking, and in decent society. No physician practising these antiquated methods has dared to publish the results of his experiences, and yet they have dared to criticise my methods and my belief in the suitability of the sunny climate of the coast of New South Wales for the treatment of pulmonary tuberculosis. There has been much glib talk about sanatorium methods, as though sanatorium treatment had been demonstrated to be the only and the best treatment. In fact, the sanatorium for the poor at Wentworth Falls is the outcome of this glib talk. It appears to me to be somewhat of a paradox that this system,

which is claimed to be indispensable for the poor who suffer from consumption, is not equally necessary for the well-to-do. Our self-appointed authorities in Sydney have exploited the charitable instincts of the wealthy and well-to-do, in order to provide sanatorium treatment for the poor, but do not consider it worth while to provide their well-to-do patients with those very means of treatment which they consider indispensable for the poor. If I had unbounded faith in the power of sanatoria to cure consumption, I should have urged long ago the establishment of sanatoria for the well-to-do. With our physicians in Sydney, a trip to the country is the panacea for their well-to-do patients—for the poor there must be sanatoria. It is quite clear to me that, as our physicians do not advocate sanatorium treatment for their rich patients, they should find it hard to convince their rich friends that sanatoria play a very important *rôle* in the treatment of consumption.

The treatment of pulmonary tuberculosis by climate has had a long trial. Hippocrates believed in change of air; Celsus recommended climate, sea voyages, and the inland climate in summer; Galen sent his patients to the mountains and recommended the milk cure. Many of our physicians mark time to the ideas of the ancients, and the value of these haphazard methods is written large in the black letters of the statistics of every country. Climate *per se* as an important factor in the treatment of pulmonary tuberculosis is now being discredited and discarded in the countries of the Old World. Else, how does it happen that sanatoria are being built in every sort of climate in England, Scotland, America, and Germany; while France, of all European countries, stands almost alone, for she has hardly deigned to consider the wisdom of spending large sums of money upon sanatoria for the poor. One of our authorities in Sydney boasts of the value of mountain air with one breath, and in another breath praises the results of sanatorium treatment in a hospital near London, where there are certainly no mountains. He even stated at a public meeting, at which I was present, that "75 per cent. of the cases at this sanatorium had been cured." I at once retorted that, if this were true, we should seriously think of sending our patients to this sanatorium, for we could

not hope to obtain such results. Such statements are not only grotesque, but cruel and dangerous to the cause he advocates. High authorities even contend that sanatorium treatment is independent of any climate, and should be carried out as far as possible under those very conditions of climate under which the patient has to live his life and earn his livelihood. This manifestly applies to the poor artisan, many of whom can barely afford to live in their own homes. For such "hewers of wood and drawers of water," who form the largest section of society, sea voyages or constant changes to the country or mountains are mere dreams that can never be realised, because they cost both time and money. Yet many a time a lodge doctor advises his lodge patient to take a sea voyage or spend a year or two in the country.

In discussing the value of sanatorium methods, I do not desire to estimate the effect upon those well-to-do people who can devote many months in every year, and even a life-time, as well as a fortune, in an earnest search for health—too often a "Will-o'-the-Wisp"—by means of sanatoria. Those who have the time, the money, and the patience to give to this object deserve every encouragement, and may be successful in their quest, but there are hundreds and thousands who have been sorrowful witnesses of the elusiveness of such methods. For a year or more there is apparent success which deceives everyone, even the physician. A relapse occurs with a recovery not so complete, and thenceforward the progress of events is ever downwards, perhaps still with some short respite, until even the physician has to despair where a few short years before hope had reigned triumphant. There are tens of thousands of people every year who have been eye-witnesses of these sad family histories. There is some consolation in the thought that faith in sanatoria, and pertinacity in exploiting its methods, may bring a good reward to 30 per cent. or 40 per cent. of those who trust this remedy. But it is surely no trifling matter that failure is stamped in big letters upon 60 per cent. to 70 per cent. of the cases that trust to sanatorium methods. At all events the great majority of the sufferers have not the means to indulge

in this costly method of treatment. *The virtue of the system is not great when it can be applied only to a very limited number and fails in* 60 *per cent. to* 70 *per cent. of the cases.* It is no exaggeration to say that in England there are at least 150,000 persons who need succour. If their only hope of salvation lies in the exploitation of sanatorium methods, their doom is sealed. No scheme for the exploitation of sanatorium measures can cope with this colossal task. If anyone replies that they cope with it in Germany in this way, I can only request those who hold such a view to study carefully and in detail the colossal work of the national insurance societies of Germany—their concrete objects and their results as published in the analyses of Engelmann and Hamel and Weicker. They will rise from the study of these great works both wiser and sadder men.

All must admit that it is very difficult to consider dispassionately and estimate accurately the value of any system of treatment in a disease so variable in its nature and course as pulmonary tuberculosis. The erroneous idea of olden times that consumption is the pitiless effect of some constitutional taint tended to encourage a fatalism—a very lethargy of ignorance and indifference—that has paralysed all rational effort for generations. Even now the medical profession seems but half awake, and medical men, who dogmatise upon the great benefits to be derived from sanatorium methods without a clear grasp of what such methods can do and cannot do, are but blind men leading the blind. A peculiar mental complacency and mental inertia may even afflict medical men. Would that one could shake authorities out of this deadly complacency so that they might at least carefully study the meaning and results of the economic and rational methods of the sanatorium, as applied to the poor, before they applaud and vaunt their efficacy. I should be the last to disparage or condemn the methods of the sanatorium merely because they were not perfect, but it is high time that, in the interests of truth and humanity, its shortcomings and failures should be as clearly recognised as its advantages and its successes are freely advertised.

Sanatorium methods are faulty and uncertain, and therefore

it is our duty to seek and find a way to better results. Even the laity has come to trust in the efficacy of sanatoria upon the somewhat glib assertions of medical men, and it is neither politic nor fair to mislead the laity upon such a vital national question. Hundreds and thousands of witnesses in England alone can testify that the sanatorium system, when put to the test on the confident advice of medical men, has signally failed.

Temporary results are common enough, but such results often follow no treatment at all, but permanent results are not by any means easy to secure. It is to me an awful mystery how any physician of wide experience in this disease can venture on the statement that the disease is curable; indeed, some physicians would almost persuade us that it is easily cured. It certainly would be easily cured if residence in a sanatorium even for six months would cure it. Many a patient is told that if he lives in the country or lives in the open air all will be well with him. Those who talk thus must rue it some day, for the germ of tuberculosis is particularly hardy and resistant and not so easy to kill, though it may remain dormant (quiescent, latent) for many years. The activity of the germ in tissue is such a variable quantity that no one can speak with any certainty of "cure," unless it has been shown by means of tuberculin that there is no evidence of such a dormant or latent tuberculous process. If, therefore, tuberculin is not used, we must remain in the dark as to the true state of the patient. *Further, these temporary improvements are so extremely common that we must discard altogether the evidence of results that have not been subjected to the test of time.*

The sequel to these temporary improvements—temporary arrest of the disease, &c.—is but a long, wearisome, disappointing chapter of relapses, terminating sooner or later in death—certainly in the majority of cases. We have a sanatorium in New South Wales supported by voluntary subscriptions, and at the end of each year the results of treatment are tabulated. Such results, neither controlled by test doses of tuberculin some months after, nor subjected to after-examinations, are absolutely worthless as evidence of the

value of sanatorium methods in curing the disease. Yet these are the records which the medical profession allow to mislead people. When I proposed that each year the cases treated in this institution should be examined in order to see whether the improvement was maintained for one year, two years, three years, &c., the authorities would not hear of it. Further, it is a rule of the institution that no patient can have more than one course of treatment. This also militates against any accurate valuation of the methods of the sanatorium. *Without such after-examinations, it is impossible to give a genuine value to sanatorium methods.* Many other diseases run a latent course, notably hydatid disease, even appendicitis. Because hydatid disease exists but progresses so slowly that it causes no symptoms, because appendicitis does not immediately cause severe symptoms, and runs a latent course, does the surgeon consider the disease to be arrested or even cured. Yet such a term is verily applied to so-called quiescent forms of tuberculosis, even when a test dose of tuberculin would reveal anything but latency. Accordingly, though the disease may be described by our sanatorium authorities as "arrested," in another few months, or later still, the disease may be rampant.

Often, indeed, all the improvement obtained by the sanatorium methods has been the abatement of the mixed infection, the specific agent of tuberculosis still lying in the tissues, little affected by the agencies of the sanatorium system. It is this abatement of a mixed infection that deceives the physician and tempts him to dream that the tuberculous process has abated. Again and again this temporary improvement is but a prelude to very bitter disappointment. *In the sanatoria for the poor there are very obvious reasons, apart from the mixed infections, for these temporary improvements.* Those who enter the sanatoria have been manfully maintaining a severe struggle for mere existence, working eight, ten, or more hours a day, ill-fed, ill-clothed, ill-housed, ill-attended. Is it a great miracle that a man who has been working from morning to night, living on bread and dripping, and breathing the air of a small, badly-

ventilated room, should improve as soon as he rests all day on a lounge, is fed every day on the best of foods, and breathes the beautiful air of the mountains or plains? Of course, he gains weight, and strength, and energy. But he may do all this and yet the disease itself persists—ready at any time to renew its attack, and slowly emaciate and exhaust the victim. Such temporary results are far from satisfactory, and, the world over, men have allowed themselves to be deceived by these transient successes until the inquiring mind of Germany—especially through the Imperial Board of Health (Engelmann and Hamel) and Weicker (the pioneer of a scheme for dealing with the disease among the poor)—set to work to inquire into the after-history of cases that had been treated in sanatoria. These after-examinations, made at intervals of one and more years after the patients have left the sanatorium, have shown that year by year the improvement vanishes, and at the end of four or five years after sanatorium treatment 50 per cent. to 60 per cent. of the persons so treated are dead of pulmonary tuberculosis. In Germany such after-examinations are the easier because the enforced system of registration of domicile helps one to track out the cases. In England and elsewhere the investigations would not be so simple, but until such investigations be carried out systematically we can but acquiesce in the conclusions based upon such investigations in Germany.

In 1903 I pointed out that English experience could not help us in determining the value of sanatorium treatment, because the sanatoria for the people had not existed long enough to supply the facts, and there had been no attempt at after-examinations. My assertion was scouted in 1903; in 1905 Dr. Ransome made the very same statement. Surely what was true in 1905 was doubly true in 1903. The whole gist of my argument is that we must have recourse to the experience and results of the People's Sanatoria in Germany if we wish to form a sound judgment upon the value of sanatorium methods; and even the experiences and results of these German sanatoria may lead us woefully astray unless these results have been checked and controlled by systematic after-examinations.

Let me repeat that I am discussing the value of sanatorium treatment for the poor. For well-to-do people sanatoria do some good, sometimes much good, prolonging life rather than curing the disease. Such people can pay for the best advice, and it is their own fault if they do not purchase the best article. So long as fads, and fancies, and prejudices exist people may pay for them, and it is not possible to protect people from self-deception or imposition. This man abhors the idea of having a needle thrust under the skin; that man fears the injection may give him a disease; another hates new-fangled notions; the imagination is given free play, and unheard-of possibilities answer to its magic call. With the poor man there are no such difficulties. His faith is simpler and truer. As his health is the potent factor in maintaining the integrity of the family and home, he is ready to trust those who know far more than he does, and I have found that he is even ready to take risks. The conservative gentleman is fastidious, case-hardened in his prejudices, and "would rather bear those ills he has than fly to others that he knows not of." It is surprising, too, what an amount of idle and irresponsible chatter there may be among laymen upon the value of any system of treatment. It took me fully five years of laborious study and work to form an opinion on the very difficult question of the treatment of consumption. A layman is ready to come to a conclusion because he had heard that some friend of his had been cured by this method or that method. The poor man has a greater respect for constituted authority, and is less often the victim of humbugs than the intelligent and cultured gentleman. Of course, the humbug lays himself out for the wealthy, and ignores the poor for obvious reasons. I have abundant reason to be thankful to the poor, for they gave me my opportunity. Again and again the rich rejected my advice, and some have suffered. The poor took risks in the hope of better things, and my records embrace a very large number of individuals in the humblest stations in life. These records may be allowed to speak for themselves.

In Germany sanatoria for working men exist on an economic basis from the outset; not on a sentimental or

humanitarian basis. By wise social legislation the artisan is compelled to insure against sickness, and the great Imperial Insurance Societies of Germany have a direct interest in maintaining the industrial efficiency of the working unit, and, perhaps, at the same time the fighting efficiency of the unit. By this system of State-enforced, not State-supported, insurance against accident and sickness, the individual workman is compelled to pay to the General Fund to which also the masters or employers contribute. Thus those who receive the benefits of these Insurance Societies—and sanatorium treatment is one of the benefits—have also to pay for it, at least in part. Every domestic servant must insure in the National Societies of Germany. These Societies, in dealing with pulmonary tuberculosis among wage-earners, devote much thought and time to the detection of the *disease in its initial stage*, because this is the most economical way of dealing with the disease. In about 65 per cent. of these cases, the disease is so early that tubercle bacilli are not to be found in the sputum, and in such cases the diagnosis is made certain by means of test doses of tuberculin. In N.S.W., hardly a case enters the sanatorium that does not show tubercle bacilli in the sputum, and, as I know to my chagrin, if a case does not show the presence of tubercle bacilli in the sputum, it is likely to be rejected. One such case of mine, in which I had already discovered tubercle bacilli in the sputum, was rejected after I had selected this very case for a clinical lecture on the early diagnosis of pulmonary tuberculosis. Accordingly our own sanatorium at Wentworth Falls is dealing with a less favourable class of cases. Indeed, under our present social system, the treatment of pulmonary tuberculosis *in an early stage* (according to German and not English or Australian ideas) is almost impracticable, because poor men, receiving daily wages, will not give up work when the disease is in such an early stage that it hardly affects their general health, and does not seriously impair their industrial efficiency. In Germany they manage these things better and compel the workman to seek treatment in the sanatorium as soon as the early signs of disease are detected, especially by means of tuberculin. One of the worst, the most dangerous and most

treacherous features of tuberculous disease of the lungs, is that often a man can work quite well, even when the disease has reached a relatively advanced stage; even when the disease is in the third stage the sufferer may be an efficient workman according to the German definition. It is a serious matter of national importance that there are no enactments on the statute-books of England or Australia that compel a workman to insure against the day of sickness. Friendly societies on a voluntary basis cannot pretend to deal with the treatment of consumption among the poor—at any rate by means of sanatoria. When the poor man ceases to work his income ceases and domestic misfortunes begin. Moreover, once pulmonary tuberculosis has reached the home of a poor man, this disease, like a vampire slowly spreading its wings, casts its blighting shadow in ever-widening circles. The father or the mother may be the first victim; and sooner or later other victims become enmeshed in the toils of this deadly family scourge. While the man, upon whom the family depends, can work, he will and must work, and in England and Australia more often than not the disease has passed beyond the hope of any remedy before he yields to the necessity of trying sanatorium or other treatment. So with us sanatorium treatment cannot be expected to yield results equal to those obtained in Germany, and even in Germany success is but partial. However, even if every man in the early stage of the disease were ready to take advantage of the sanatorium system, it would not be possible to provide enough accommodation for one-tenth part of the contingent afflicted with the disease in an early stage. Even in Germany, which provides for 30,000 cases every year at a cost of many millions of pounds upon building and close upon £1,000,000 a year for maintenance, there are nearly 200,000 cases in all stages which cannot have treatment, and the net result of treatment is that after a lapse of five years 60 per cent. of those already treated have succumbed to the disease. Hence it would seem to us that the sanatorium system, as a means of dealing with pulmonary tuberculosis among the poor, is at best a poor thing, often inapplicable, often inadequate and always expensive. With us the sanatorium system fails, (1) because men

and women suffering from disease in the early stage are loth to seek refuge in the sanatorium, and there is no law to compel them; (2) because the results are not permanent, and (3) because it is far too costly to be of general application.

Let us now examine the evidence obtained from the experiences of German sanatoria for the poor, in order to see whether, after all, it is worth while in England to apply the same method *under less favourable conditions*. Even in Germany, where the system of sanatorium treatment has reached a climax, such as has not been approached in other countries, the net result has been that at enormous cost of money and labour, and by means of a colossal piece of State machinery, 30,000 to 50,000 out of an army of nearly a quarter of a million suffering from pulmonary tuberculosis are relieved from the stress of their disease for a limited number of years. Let us not misread and misunderstand the purpose of this treatment in public sanatoria. The system of sanatorium treatment for the artisan is based on economic principles, and the economy is manifest if the artisan, after treatment, maintains his industrial competency for a period of three years. For the insurance society and its economics it is enough if at the end of three or four years all those who have had sanatorium treatment are dead. Looking at the system from the points of view of the insurance society, the system is an economic success. But from the point of view of the *individual* artisan it may mean anything but success. In the majority of cases the individual dies of the disease in four or five years. Thus the net result of sanatorium treatment for the poor man is that in spite of his disease he is able to work (*arbeits fähig*) for two or three years longer. The poor artisan is not cured of his disease, but is merely improved for a few years as a working unit. I refuse to believe that we have reached finality in securing no more than this economic success. He who is satisfied with these results of sanatorium treatment among the poor is lost. Nevertheless I would add that, although the sanatorium system hardly touches the fringe of this great problem, and is anything but an ideal system, it has some advantages, and may be almost indispensable in certain phases of pulmonary tuberculosis, especially

when complications such as mixed infections, hæmorrhage, and even pleurisy of an acute type have developed. It is not their value under these special circumstances that is open to criticism, but their great cost, especially when they are exploited in routine fashion for any and every case of pulmonary tuberculosis. I have proved that in the early stage the disease can be treated at home without even the sacrifice of the daily wage—*with tuberculin alone*—and the cost of treatment is within reach of the artisan, if special dispensaries are created for this specific object (see address to Sydney Municipal Council). In the later stage, when there is fever or great loss of vitality and tissue energy, sanatoria help greatly; but under these circumstances three or four weeks in a sanatorium is generally ample. If, therefore, my ideas were given practical form, it would mean that, *with a given sum of money*, one can do fifteen to twenty times as much good as the methods in vogue can do, and the good results would, beyond a doubt, last very much longer. My records demonstrate this fact beyond any question. Again, I say, if money is no object, people may enjoy sanatorium methods, for they certainly do good. But I have proved in very many cases that where sanatorium methods fail, tuberculin sometimes works wonders; and I am convinced that where sanatorium methods succeed, success by means of tuberculin is almost inevitable, and to a far higher level; because the successes with tuberculin have this great virtue—*they last.*

Before we consider the results obtained in the German sanatoria for the poor, it is necessary to criticise also the common notion that sanatoria *per se* play an important *rôle* in the prevention of consumption. With our present knowledge this notion should be buried in the scrap-heap. Sanatoria deal essentially with the disease in its non-infectious stage, so that the temporary segregation of early cases removes no dangerous source of infection. Again, the most infectious and most dangerous forms of the disease are rarely admitted into public sanatoria. It is therefore nonsense to suppose that institutions, whose avowed policy is to admit for a few months early cases, in which the risk of infection is minimal, and rigidly to reject the most infectious cases, can play a serious

rôle in the prevention of tuberculosis. At any rate, I am quite clear in my own mind that no government or municipal body, handling public funds, should devote any money to sanatoria until it has already made ample provision for the prevention of consumption in accordance with the orthodox principles of hygiene, and it is one of the essential principles of hygiene to attack the dangerous sources of infection—either by destroying them or by removing them out of range of danger. Sanatoria exist for the treatment of pulmonary tuberculosis, and it is not the function of governing bodies to undertake the treatment of disease except in so far as the treatment may be incidental to a policy of prevention. Thus in smallpox and plague, isolation of the sick is enforced, and therewith the government must also assume the responsibility of treating the disease till the period of infection is past. But no government enforces the isolation of those suffering from pulmonary tuberculosis, and therefore the government incurs no responsibility for treatment. If ample provision has been made by the central government for the prevention of consumption by the erection and maintenance of hospitals or refuges for the reception of the poor afflicted with the disease in a late stage, and municipalities provide measures for the notification of the disease with a view to the disinfection of premises known to be infected, then the treatment of the disease can be left to medical men either in the homes of the patients or in special wards in general hospitals or in sanatoria for the well-to-do : or in dispensaries for the poor. Thus the State government will have its hands full within its own clearly defined sphere of action, municipalities will have plenty to do within their own spheres, and there will be no interference with the medical profession. Thus there will be no overlapping of functions. It is not for State governments or municipalities to take in hand the costly business of providing and maintaining sanatoria for the treatment of this disease, though it may or may not be within the spirit of the law to support any and every system, especially the dispensary system, that aims at mitigating the intensity of the injury and suffering caused by this disease in every class of society. A last word upon sanatoria as a means towards prophylaxis.

Much is often said of the educational value of sanatoria, and I admit that the lessons of hygiene—of personal and domestic cleanliness—inculcated by the discipline of the sanatorium—especially with regard to the disposal of sputum and the proper way of coughing—are very valuable, but to my mind these lessons can be taught just as well by the properly trained district nurse in the homes of the poor. The proper precautions involve some irksome duties and, unless there is constant supervision and insistence, the victim of a long lasting form of pulmonary tuberculosis soon lapses from grace. Even the pupils of the sanatorium, as I know full well, have often surprisingly short memories for inconvenient and irksome instructions, while the district nurse can daily urge the value and necessity of personal and domestic cleanliness and is bound to become in the future an indispensable handmaiden in the great work of preventing consumption in the homes of the poor. Again, the lessons at the sanatoria are very costly, while the lessons given by the district nurse are quite as effective and far cheaper. But from the point of view of prophylaxis tuberculin can claim advantage over every other system, for tuberculin properly used prevents the non-infectious form from becoming infectious and converts many infectious into non-infectious forms. All authorities agree that no method has such a marked effect in causing tubercle bacilli to disappear from the sputum as tuberculin. Hence tuberculin is also a valuable agent in the prevention of this disease.

At length we may turn to consider the value of sanatorium treatment for the poor as disclosed in the enormous labours of Weicker, Engelmann, and Hamel. Even in this work of the sanatoria tuberculin as a diagnostic agent is used to detect those early cases which are treated so successfully in the sanatorium. Without tuberculin mistakes would be inevitable. Thus by the use of tuberculin in diagnosis the value of the sanatorium is greatly enhanced.

Further, the sanatorium system starts with one immense advantage: it can pick and choose. In every sanatorium, in Australia as well as in Germany and England, the most favourable cases are selected for treatment, and chiefly by

means of tuberculin. Thus by rejecting bad cases and accepting many that are either not in an active condition or might get well without any treatment at all, the sanatorium has as enormous advantage over the poor practising physician who has to do his very best with all the cases, good and bad alike, which come for advice and treatment. As a practising physician in this plight I should hardly dare to make any comparison with sanatorium records, if it were not that I have been using a remedy which too many sanatoria have unwisely excluded. Sanatorium authorities admit that cases in the late second stage (II.–III.) and in the third stage (III.) are not fit for sanatorium treatment. It is natural too that many of the failures of other medical men find their way to him who has long insisted that haphazard methods, such as are generally practised in Sydney—not even sanatorium methods, but methods essentially based on the superstition that the climate of the mountains or of the plains of Australia has specific virtues denied to the climate of the sea-coast—are utterly untrustworthy. Scores of patients have proved the soundness of my contentions by the progress of the disease in their own bodies, when they have been hustled into the country and have come to me in the last throes of the disease. Naturally these cases belong to the statistical tables of the other fellow, though they may never appear there. I shall not damage my own results by inserting them. Accordingly I shall merely attempt to compare like with like, and shall place cases in the third stage of the disease in a class by themselves. Yet I must admit that my belief in tuberculin was profoundly strengthened by the extraordinary effects in a hopeless case in the third stage, which I treated about seventeen years ago, in 1891. A young man, *aet.* 26, came to Sydney and consulted a doctor who told him that there were cavities in both lungs, and he could not live three months. He came to me, and there was no doubt the first doctor was right. The patient was a walking skeleton. He had very abundant expectoration, amounting to many ounces of nummulated masses. I refused to treat him, fearing the catastrophe one read of in German and English journals in that year, but he was willing to take any risks.

The discovery of the remedy had only been heralded in the journals a few months before, and I had at once cabled for the tuberculin. I had already used it in half-a-dozen cases at Mr. Goodlet's Home, and had seen no catastrophes —indeed, nothing but good from it. In one case, the man had been under me at the Sydney hospital, and I had lectured upon him as an early case of pulmonary tuberculosis. He reminded me of this himself at Mr. Goodlet's Home. I gave him a test dose of ·001 Old. T. He reacted to 104°. I treated him for some months. This man had been in the home for two years—*in statu quo*— and had never felt strong. Within five months of the first dose of tuberculin he went out a different man, having gained greatly in weight and recovered his energy, and eleven years after I heard of him as being absolutely well and at work. Accordingly, at the earnest solicitations of this hopeless case "from the country," I treated him, and he improved beyond my wildest expectations. Not only so : he returned to his work, and, as he was very poor, I taught him in 1891 to give himself the injections. I sent him injections for years, but I learnt too late that he knew more of the value of tuberculin at that time than I did. He assured me that *only the large doses did him any good.* Sometimes, to pacify him, I would send weak doses. In the face of European opinion I thought it risky for a young practitioner to yield to his piteous prayers for large doses. If I had given him tuberculin according to my later ideas, he would have lived longer. As it was he remained at his work for another six years— very much to my surprise. This history also shows that I am no new convert to the teachings of Professor Koch.

What lessons, then, may be learnt from German experience in sanatoria? No one in Germany can command greater respect upon the question of the value of sanatorium methods than Weicker, who has devoted his whole life to the study of measures for the relief of consumption among the poorer classes of society. Weicker began his noble work as an enthusiastic advocate of sanatorium methods *per se*, and, determined to arrive at the truth, he established and carried out a long and laborious series of after-examinations in the cases

which he had already treated by sanatorium methods. These after-examinations told the fatal tale. He discovered that of every 100 cases treated in sanatoria by rational methods alone, at the end of 1–1½ years 41, at the end of 2–2½ years 51, at the end of 3–3½ years 65·8, and at the end of 4 years 80 had lost their capacity for work or were dead. Such were the fatal figures revealed by his careful inquiries into the after-history of some *thousands* of cases that had received sanatorium treatment under his very eye. The death-roll was as follows :—

In 1–1½ years	25 %
In 2–2½ years	38·4 %
In 3–3½ years	55 %
In 4 years	66·7 %

Such figures may give a little satisfaction to those who were spared for a year or two the necessity of paying the tax for an employee's invalidism, but the victim has not much respite, if the result is merely a postponement of death for three or four years. Again, the aspect of sanatorium treatment is not encouraging when we read that of cases in Stage I.—

7·6 per cent. are unfit to work or are dead in 1–1½ *years,* 19 *per cent. in* 2–2½ *years,* 33·3 *per cent. in* 3–3½ *years, and* 55·6 *per cent. in* 4 *years.*

Such appalling records caused Weicker and others to pause and think. Weicker had carefully weighed sanatorium treatment in the scales and found it wanting, but, thank Heaven, Weicker has taken heart again in his noble effort to save the poor from the terrible results of this disease by becoming an earnest convert to tuberculin treatment. I regret, however, to find in his latest work (Wein, 1908) that he is still timid and fearful, for he approves and practises the use of very small doses. Perhaps my experiences may give him courage, and thenceforward he will obtain results that leave little to be desired. At any rate, both he and Moeller (formerly an utter sceptic, as I have reason to know, for I spent three days with him at Goerbersdorf in 1899) are convinced converts, and among the most enthusiastic advocates of tuberculin treatment in Germany. If Weicker's evidence is not enough to gain

a hearing for me, I would recommend any seeker after truth to read and inwardly digest (it will need a good digestion) the statistics of Engelmann and Hamel in their analyses of about 30,000 cases treated by sanatorium methods in various sanatoria in Germany. As soon as the searchlight of after-examinations is directed upon the temporary results recorded immediately after the course of treatment, the vaunted benefits of sanatorium methods gradually dwindle away, and the naked truth in all its sadness stands up in bold relief to tell us that the sequel to the long chapter of improvements is but a long, wearisome, and heart-breaking chapter of relapses, ending in death after a lapse of a few years in most cases. *Such a result, too, in cases that have been specially selected!* These publications are regular store-houses of information on this great question, and originally I intended to place many striking facts before my readers, but in substance these works merely show the same results as Weicker's investigations—demonstrating the conditions, limitations, and shortcomings of sanatorium methods.

Engelmann, as the officer of the Imperial Board of Health of Berlin, investigated the after-history of 20,878 cases that had been treated in sanatoria by the various Imperial Insurance Societies, and found as follows:—

In these 20,878 cases treated in

1897	1898	1899	1900

and examined in 1901, the results lasted

3–4 years in 30 %	2–3 years in 39 %	1–2 years in 48 %	1 year in 66 %

Weicker's inquiries in 2,469 cases show that in 35 per cent. of the cases the results lasted four years, in 41 per cent. three years, in 50·7 per cent. two years, and in 55 per cent. one year.

Certainly the records of the Hanseatic States and of Turban-Rumpf are better, but even Turban only claims permanent results in 38·6 per cent. after four years, and 38·8 per cent. of deaths. Turban, however, includes also results in the well-to-do patients, and the average duration of

treatment was 222 days. The following table is also instructive. The cases were treated in years 1896–1901, and examined in 1901.

Of cases treated in

1900,	52 %	were fit to work,	15·1 %	were dead
1899,	48 %	,,	,,	26·3 %	,,	,,
1898,	36·2 %	,,	,,	41·5 %	,,	,,
1897,	33·3 %	,,	,,	48·5 %	,,	,,
1896,	18·1 %	,,	,,	62·3 %	,,	,,

Thus in two years 25 per cent., in four years 50 per cent., in six years 60 per cent. are dead, even though special care was exercised in selecting favourable cases for treatment.

If results approaching these had been my lot in practice, I should have been almost in despair. As it is, I turn with a sigh of relief and the buoyancy of hope to the other side of the page, where we shall consider this great national problem from the point of view of those who believe in the specific treatment of tuberculosis by means of tuberculin, either with or without the aid of sanatoria.

CHAPTER V

THE PROBLEM WITH TUBERCULIN AS A REMEDY

WE have already discussed the use and value of tuberculin in prognosis. Tuberculin in diagnosis is a very fascinating subject for investigation, and those of us—all too few—who have used tuberculin in diagnosis as a matter of routine and with no sparing hand can but marvel, whence those fearful results had their being and origin, of which one heard so much from isolated individuals, whose names have since remained in oblivion. I knew of a man who had been treated with tuberculin by three doctors; one prepared the mysterious fluid, another man watched the proceeding, and the third inserted the needle. These doses were given every four hours. The patient had severe attacks of fever! and was alive thirteen years after to tell his story. Like other workers I had a very narrow escape more than once. On one occasion for some chance reason I postponed the dose for two days. The patient had some sudden accident in the abdominal cavity next day and died within twenty-four hours. If I had but given that dose what a fuss there would have been! Fortune favoured me that time. On two or three other occasions fever suddenly appeared when I had refrained from giving an injection. Of course, if men choose to generalise from an isolated incident they must err; but they must not expect others, who have more logical minds, to err also. After seventeen years of constant use I am able to say that I have not yet seen a single bad effect from one or several test doses of tuberculin. There may

have been a severe reaction, but always a few days later the patient would volunteer the statement that he felt better than he had felt for some time, and almost invariably he would gain one or two pounds.

At first they generally lose a pound or two, but they soon regain this and an extra pound or two. One has not to wait ten days for this gain. It often occurs in half that time, which is further damaging evidence against the long duration of the negative phase as indicated by the opsonic index. I am sure that gain in weight under the same conditions is a far safer and infinitely easier index to the graduation of doses than Wright's opsonic index.

In all my work I have paid more attention to variations in weight than to any other single phenomenon, and I do not think it is safe to dispense with it. This gain of weight, too, may help to settle the diagnosis when the rise of temperature fails, or is of mild degree (99·5–99·8°). I consider a temperature of 100° to be conclusive, if accidental causes can be eliminated. If there is any doubt, it is best to repeat the dose, and then the most characteristic effect of tuberculin may be manifested—a still higher temperature, 101° or more, due to oversensitiveness to tuberculin (see nature of tuberculin reaction). In one interesting nervous case ·002 Old T.—the second dose—caused a temperature of 99·6°. I declined to give a positive opinion, and the young patient refused to have another dose. He went to another doctor, and probably told him that I had said he had tuberculosis. I refused then as always to give a positive opinion on a rise of temperature under 100° F.

Tuberculin as a diagnostic agent gives no uncertain answer in two classes of cases. It detects tuberculous disease almost with the certainty of an electric current along a telephone wire, and excites a local response in the diseased area. On the other hand in many cases I have proved beyond all doubt that there was no pulmonary tuberculosis, when other doctors told the patients that there was disease and that they should go into the country for a year or two.

I saw one young woman who had spent a year and a half in a private sanatorium managed by a doctor in Melbourne.

When she came to me I could find no evidence at all of pulmonary tuberculosis, but she had just the most typical physical signs of mitral stenosis one could possibly imagine. Her sister was a nurse, and I told this sister that her complaint was heart disease and not lung disease. She was shocked to hear this, and determined to take her to another doctor, who absolutely agreed with my verdict. There was no escape from this conclusion, and yet this case had been treated for a year and a half for pulmonary tuberculosis because she had pulmonary hæmorrhage, and I doubt not this case is recorded among the successful cases of this sanatorium. I also tested the two sons of a doctor in Queensland, who had already sent one of these boys to a sanatorium for a year without any benefit. When he brought the boys to me, he said they were just as bad as ever. I examined them, and found absolutely no evidence of disease. I also tested them up to ·01 grm. Old T., and the result was negative. The father, a medical man, was staggered when I told him that neither of his sons had tuberculosis.

I also tested a woman who had been recommended by two or three doctors at the Prince Alfred Hospital for the Sanatorium for Consumption at Thirlmere, where there were many advanced and infectious cases. Her husband had recently died of consumption. She gave a negative reaction to ·01 grm. Old T., and was so pleased to hear the truth that she brought her daughter afterwards to be tested. Yet two doctors had no scruples in letting her go and live with advanced cases of the disease.

I have tested also several nurses who had been much with consumptive patients, and in every case but one removed a great load from their minds by proving that they were quite free from tuberculosis. I also tested a young woman who was on her way to the dry air for consumption. Her doctor said she had lung disease, and she took it for granted that she had the disease because she had lost weight. However, a friend of hers brought her to me, and I proved that there was no tuberculous disease of the lungs.

Another very peculiar case was sent to me by Dr. H. H. Examining the blood, I found well-marked lymphocytosis,

and concluded that there might be either tuberculosis or syphilis. I excluded tuberculosis by means of tuberculin, and then sent back the case to Dr. H. H. as probably syphilitic. Antisyphilitic treatment had a rapid effect in dispelling severe abdominal symptoms which had persisted for many months.

In another case there was a lesion on a knuckle of a girl, which looked to be tuberculous. I called in a specialist who agreed with me that it was either tuberculous or syphilitic. I excluded tuberculosis by means of tuberculin and refrained from adopting antisyphilitic medication till I had more grounds for the diagnosis. By simple treatment this chronic sore healed up and the girl has been well ever since. In two cases of bronchiactasis also I was able to exclude tuberculosis. In two cases of well-marked hæmorrhage from the lungs, I proved by means of tuberculin that there was no tuberculosis and the only other obvious diagnosis was hydatid disease. Two other cases came out to Sydney from England: one of them had been advised to give up a very good appointment and go to Australia, as his lungs were affected. He came to me within a day or two of his arrival, and finding no physical signs at all, I tested him and proved he had not tuberculosis. This case was a cruel illustration of a haphazard diagnosis. Surely before advising a poor man to give up a good position, a *few small doses of tuberculin might be given to save these unnecessary sacrifices.* Medical men may not seriously realise what such advice means. Would they be ready to sacrifice their own practice in obedience to such a random diagnosis? Another Englishman was sent out because he had developed a pneumothorax after a serious fall in the hunting field, and Dr. K. F. was not sure whether there might not have been a localised tuberculous lesion. I proved by means of a test dose up to ·01 grm. Old T. that there was no tuberculosis. In no less than sixty cases I proved that, although tuberculosis might have been suspected, it could be excluded with certainty by means of tuberculin. Many of these cases would have been sent up the country as cases of early tuberculosis and would be recorded as "cures." Unfortunately I have to be honest and exclude these cases from my list, although they

are such easy cases to cure. But at least I have saved many from wandering about the country, as other doctors had advised, in search of a cure for a disease they had not got. On the other hand, needless to say, I have detected by means of tuberculin many and many a case of tuberculosis which had not been recognised. This is of course a much more serious mistake, because if the disease is overlooked in the early stage, an irreparable injury may be done to the person who seeks your advice.

(C) *Treatment by means of Sanatoria with Tuberculin as a remedy.*

Infectious diseases of specific origin are the result of an affinity existing between the specific cause or its product or both and the tissue elements of the man or animal. The result of the disease depends on the action and reaction of these two essential factors. If the specific cause is very virulent or very abundant, it overpowers the vitality of the invaded tissues and destroys the organism. On the other hand, the resisting energy of the tissues may overpower the agents of disease and either destroy them or neutralise their dangerous products. Tubercle bacilli, the specific cause of all forms of tuberculosis, manifest a remarkable affinity for certain parts of the human body, notably the lungs, and the result of tuberculous disease of the lungs depends on the virulence and number of tubercle bacilli and the resisting energy of the invaded tissues.

In the treatment of such an infectious disease there are two rational methods—one directed against the specific cause and therefore called specific, the other aiming rather at strengthening the resisting energy of the invaded tissues.

These two distinct methods of specific and general treatment should be combined if we wish to get the best results.

The specific method of treating pulmonary tuberculosis consists essentially in the use of tuberculin in its various forms, which helps to destroy the bacillus and to neutralise its toxic effects. This method is in fact a process of active

immunisation entirely different from the antitoxic treatment of diphtheria, in which the antitoxin is supplied ready made. In principle there is no difference between tuberculin treatment for the purpose of curing tuberculosis and the use of Haffkine's fluid or similar agents for the purpose of preventing for a short time such diseases as plague, cholera, or typhoid fever. If, then, the method is advocated in order to protect a healthy person against a disease, which in a mathematical sense he has a very small chance of contracting, it is not unreasonable to urge the use of the method if it offers even a small chance of cure to one who has already fallen a victim to the disease. Those who have used and advocated the use of specific prophylactic fluids containing germ products for the prevention of plague cannot with any show of consistency oppose the use of tuberculin, which is, after all, merely a vegetable product of the living tubercle bacillus. Many are, in their ignorance, guilty of this strange inconsistency. On the other hand, I may disarm some opposition by saying at the outset that the *extraneous use* of tuberculin is not the one only indispensable means of curing tuberculosis, even in an anatomical sense. We know of no disease in which death is inevitable. Always some individuals are ordained by nature or strong enough to survive an attack of the most virulent disease. Plague does not kill all its victims; nor yet tetanus or anthrax. This power to resist lies in the tissues. Let us go a little farther. This power to resist depends upon the ability of the tissues to produce a sufficiency of antibodies which either kill the specific agent or render its products harmless. It is too often forgotten that the very and only cause of the production of these antibodies in the tissues is the specific cause of the disease. *The disease works out its own cure by means of its own cause.* Not only the disease, but even the mechanism of immunity that cures it, are both the results of the living germ through its specific products. Except in this way no infectious disease cures itself or protects against itself. Even when we seek to strengthen the energy of tissue so that the disease may be resisted, we are blindly assisting nature to work a cure in nature's own way. In pulmonary tuberculosis sanatorium

treatment, open-air treatment, the rest cure, and other methods act by stimulating the system to evolve the mechanism of immunity in response to the action of the products of the tubercle bacilli in the tissues, the most important of which is tuberculin. It may be a shock to some of the advocates of sanatorium treatment to hear that they themselves, without knowing it, have been innocently vaunting the efficacy of tuberculin in the treatment of tuberculosis. On the other hand, those who advocate the artificial supplementing of nature's own direct weapon of defence are quite consistent in upholding the value of other methods, especially sanatorium treatment. Daily experience tells us that pulmonary tuberculosis is a disease that too often fails to work out its own cure. Some fault in the mechanism of immunity allows the germ to continue its work of destruction or, maybe, to lie in ambush for months and years. The whole purpose of treatment is to stimulate the tissues to build up and finish the mechanism of immunity so that the tissues shall no longer be at the mercy of the tubercle bacillus. *A priori* it seems logical to stimulate the mechanism by means of the specific products called tuberculin. *A posteriori* other methods evolved through empiricism are vaunted as the *ne plus ultra* and specific methods are held to be superfluous.

One sees cases of pulmonary tuberculosis, even complicated with tuberculous ulceration of the larynx or epiglottis, get well of themselves without any special treatment, without sanatorium treatment, without injections of any sort, without local applications.

In the first place, early diagnosis is the key to successful treatment. In the early stages the lesion of active or latent tuberculosis may produce physical signs like those of obsolete foci, and tubercle bacilli may not yet have escaped from the tissues. Obsolete lesions are not tuberculous, but post-tuberculous. How can the physician attempt to unravel the mystery of these various lesions? If the tubercle bacilli have disappeared there can be no tuberculosis, and if there is no active tuberculosis there is no reaction to tuberculin. This is a vital point. Tuberculin alone gives the clue whether the lesion is active or inert. If the focus is inert it may harbour

tubercle bacilli that will become active at some future time. Yet, if the tuberculin reaction fails, it is likely that the tubercle bacilli are so surrounded that the tissues can themselves destroy the bacilli. On the other hand, a reaction to tuberculin proves that the lesion is not inert. This reaction is a danger signal of the first importance. It warns the physician of a danger hidden from sight, just as the fog signal warns the sailor of a danger in a mist which the eye cannot penetrate. By the systematic use of tuberculin we may learn the true nature of many of those lesions which Naegeli too readily assumed to be tuberculous. Many of these lesions may be merely passive—quiescent, but still ready to be kindled into activity by inflammations or other infections. Sad to say, these closed lesions thus neglected may become the source of the acute and rapidly-progressing forms that develop in early adolescence. Now, if these terrible disasters may happen, there has been no cure, even though the lesion is for a time closed and inert. By no other way than by the systematic use of tuberculin can we distinguish these latent cases from absolute cures; and if tuberculin is not used, no one has a right to use the term *cure*. If the tuberculin test is negative, and again after an interval of three months or six months again negative, there is some certainty that a cure has been accomplished. When the prejudice against tuberculin has died its natural and inevitable death, one of the most valuable uses of tuberculin will be found in determining the effect of treatment of any and every sort. Meanwhile, except by those who use the test, the term *cure* must be avoided.

Let us add that the Tuberculosis Congress in London, 1901, deliberately adopted the opinion that tuberculin was an invaluable agent in diagnosis. The education of English opinion needed ten years before the truth of Koch's original statement was recognised.

Sanatoria cannot dispense with tuberculin as a diagnostic agent, and gradually tuberculin is taking its right place in all the sanatoria of Germany. If tuberculin is not used, mistakes must occur. True cases of tuberculosis are overlooked, and, on the other hand, there is the risk of condemning a man to an expensive and irksome course of treat-

ment for a disease which he has not got. In one year Weicker rejected forty-eight spurious cases already diagnosed by good examiners as early cases. In the eager chase after early cases, otherwise trustworthy examiners may condemn to sanatorium treatment many cases in which the diagnosis rests not upon trustworthy evidence, such as tubercle bacilli in the sputum or a definite reaction to tuberculin, but rather upon the subjective impressions of the observer as to the character of respiratory movement or auscultatory signs. The tuberculin test alone can save the profession from the Yes-No diagnosis. But for the tuberculin test Weicker might have greatly improved his statistics by including the rejected cases, and ultimately *recorded them as cases of cured tuberculosis!* Christian Science can do as much for these cases as the best sanatoria. Let us take care we give no such chances to the Christian Scientists to show their skill. It is an easy offhand criticism to offer that tuberculin in diagnosis may lead to the inclusion of cases of latent tuberculosis that may never further develop. The same may be said of many of the cases upon which the statistics of sanatoria are based. Who will dare to say that it will not develop if a reaction to tuberculin occurs? On the other hand, by the use of tuberculin I have saved many persons the expense and inconvenience of exile to the country, to which they had been sentenced by various medical men. Further, when one uses tuberculin for treatment also, if perchance tuberculin has given a doubtful or false indication, the mistake is soon remedied. Thus, if the reaction be no greater than 99·5° to 100° F., and tuberculin is given for treatment, a constant absence of reactions with larger doses rapidly increased arouses suspicion. In my experience, with proper doses a temperature of 100° is positive. A rise of temperature to 99·5° to 100° is doubtful. *Tuberculin is quite harmless, if there is no tuberculosis.* Why, then, should we fear to use it in the cause of sanatorium or other methods of treatment, when it surely reveals the presence of tuberculous lesions? I have no hesitation in saying that tuberculin is an invaluable agent in the selection of suitable cases for sanatoria, and also a ready means of gauging the success or failure of treatment.

A more open question is the value of tuberculin as a curative agent. It is extremely difficult to measure the value of different methods of treatment in a disease that runs such a variable course. For any and every method of treatment successes will be claimed. The only test is the *relation of successes to failures.* If we investigate the statistics of sanatoria, we find that the successes of to-day are the failures of to-morrow. Every medical man has had successes, but far more failures. Moreover, records are practically worthless, *unless they show that after an interval of time, say two or three years, the patient is the better for treatment.* It is the tradition of sanatoria to publish results as soon as patients leave the institution. This pernicious practice must give rise to misconceptions and disappointments. Many of the apparent successes of to-day prove the certain failures of to-morrow. Still the after-history of these recorded cases year by year will show the truth about the results of sanatorium treatment.

Three months' residence in a sanatorium may be well enough from an insurance point of view, since it may restore the industrial competency of a workman for one or more years, but in three months one cannot expect to arrest the disease, and it is arrest of the disease which the workman is led to expect and desires. The workman wishes not only to be able to work but to recover his health. In most cases the work of sanatoria is patchy and unfinished. Patients are discharged—improved no doubt—but with the disease still potential, if not active, and merely awaiting a favourable opportunity to reassert itself. My purpose is to bring forward reasons for advocating the use of tuberculin as a remedy so as to bring the work of the sanatorium to a higher level of efficiency and completeness, for the benefit of the unfortunate victim of pulmonary tuberculosis, and even of the whole community. The better the work of the sanatorium the better for the community, and at the present time the inmates of our sanatoria are denied the advantages of tuberculin treatment that are enjoyed by patients in some of the best German sanatoria. The ideal treatment is specific treatment combined with those general methods adopted in the

sanatorium that are of undoubted value in increasing that tissue energy upon which the success of tuberculin treatment depends. Nevertheless, in very many cases, although treatment in a sanatoria may be useful, it is by no means indispensable. In the next chapter I shall proceed to show that, without any sanatorium, treatment, tuberculin can achieve all the success one would desire in a large proportion of cases of pulmonary tuberculosis.

Years ago, when I was making my early observations with tuberculin, I studiously avoided the introduction of factors that might vitiate my conclusions. Accordingly I instructed patients to live under the conditions to which they had been accustomed. Having thus proved that the uniform improvement could be only due to the new factor—the tuberculin—I lost no time in adopting all measures that might reasonably be supposed to help in the arrest of the disease and the cure of the patient.

Strange as it may seem, I am the only specialist in diseases of the lungs in Sydney who has gone to the trouble of providing sanatorium treatment for private patients, even though I consider the sanatorium methods of subordinate importance. Other medical men who openly proclaim that sanatorium treatment is of primary importance have taken no steps to secure for their private patients that method of treatment which they tell the world is the best and indeed the only treatment. It is idle to pretend that the haphazard exile of patients to the country constitutes sanatorium treatment. I leave it to the over-zealous advocates of sanatorium treatment for the poor to explain why it should not be equally successful in their well-to-do and rich patients. Let those who wish to be enlightened upon the great virtues of sanatorium methods read Dr. Ransome's article in the *British Medical Journal.* He quotes Walther of Nordrach to this effect, that, if by cure we mean all subjective and objective signs of disease have disappeared, no more than 11·20 per cent. can be so classed, although improvement occurs in 70·80 per cent. of the cases. Ransome writes himself, " I think it may fairly be said, as the result of a large mass of Continental statistics, that only about 30 per cent. of patients discharged from sanatoriums for the

poor maintain the capacity for work more than four years." He also writes, "In England sanatoria for the poor have existed too short a time [writing himself in 1905] to allow of statistics of much value being collected from them." I said the very same thing quite two years before—in 1903. Later Ransome says, "Exactly half the patients discharged have maintained the improvement gained for over a year, while half have got worse or are dead."

Professor Moeller, of the Belzig Sanatorium near Berlin, and a recognised authority on tuberculosis, eight years ago an utter sceptic upon tuberculin treatment, is now one of its most earnest advocates. He tells us that in cases in which sanatorium methods have entirely failed, tuberculin has been proved to possess undoubted curative properties, and that while sanatorium treatment never cures in three months, permanent cures are often obtained with tuberculin. Strange to say, Dr. Moeller uses tuberculin mainly in cases in which sanatorium methods have failed. My criticism of this half-hearted policy is simply this: If tuberculin succeeds where sanatorium methods fail, why ever should tuberculin fail when sanatorium methods succeed? Experience proves to me that tuberculin never fails when sanatorium methods succeed.

In his first record Moeller showed that

Sanatorium methods alone cured	10·9 %
Sanatorium methods and tuberculin cured	36·3 %

Arranged according to stages the results were:

Stage I.—Sanatorium methods alone cured	31·8 %
	Sanatorium methods and tuberculin cured	75·0 %
Stage II.—Sanatorium methods alone cured	1·9 %
	Sanatorium methods and tuberculin cured	20·0 %
Stage III.—No cures possible except as curiosities.		

According to Moeller's later experiences, *sanatorium methods alone* may cure 25·30 per cent. of the cases of pulmonary tuberculosis in the first stage (German Board of Health classification), while *tuberculin* in his hands has secured the same result in 84·6 per cent. of the cases. I

venture to say that when Professor Moeller uses larger doses or repeats the course of treatment, he will find that tuberculin will cure 90 to 100 per cent. of these early cases. In fact, Professor Moeller will agree, as I do, with our great teacher, Professor Koch, that "*tuberculosis of the lungs in the first stage can be cured with certainty by tuberculin.*" In the second stage, which Moeller maintains is never cured by sanatorium treatment alone, tuberculin gives permanent results in 40 per cent. to 60 per cent. of the cases. Even in the third stage tuberculin sometimes yields remarkable results that cannot be approached by sanatorium methods. When these splendid results are claimed for tuberculin by those who have had an extensive and prolonged experience with it, are we going to be so foolish as to pay any heed to the carping, surreptitious criticism of those sterile authorities who have the hardihood to express adverse opinions on a matter concerning which they have no personal knowledge?

In medicine, as in surgery, pioneers have often had to bear much unfair criticism. Experience is our great teacher. I do not know any great authority who has used tuberculin with the conditions and limitations laid down by Koch and found tuberculin wanting. On the other hand, I have seen no carefully compiled records that prove tuberculin to be either harmful or useless. We do not condemn operations because novices and those who have not learned their art make mistakes and fail. There have been terrible tragedies arising from various operations in their early history, but the operations have survived. The failures and tragedies associated with tuberculin are not the fault of tuberculin, but the fault of those "fools who rush in where angels fear to tread."

Failures may be the stepping stones to high success; and the failures of to-day are converted by increasing knowledge and experience into the successes of to-morrow.

(D) *The Problem with Tuberculin alone as* the *Specific Curative Agent.*

Thus far we have considered the value of the sanatorium treatment of pulmonary tuberculosis in its various stages *without and with tuberculin.* Weicker, Moeller, and others who closely watched the after-histories of patients treated by sanatorium methods *alone*, came to the conclusion that these methods failed except in a minority of cases. But these authors have proved that by the use of tuberculin the value of sanatorium methods is enormously enhanced, because tuberculin has the great virtue of converting temporary into permanent successes. From a logical standpoint, we have still to consider what success can be achieved when tuberculin alone is used. This was the problem which I approached at the outset, because, seeing that the sanatorium is quite beyond the reach of the vast majority of sufferers, I wished to discover whether there was any means of helping the poor, who can neither help themselves nor obtain relief at the sanatorium. I have come to the conclusion that in at least 80 per cent. of the cases in the first stage one can dispense altogether with sanatoria, that at least in 60 per cent. of the cases in the second stage sanatorium treatment is not indispensable, and that even in cases in which sanatoria utterly fail to do any permanent good, tuberculin can often bring the disease to a standstill, at any rate for many years. These are the general conclusions at which I arrived at least eight years ago, and it was because I had the courage to express my opinions candidly in the public interests that I aroused the opposition of the whole of the profession in Australia. The profession was wedded to sanatoria, and in attacking sanatoria I was guilty of creating dissension in this family party. Then it was that, independent of Calmette and of Mr. R. W. Philip, of Edinburgh, I worked out the idea of establishing and maintaining a dispensary in the city of Sydney, at which early diagnosis and prompt treatment by means of tuberculin should offer to poor patients the relief which they could not possibly obtain at sanatoria. By means of this dispensary system of dealing with pulmonary tuberculosis among the poor,

I have every reason to hope that in the majority of cases treatment with tuberculin at the dispensary could be carried out without loss of employment, and, therefore, without the loss of the daily wage. This is a great advance upon any other system, if it can be applied with success.

In the results which I published in 1901 I had adopted tuberculin treatment pure and simple, because I did not wish to disturb the simplicity of the logical problem which I attempted to unravel—viz., whether tuberculin was a remedy for pulmonary tuberculosis. Every patient remained in or near Sydney, and lived his ordinary life. I had used Old T., T.R., and T.R. Emulsion (T.E.) in full doses ; indeed, all the preparations which were magnanimously offered to a medical profession, which, like the Pharisees of old, rejected the one means of salvation for the multitude of victims held tightly in the grip of pulmonary tuberculosis. In 1901 I reported twelve cases of pulmonary tuberculosis in Stage I. which had been treated by tuberculin. Out of these twelve cases I know nothing of two. Of the remaining ten, one had to go to Victoria before she had received full treatment. Under partial treatment she improved immensely, gaining 18 lbs. and feeling well and strong. I know no more. Another case, a young girl *aet.* 17, improved greatly; she lost her cough and phlegm, tubercle bacilli (formerly G. 8.9) disappeared from the sputum, but after several months' treatment she became very sensitive to tuberculin, and, in accordance with Petruschky's method, I advised a pause of six months. She went away to the country and felt so well, that, without consulting me, she took up nursing. Later, I learnt that she had been in effect prevented from having further tuberculin treatment by a medical uncle, who was an authority on lunacy, and had a strong prejudice against tuberculin treatment. As I foresaw, the disease relapsed, and in spite of the uncle's methods, the poor girl died about a year and a half after she left me. The father afterwards told me that he had regretted ever since that his daughter had been removed from my care and subsequently he sent his other daughter to me for treatment. In this latter case the result has been entirely satisfactory. I am therefore justified in scoring these cases out of my list, and may treat

the second case as a "control" case. This case is the only case which refused my advice; and she is dead.[1]

Case 1, treated in 1892, was quite well in 1903.

Case 2, treated in 1898, had a slight relapse, but in 1908 was well.

Case 3, treated in 1901, was well in 1907.

Case 4, treated in 1899, had a relapse in 1904-5, but after another course was quite well in 1907.

Case 5, treated in 1900, was well in 1907.

Case 6, treated in 1900, was well in 1908. This man had four "control" cases in his brothers. I abstract this account from my article in 1901 :—"Mr. E., *aet.* 47, gave an ominous "family history. Four brothers, one after the other, had "succumbed to the disease. They had been under various "physicians for treatment. They had been sent to the "country and treated in the usual way; but it was of no avail. "The last survivor, having seen the failure of ordinary "methods, came to me (from Melbourne) when the fatal "disease showed itself in him, as in his brothers, by cough and "expectoration, and general loss of health and weight. I "examined him and found very evident signs of early disease. "No tubercle bacilli found in sputum. I therefore gave a "test dose, and ·001 grm. old T. raised temperature to 103° F. "All doubt was at an end. He went under a course of T.R. "up to 2 grms. The patient proved to be very sensitive, and "the course occupied six months. During the course the "patient had severe attacks of gastritis and enteritis, with "diarrhœa. At one time the weight fell considerably, and I "almost despaired; but ultimately the result was satisfactory. "The cough hardly troubled him, the expectoration ceased, "and he felt greatly improved. The improvement continued "for a few months, and then the old symptoms began to re-"assert themselves. I gave him a second course. The second "course he took very well: in two months one was able to "give him the full dose (2 grm. T.R.). Again the improvement "was very marked. Some months afterwards I saw him, and "he seemed in perfect health. His weight increased from "11st. 7lbs. to 12st. 5¼lbs., though during early treatment his

[1] 1908. Case 8 died.

"weight was only about 10st. 10lbs. He told me that in the "same time, from the beginning of their illness, his brothers "had all succumbed to the disease. The treatment was "carried out while the patient continued at his business, "though he lived out of town. The second course hardly even "upset him. Surely some great change had been worked in "his system that he bore the second course so easily."

In 1908 this man enjoys perfect health.

Case 7 was treated in 1897 (Tb. G. 3), and was well late in 1907.

Case 8 was really in the second stage; severe mixed infection, sputum abundant, and tubercle bacilli in myriads (G. 9). He was treated in 1901, and remained well till 1907, when he had a relapse, with fever and hæmorrhage, and later laryngeal tuberculosis. This patient, in spite of my urgent entreaties, would not come to be tested. He had a very large practice, and brought on his relapse by sheer overwork. He had to use his voice to straining point in his work, and to keep his worn-out body up to the mark he took far too much alcohol. It was inevitable that the body would break down, and in this case the breakdown occurred in the over-used organ—the larynx. He died in May, 1908, and I have little doubt that if he had been tested two or three years ago he would have reacted, and we could again have stepped in with tuberculin and prevented the catastrophe that occurred.

Thus, of eight cases treated from 1892 to 1901, seven cases are well, and one case died this year under conditions which show that tuberculin worked wonders for him. Of cases in the second stage, one case, complicated with laryngeal tuberculosis, survived nine years; two others, in the late second stage, survived respectively eight years and nine years; one died from a sudden hæmorrhage in five years; five others I have seen alive in 1907, treated respectively in 1899, 1900, 1901, 1899, and 1900. I know also that two cases in the third stage, treated in 1900 and 1901-2, were alive in December, 1907.

Such after-histories will compare favourably with any after-histories of cases treated by other methods. My own experience teaches me that the effect of tuberculin treatment,

although always good—indeed far better than any other treatment—varies in degree and extent (of time). Beyond all doubt, so far from Koch recommending too large doses, as Wright, trusting to his opsonic method, maintains, the doses of tuberculin, recommended by Professor Koch, are hardly sufficient to do the work asked of the remedy. For many years since these early observations of mine I have been giving large doses of T.E., and more recently still of Old T. and P.T., with most encouraging results. I frequently give 4 grms. of T.E., and if this be not sufficient it is wise perhaps to try other forms, especially P.T. or T.A. My observations in 1901, in the light of later experience, show that T.R. is a very useful remedy in early cases, but may or may not be sufficient to prevent relapses. *There is no better way of testing the value of treatment than by its effect in preventing relapses.* We cannot expect to know whether there are still living bacilli in the tissues, but we may be able to tell by means of tuberculin whether the bacilli are active. In cases therefore that have been treated by tuberculin, if the reaction to progressive doses—say to ·005 grm., ·015 grm., ·03 grm., ·045 or ·05 grm. of Old T.—fails to develop in any one who a year before had had a full course of tuberculin treatment, it is fairly certain that either there are no germs living in the tissues, or their action has been entirely inhibited. At any rate I have proved in many cases that the reaction fails when all the clinical features and symptoms suggest that the disease has been arrested, while the reaction develops invariably when the symptoms and signs point to fresh activity of the disease. One knows that cases occur, in which, although tubercle bacilli are being expelled from the lungs, there are none of the usual symptoms of open tuberculosis—at most a slight cough and slight germ-laden expectoration. I have seen no such cases, but it is said that such a condition may continue for years and be compatible with apparent health. The lesson of such cases cannot help us until we know the effect of test doses of tuberculin upon them. Moreover, by means of tuberculin these open infectious forms can be converted into harmless, non-infectious forms—surely a "consummation devoutly to be wished."

Accordingly *I regard this system of gauging the effect of treatment by these test doses of tuberculin not less than nine months or a year after treatment as an invaluable adjunct in the treatment of tuberculosis by specific methods.* More than this, I hold it to be very sound practice to submit patients to such tests regularly every year. These doses cause no trouble, no pain, no disturbance of any sort, except in cases that have relapsed ; and in such, this discovery of a positive kind is well worth paying for at the trifling cost of many purely negative results that are totally devoid of any risk. One knows how the surgeon argues who wishes to operate upon every case of simple catarrhal appendicitis. One may say that these cases left to themselves do not cause 10 per cent. of deaths. Accordingly the surgeon literally advises that he should be allowed to do 90 unnecessary operations with all the attendant risks in order to reduce the number of deaths. The gain to the individual depends absolutely on the special skill that can reduce the risk of the operation to a minimum, but in any case ninety operations are performed which do no good. Am I, therefore, asking much in proposing that all patients who have been treated for tuberculosis by specific methods should be tested from time to time by injections that can do no possible harm in the hands of an experienced physician? In fact, until tuberculin is thus exploited in diagnosis, in treatment, and also in prognosis, we shall fail to make full use of this great boon to humanity. If I had had my way and tested the one case which after tuberculin treatment relapsed and died, I believe this patient would be alive to-day. But the patient had been well for so many years, and was so busy, that he did not think it necessary, and meanwhile tuberculosis laryngitis supervened. I am sure that *in the treatment of this disease, even with tuberculin, we dare not be over-confident.*

Thus I may claim in Stage I., after an interval of from seven to ten years and more, nearly 90 per cent. of successes. While I was using tuberculin, I scrupulously avoided sanatorium treatment. At most I told the patients to take good food and plenty of it. I did not send a single case into the country at any time, and ever since these patients have led the ordinary lives, not of invalids but of strenuous citizens. How

many of the sanatorium successes are given this freedom? They are enjoined to be always in the country—to do this and not to do that—as though poor people had it in their power to choose the good only and reject the evil. I have therefore shown than in those very cases, which are supposed to be *par excellence* the cases for sanatorium treatment, much greater success can be achieved by means of tuberculin than by means of sanatoria, and at a very much smaller cost.

In short, while the rich and well-to-do may, if they choose, at the behest of their medical adviser, spend money and time —even a lifetime—in seeking the will-o'-the-wisp health, now at this sanatorium, now at that, it is high time for people of ordinary means, especially the wage-earners of the community, to know that even though they have fallen a prey to tuberculosis they may get the better of their disease and recover their health, more easily, more rapidly, more certainly, not to say at a far less cost of time and money and without loss of wages or work, by means of the remedy magnanimously thrust into our hands by the genius of Professor Koch,—a priceless boon to suffering humanity from the hand of the scientific spirit of Germany.

Beyond all doubt this remedy can be best brought within reach of the poor of cities by means of the dispensary system, which I fully discussed when I brought this subject before the Municipal Council of the city of Sydney as one of the aldermen. (See Address.)

Since 1901, when I first published my experiences with tuberculin, I have had no reason for excluding sanatorium methods, and I have adopted a mild adaptation of sanatorium treatment for all patients who could well afford these other methods. However, I do not consider sanatorium methods a *sine qua non* to success. I have treated many scores of cases without any suggestion of sanatorium methods with complete success, and most of these patients belong to the class for which tuberculin treatment can be easily exploited at the dispensary and through the dispensary system. Among my patients were many servant girls (8), shop girls (5), other poor girls (8), nurses (3), many poor mothers, teachers in public schools (6), labourers (about 12), printers (2), butchers (2),

actors (4), tram-guards (3), railway men (2), barmaids (3), hotel-keepers (3), clergymen (4), singers (3), clerks (about 25), waiter (1), milkmen (2), fruiterers (2).

In all these cases they spent their time as they liked and went daily to their ordinary avocations.

It is therefore abundantly clear to me that the energy that is now waiting to be expended for the lasting benefit of the poor already in the thraldom of this prevalent disease should be directed along that channel which offers the best results at the least cost. Sanatoria are far too costly to bring much relief to the poor, and, besides being inadequate in so many cases, are decidedly inferior to systematic treatment by specific remedies. In my opinion the logical force of my argument is absolutely unassailable, unless later workers can show that my results are untrustworthy.

I shall await with perfect confidence the verdict of any system designed to put my experience to the test of actual practice, but I fully recognise that it will not be possible to test the truth of my observations in a short space of time. It needs several years to prove the uncertainty of the inferior methods of the sanatorium. It must take a longer time to test the results of a method which is held to be superior. Although I know that public opinion is entirely against me, I still assert, on the strength of a long and laborious period of close and constant observation, that in a few years' time the specific treatment of pulmonary tuberculosis by means of tuberculin will be the recognised system of the scientific and conscientious physician.

This final chapter upon the results of tuberculin treatment must be read with the records of cases which have been treated by me since 1892. I admit that after the wave of condemnation that swept over us in 1893–1894, it was extremely difficult to find any who were ready to submit themselves to treatment. Such good things were promised by the advocates of sanatoria, and such bad things were said of tuberculin treatment, that the patients who came to us were chiefly the failures of other systems. We rarely had the chance of treating even a case in Stage II., and the methods of 1893–1897 were hardly suitable for Stage II.–III. and Stage III. In 1897 tuberculin T.R. (new

tuberculin) was given to us, and the early experiments proved it to be of value especially in simple, early, uncomplicated cases. However, I doubt if T.R. can often help us much in Stage II.–III. or Stage III. T.E. was then tried, and I continued to use T.E. for all cases that were at all advanced; sometimes, however, after some preliminary treament with T.R. On the whole, however, I came to the conclusion that while these preparations were of great value in Stage I. and Stage II., it was questionable whether permanent good could be obtained in Stage II.–III. and Stage III. by either T.R. or T.E. Often it seemed to me that the Old T. was better than T.R.+T.E., especially when the disease was advanced. It was a great step forward when in 1904 and subsequently I began to use P.T. As I have already pointed out, P.T. is useful in all stages, but especially in Stage II.–III. and Stage III.; and a previous course with the milder remedy P.T.O. is very useful, because this preparation is borne so well.

Method.—For very many years I have adopted the plan of injecting the doses into the forearm or outer part of the upper arm. The skin should be rubbed well with spirit, and strict asepsis must be practised. In two cases, in which I allowed a doctor and a nurse in the country to give the doses, abscesses appeared at the site of inoculation. Thereafter I taught many patients to give themselves the doses, which I used to send at certain intervals. In this way I was able to treat cases in New Zealand, Tasmania, Queensland, and in the inland parts of New South Wales.

As I found no serious trouble followed even well-marked reactions, provided they were not too severe nor too frequent, I did not use extreme care to avoid reactions. Indeed, I am convinced that an occasional reaction with fever often does great good, especially in the early stages of the disease, and in the earlier stages of treatment (see records). When no reaction occurs, I generally give another and larger dose in three or four days. If a reaction occurs, I increase the interval according to the degree of fever. Thus, if the temperature be 100° I give the next dose in five days, if 101° in six days, if 102° I wait a week

or more, but I rarely wait more than eight to ten days, except in the case of the larger doses (2 grm., 3 grm., 4 grm. of T.E.) Moreover, if the temperature be above 100·5° I generally repeat the same dose. If the temperature be above 102° it may be wise to diminish the dose or to suspend the injections altogether for three or four weeks. After a fortnight's interval of time it is not wise to increase the dose, and it may be best to diminish it. After a month's interval the dose must be reduced to $\frac{1}{4}$ or $\frac{1}{5}$ of the previous dose. Then it is often an easy matter to proceed to larger doses rapidly—at intervals of three or four days. However, these details depend upon circumstances, and we should always bear in mind Koch's original warning that there must be no routine method, since each case has to be treated on its own merits. Again, it is not easy to say what should be the maximum dose in each case. I think the body weight must be taken into account, and as a matter of principle I try and give as large doses as possible, provided the condition of the patient improves and he gains energy and weight. To a full-sized adult man I give as a maximum dose: 1 grm. P.T., 1 grm. Old T., 2 grms. T.R., 4 grms. T.E. I have used T.O., and T.O.A., and P.T.O. (T.O. for intravenous injection), but I am never satisfied with these alone. According to my experience these doses are not too large, if they can be reached without too severe reactions. It may take four months, six months, eight months, or longer to complete a course of treatment; but seeing that the treatment can be carried out without interfering seriously with a man's business or work, this question of time is of no great importance. If the course takes a long time, the intervals between the doses are also longer.

Besides the injections of tuberculin, I merely give rational instructions to the patient to maintain the tone and vitality of the tissues, and to avoid excitement, over-exertion, and excesses of all kinds. I am sure that alcohol very rarely does any good in pulmonary tuberculosis. Good health depends largely upon good food and good digestion, and

good health must be maintained throughout the treatment. As for drugs, I have no belief in the drugs so much in fashion. I have not given a single dose of creasote, or substances of this class, and I have no intention of ever doing so. They may do great harm; I cannot imagine how they can do good.

PART V

RECORDS

PART V

RECORDS

Introduction to Records.

One cannot pretend to give a full account of several hundred cases, nor is it easy to condense into a few lines the observations of many months. In defining the stage, I have used mainly Turban's scheme of classification. By Stage I., however, I mean that the physical signs, if any, are limited to a small area, chiefly the apex of the lung, not below the level of the clavicle in front and the spine of the scapula at the back. By Stage I.–II. I mean that the physical signs extend rather lower, as far as the second rib. These stages are fairly definite and imply an early stage. If the disease has extended still further, say below the second rib, and involves scattered areas of small size in the upper lobe, it is Stage II. In this stage good results are still possible if the lesion is open, and mixed infection is not present. With mixed infection the expectoration increases in amount and the specific pyogenic organisms abound in the sputum. Then, also, softening is likely to occur, and if it be at all extensive, and especially if similar though less advanced changes are occurring in the other lung, I speak of Stage II.–III. The extent of the dulness and the degree of the softening determine this stage, and with these changes also the character of the adventitious sounds. The amount and character of the sputum help to define this stage. If the physical signs are observed over most of one lobe or over one half of two lobes and the sputum exceeds an ounce or two, Stage II.–III. is present. Any more advanced stage, in

which the whole of the lobe is involved, or evidence of well-marked cavitation exists, is Stage III. It is relatively easy to classify cases when the disease has not passed Stage I.–II. ; but it is often very difficult to define the limits of Stage II., Stage II.–III., and Stage III. Cases of pleurisy, with or without effusion, I define as Stage I. unless there are other lesions elsewhere. The duration of the disease may help but may mislead. Stage I. or I.–II. may persist for a year or more, and on the other hand Stage III. may be reached in six months from the onset of symptoms.

Turban's classification of the stages of pulmonary tuberculosis is largely followed. He recognises three main stages (I., II., III.), with two intermediate stages (I.–II. and II.–III.). It will be convenient to include this classification as it appears in Weicker's "Beiträge zur Frage der Volksheilstätten."

TURBAN'S STAGES OF PULMONARY TUBERCULOSIS.

	Pathological.	Physical Signs.		
		Percussion.	Auscultation. Breath Sounds.	Râles.
Stage I.	Bronchitis, Peribronchitis, scattered foci in one apex. *No or little expectoration.* (Often no tubercle bacilli in sputum.)	Normal to slight, want of resonance over area of one lobe or over half of two lobes at most.	Harsh, weak vesiculo bronchial or broncho vesicular over one or both apices.	None, or crackles fine or medium, over area of altered resonance.
Stage II.	Bronchitis, Peribronchitis, infiltration of slight degree over both supraclavicular & supraspinous or marked infiltration of one lobe *exclusively.*	Decided localised dulness over one lobe or some moderate loss of resonance over half of two lobes.	Harsh to broncho-vesicular breathing over dull areas.	Râles of medium quality.
Stage III.	All cases more advanced than Stage II.	Pronounced dulness may be tympanitic note. (Cracked pot sound.)	Bronchial Tubular Amphoric	Metallic coarse râles, &c.

Cases between I. and II. marked I.–II.
Cases between II. and III. marked II.–III.

Explanation and Plan of Records.

In the subjoined tabulated form of records I have followed in the main Turban's scheme. It is, of course, impossible to do more than give a bird's-eye view. The plan is as follows :—

Name (A or B), age, weight (in stones), family history.
1 = Stage of disease (I, I–II, II, II–III, III).
2 ⌒ if quiescent, → if active and progressive.
3 Duration of disease.
4 State of nutrition : A = good, X = bad.
5 State of digestion : B = good, Y = bad.
6 Pulse rate.
7 Temperature : N = normal, *f* 99°–100·5°, F = (high) 101°–105°.
8 Tubercle bacilli absent or present according to Gaffky : M = mixed infection, S.P. I (streptococci, pneumococci, influenza). Amount of sputum.
9 Tuberculous complications : pleurisy, hæm.
10 Other complications.
11 Energy : capacity for work, *a*, *b*, or *c*.
12 Treatment : previous, present.
Doses of tuberculin always expressed in grammes. ·01 g. or ·01 gr. or ·01 means $\frac{1}{100}$ gramme.

STAGE I.

1902

1

xi.02. 38. 9st. 10lbs.
No family history. Singer. Consulted for strained voice.

1 R. I—II.
2 ⌒
3 1 year.
4 A.
5 B.
6 N.
7 N.
8 No Tb. Reacted.
9 Complete paralysis of *right* vocal cord; nerve involved in scar at apex of lung.
10
11
12 T.R. up to ·2 gr. (103°), then up to 1·5 gr. with moderate reactions. Great improvement in general health. Cord always paralysed. Well.

2

iii.02. 36. 8st. 10lbs.
Sister θφ [1] year ago.

1 R. I—II >.
2 →
3 6 months.
4 X.
5 Y.
6 N. 90.
7 *f* 100°—102°.
8 No Tb. Reacted ·001 102·4°.
9 Pains in left wrist, and in ankles.
10 Later wt. 10st. 10lbs. and 11st.
11
12 T.R. up to 1·8 gr., 10st., many reactions, but quite well for 5 years.
iii.07. Tested. Reacted. Second course P.T.O. to ·9 gr., wt. 10st. 6lbs.; then P.T. up to ·6 gr.
3.vii.07. ·55 gr. P.T. 101°. No doubt injected into veins. Shivering ¼ hour after dose; no swelling in arm; well in evening.
28.vii.07. ·6 gr. P.T. 98·4°.
Well.

3

x.02. 30.

1 R. I—II.
2 →
3 ?
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001, 102·6°.
9
10
11
12 Treated with T.E. to ·25 gr.; then T.R. to ·55 gr.; then Old T. to ·01; then T.R. to 1·7 gr. (101°), wt. 9st. 5lbs.; then T.E. to 1·8 gr., wt. 9st. 9¼lbs.
Some reactions:—
3.v.03. ·3 gr. T.E. 103·6°.
13.v.03. ·4 gr. T.E. 99°.
29.v.03. 1·4 gr. T.E. 102°.
1.vi.03. 1·8 gr. T.E. 100·4°, and gained 2lbs.
Felt quite well.

[1] θφ means died of phthisis.

4

viii.02. 9st. 5lbs.

1 L. I—II.
2
3
4
5
6
7
8 No Tb. ·001 marked local reaction.
9 ·004, 100·4°, and great local reaction.
10
11
12 ·0002 T.E. 99·2°.

viii.02. ·0006 gr. T.E. 103·6°.

25.x.02. ·15 gr. T.E. 103°. Several reactions of 100°—101°. Two intravenous T.O. (·05 and ·1).

17.xii.02. ·1 T.O. 102°.

In Jan. 02. T.O. (intravenous) up to ·25 gr. Wt. at end 9st. 6lbs.

Well when seen some years later.

5

—— 29. 7st. 11½lbs.

Sister $\theta\phi$ and sister's boy has T.

1 R. I—II>.
2 →
3 "Years."
4 X.
5 Y.
6 84—92.
7 *f* (evening 100·2°).
8 No Tb. Reacted.
9 Hæm.: small.
10
11
12 Treated with T.R. In 14 days temp. N.

35.vi.02. ·15 T.R. 100·2. Several doses 100°.

4.vii.02. 1 gr. T.R. 101° wt. 8st. 7¾lbs.

17.vii.02. 1·5 gr. T.R. 101°. 8st. 7lbs.

3.viii.02. 2 gr. T.R. 100°.

12.viii 02. Wt. 8st. 8lbs. Gained 10½lbs. and lost cough.

1908. Well. Married in 1906.

6

—— 26. 7st. 12½lbs.

Brother θ[1] of Addison's disease (T.)

1 R.O. L. I—II.
2 ⌒
3 Some time.
4 X.
5 Y.
6 120—110°.
7 *f* 100, then N.
8 No Tb. ·001, 99·8°; ·002, 100·2°; ·005, 100·4°; ·01 102°, ·01 103·4°.
9
10 Erythema nodosum, and swollen ankles.
11
12 Old T. up to ·2 gr.; then 9st. 10¾lbs.

xii.03. Quite well. 9st. 7¾lbs. Reactions 101°, 102°, 103·4°. First course to ·1 gr.; second course to ·2 gr.

5 years after quite well.

1908. Well.

7

ix.02. 7st. 10lbs.

Missionary in New Guinea.

1 Stage I.
2 ⌒
3 Recent.
4 X.
5 Y.
6 N.
7 *f* + F. Malaria. Double Tertian.
8 No Tb. After treating malaria according to Koch's method, and freeing blood of parasite, I tested with tuberculin. ·001, ·005, 102°. Again ·005, 100·8°
9
10
11
12 In the presence of malaria, testing was out of the question; stained films according to Ziemann's modification of Romanoskwy's method showed double Tertian infection; found some beautiful specimens in the sporulation stage showing achromatic zone and chromatic masses brilliantly. Treatment with T.E. up to 2 gr. Reactions with several doses to 101°, 100·8°, 100°. Improved very much. Returned to New Guinea, and two years later died of acute fever with cerebral symptoms (almost certain "cerebral" malaria).

[1] θ means "died."

1903

8

ii.03. 23. 6st. 6¼lbs.
1 Stage I.
2 ⌒
3 More than a year.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001, 100·2°; ·001, 100°; ·002, 102°.
9
10
11
12 T.R. up to ·8 gr.; few reactions to 100°.
vi.03. 2 gr. T.E.; wt. 6st. 11¾lbs.
x.04. Wt. 6st. 11lbs.
1908. Quite well.

9

iii.03. 22. 11st.
Singer. Mother θφ[1] Peritonitis.
1 R. I—II >.
2 →
3 13 weeks; lost voice.
4 A.
5 B.
6 N.
7 N.
8 No Tb. Reacted. ·001, 100·6°; ·0015, 100·8°.
9 Laryngeal catarrh.
10
11
12 Treated with T.E. to 4 gr.; gained a stone; lost all symptoms; recovered voice.
1906. Married.
1908. Five years after treatment, quite well; wt. 12st. 2lbs.
Quite well.

10

—— 30. 6st. 13¼lbs.
Husband θφ.
1 R. I—II.
2 →
3 Six months.
4 X.
5 Y.
6 N.
7 N. ·001, ·002, 101·4°.
8 No Tb. Reaction. Headache; cough.
9
10 Uterine troubles.
11
12 Treated with T.R. up to 2 gr.; improved greatly in health and looks; wt. 7st. 6lbs.
In 1907 had severe abdominal operation with laparotomy and died next day.

11

viii.03. 36. 8st. 10lbs.
1 R. I—II—II.
2 →
3 Some months.
4 X.
5 Moderate.
6 84.
7 *f.*
8 Tb. Reacted. ·001, 103°.
9 Eul. cervical glands.
10
11
12 Treated with Old T. up to ·2 gr.; lost cough and all symptoms; gained a stone and regained energy.
Was well 3 years after; lost sight of him since; very poor.

12

ix.03. 35. 14st. 9lbs.
Brother, *aet.* 32, θφ.
1 R. I.
2 ⌒
3 Less than a year.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·0035, 102·2°.
9
10 Spinalgia; nervous chronic atrophic rhinitis.
11
12 Treated with T.E. to 4 gr.; after course equal to anything; swam ½-mile; reactions 100° (7.ix. and 12.ix.).
1.ix.03. ·005 gr. T.R. 101°.
28.ix.03. ·2 gr. T.R. 102°.
In 1908, quite well.

[1] θ=θάνατος, φ=φθίσις.

13

v.03. 38. 10st. 2½lbs.

1 R. I—II.
2 ⌒
3 Many years
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001, 99°; ·004, 99·4; ·008, 100·2°, 100·4°; reaction lasting 24 hours; "played out."
9
10
11 *a*.
12 Treated with T.E. to 4 gr.; gained 3lbs. and recovered normal energy. Reactions of 100° for first two months of treatment; and later 101°, 102°, 103·4°. After treatment had a very heavy spell of work. He "could not have got through the work before he had treatment."

In 1908, quite well.

14

iv.03. 19. 9st. 7lbs.

1 I—II.
2 ⌒
3 6 months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001; ·002; ·005, 100°; ·006, 101°.
9
10
11 *a*.
12 Treated with T.E. to 2·8 gr.; gained 8lbs.

Well since.

15

v.03. 19. 8st. 6lbs.

Sister *θφ*.

1 R. I—II.
2 ⌒
3 9 months.
4 X.
5 Y.
6 84—92.
7 N.
8 No Tb. ·003, 100°; ·004, 100·8°; sweats and weakness.
9
10
11
12 Treated with T.E. up to 3·5 gr.; gained 12 lbs.

ix.03. Wt. 9st. 6lbs.; lost cough and expectoration.

Seen late in 1907, quite well.

Control Case.

1902. A.B. 30. Hæmorrhage; Stage I; physical signs, doubtful. No Tb. Reacted ·001, 103°; went out of hospital apparently well; refused treatment; returned to hospital in 1903. Stage III; hopeless.

16

—— 16. 7st. 6lbs.

1 I—II.
2 →
3 Nearly a year.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·00075, 98°; ·003, 100·6; ·004 gr. T.R. 101·6°.
9
10
11
12 Treated with T.R.

15.viii.03. ·0003 T.R. 101·6°.
19.viii.03. ·0006 gr. T.R. 99·6°.
23.viii.03. ·0012, 98°.
30.viii 03. ·004 gr. T.R. 100·2; increased rapidly to 1 gr. T.R.; no more trouble; then T.E. up to 3 gr.
29.iii.04. 3 gr. T.E. 99·4°; wt. 8st. 4lbs. Asthma cured by injection though worse after severe doses.
1908. *Well*; no asthma; 9st.

17

viii.03. 32. 8st. 1lb.

1 R. I—II.
2
3 ? years; sympt. of abd. tub. and tuberculosis of Fallopian tubes.
4 X.
5 Y.
6 90—110; nervous.
7 N.

8 No Tb. ·001, 98°; ·002, 99·6°; ·004, 100·2°; and second day after dose, 98·6°, 101°, 100·2°, 101°, 100°, 100·6°, 100°, 100·4; third day, normal.
9, 10, 11
12 Treated with T.R. to 2 gr.; several reactions of 100° to 100·6°; 5 months after treatment, slight reaction; T.E. up to 2·6 gr.; and in ·05; P.T. up to ·85 gr.; tested May 1907, Old T. (·025) negative.
1908. Delicate, but no sign of tuberculosis.

18

—— 28. 8st.

Sister, mother, and brother $\theta\phi$.

1 R.O. L. I—II.
2 →
3 6 months.
4 A.
5 Y.
6 N.
7 N.
8 No Tb. ·001, 99·2°; ·002, 100·4°.
9—11 Nil.
12 Treated with T.E. up to 4 gr.; gained 8lbs.; lost cough; very severe cramps, with reactions; had to go to bed.
May 1906, did not react to tuberculin (·02 Old T.).
1908. Well.

19

iv.03. 28. 7st. 12lbs.

1 R.O. L. I; also interscapular signs.
2 →
3 Suffered from "gastritis" and vomiting for 15 months; sent to hospital as "gastric ulcer."
4 X; 7st. 12lbs.
5 Y; epigastric pain; good appetite but vomiting all food.
6 76.
7 *f.*
8 No Tb. ·001 Old T. 98·8°; ·004 Old T. 100·2°.
9–11 Nil.
12 Treated with T.E.; in 6 months, wt. 9st. 11½lbs.; T.E. up to 2 gr.; gained 27lbs.
Saw her afterwards, was quite well.
Well in 1906; not seen since.

20

ix.03. 33. 6st. 9lbs.

Sister reacted.

1 R. I L. I.
2 ⌒
3 A year.
4 X.
5 Y; no appetite.
6 N.
7 N.
8 No Tb. ·001, ·004, ·008; 100·4°, 99·2°, 100 5; delirious, and severe headache and pains persistent.
9 Interesting acnelike skin lesion only found in tuberculous subjects (Dr. Munro).
1908. Well.

21

—— 17.

1 Stage I.
2 →
3 Recent hæm.
4 A.
5 B.
6 N.
7 N.
8
9–11
12 Had severe hæm. recurring; gave tuberculin for a month; hæm. ceased, and more than a year later no recurrence, and patient apparently well. Well two years later.

1904

22

ii.04. 24. 9st. 6lbs.

1 R. I—II.
2 →
3 1 year.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001, 99·8°.
9
10
11
12 Few doses of T.E. sensitive; then P.T. to ·175; reactions at first, even 103°.
xi.04. 9st. 10lbs.; tested in 1905, ·02 Old T. 100°. Continued treatment.
Heard of him in 1907, but have not seen him since 1906.

23

ix.04. 6. 2st.

1 Hydatid disease of lung and bronchial tuberculosis.
2
3
4
5
6
7
8 No Tb. Reacted. ·0005, 101°.
9–11 Liable to colds.
12 Case sent in as one of pulmonary tuberculosis; deficient movement and absolute dulness at right apex; but bronchial breathing, occasional clicks; treated with tuberculin; P.T.O. up to ·9 gr.; in two months gained 10 lbs.; then suspected hydatid disease in right lung; X-rays proved presence of hydatid. Successful operation and now (1908) child is well. Tested in 1906, ·01 Old T. ——

24

xi.04. 23. 8st. 9½lbs.

1 R. I—II.
2 ⌒
3 Within a year.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001—·0045, 100·6°; gen. and local sympts.
9
10
11
12 Treated with T.E. to ·55 gr.; caught cold; pause for 18 days; then ·35 gr. T.E. 103·4°. Cold irritated focus and made him more sensitive; T.E. again up to ·35 gr.
vi.05. Wt. 8st. 13lbs.; no cough or phlegm, and great gain in energy.
1908. Well.

25

ii.04. 20. 8st. 9½lbs.

Father and mother well. One sister $\theta\phi$.

1 R. I—II L I.
2
3 6 months.
4 X; lost 10 lbs.
5 Y.
6 N.; irritable weakness.
7 N.
8 No Tb. ·0015, 100·2°; ·003, 102°; severe sympts.
9
10 Insomnia; nervous.
11 *b*.
12 Treated with T.E. up to 4 gr.; till ii.05; pause in June and July.
vi.05. 9st. 6½lbs.; never so heavy before, sleeps quite well now.
Reactions:—
13.iii.04. ·02 gr. T.E. 102·6°.
18.iii.04. ·02 gr. T.E. 98·4°.
22.iii.04. ·04 gr. T.E. 100·8°.
24.iii.04. ·06 gr. T.E. 98·4°.
16.iv.04. ·27 gr. T.E. 101.4°.
Sileat Wright.
1908. Well.

26

vi.04. 28. 9st.

1 R. I.
2 —→
3 9 months.
4 A.

5 B.
6 N.
7 N.
8 No Tb. ·001—·002, 99·8°; headache and vomiting.
9
10
11
12 Treated with T.E. ·0002 gr. T.E. 100·4°, ·0003 gr. T.E. 100·6°, T.E. up to ·2 gr.; then T.R. and T.O.; returned to T.E.; T.E. up to 1·5 gr. (101·6°); 8st. 13½lbs.
vi.05.
2 gr. T.E. 102·4°; wt. 9st. 2½lbs.
Well since.

27

x.04. 22. 5st. 9lbs.
Sister of Mrs. Sharp.

1 I—II.
2 —→
3 6 months; treated for "gastric ulcer," vomiting, &c.
4 X. 5st. 9lbs.
5 Y. Vomiting and pain in stomach.
6 98.
7 /.
8 No Tb. ·00125 Old T., 102·8°; ·001, 101·8°; 001, ·100·4°.
9
10
11
12 Treated with T.E. up to ·225 gr.; gained 10lbs; 6st. 5½lbs; lost cough and gastric symptoms.
Felt well, and being a hospital case, went to a situation in country.
Well since, no return of gastric symptoms.

28

xi.04. 22.

1 Case of tub. arthritis of wrist-sinus formerly into hand, now closed; diagn. as gon.; arthritis no other joint affected, and all the appearance of a tub. arthritis, swelling, stiffness.
2 →
3 One year.
4–7
8 Test ·001, Old T. 102·2°. Reacted many times to tuberculin, but patient played tricks with thermometer, and had to be watched; also suspected of applying tight ligature around arm to excite pity.
12 Under tuberculin great improvement in joint and arm, and gained nearly a stone.
P.T. up to ·159 gr.; reactions (100°, 102°); then Old T. up to ·4 gr.; wt. 8st. 10¾lbs.

29

iv.04. 24. 8st. 11lbs.
Mother θφ ("galloping").

1 R. I—II. Dr. P. and MacC. "weak lungs."
2 (·)
3 Few months.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001; ·004, 100°, ·005, 100°; ·008, 102°.
9
10 Hysteria.
11
12 T.E. up to 3·5 gr.; few reactions 100°—101° gained 5½lbs.
Well in 1906; not seen since.

30

ii.04. 17. 9st. 4lbs.

1 R. I—II.
2 →
3 6 months.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001 Old T., 102°; 100°, 102·4°; severe symptons.
9–11
12 Treated with T.E.
22.iv.04. (3rd dose) ·0005 gr. T.E. 101·4°; then T.R.; no reaction till 15.vi.04. ·85 gr. T.R., 102°; wt. 9st. 9lbs.
Pause ix.04; 10st. 6½lbs.; then in x.04 T.R. to ·4 gr.; then P.T. to ·125 gr.
Mother thought he had had enough treatment; wt. 10st. 8lbs.; gained 18lbs.
Well a year later.

31

iv.04. 23. 8st.

1 R. I—II.
2 →
3 Few months.
4 X.
5 Y.
6
7
8 No Tb. ·001 gr. Old T., 100·4; ·0015, 100°.
9–12 Nil.
12 Treated with T.E.

24.iv.04. ·0005 T.E. 100°, 8st.
1.v.04. ·0008 gr. T.E. 100°.
4.v.04. ·001 gr. T.E. 98·4°.
6.v.04. ·002 gr. T.E. 100·2°.
11.v.04. ·0025 gr. T.E. 100°.
18.v.04. ·005 gr. T.E. 101·4°.
23.v.04. ·01 gr. T.E. 99·2°.
25.v.04. ·02 gr. T.E. 98·4°.
27.v.04. ·04 gr. T.E. 102·4°.
3.vi.04. ·05 gr. T.E. 100·4°, 8st. 4¼lbs.; then T.E. up to 1·8 gr., 8st. 8lbs.; gained 8lbs.

Not seen since 1904.

32

x.04. 38. 8st. 4lbs.

1 R. I—II, L. I.
2 →
3 Influenza half year ago.
4 X.
5 Y. Symptoms of severe nervous dyspepsia; poor appetite.
6 N.
7 N.
8 No Tb. ·001 gr. Old T., 103·2° gr., six hours.
9
10 Dyspepsia for *six* months.
11 Treated by Dr. J. for some months.
12 Treated with T.E. up to ·2 gr.; few reactions in first two months (100°, 101°, 101·6°, 100·6°); then on to ·6 gr. T.E.

Pause from Dec. to Mar., 1905; then T.E. up to 3·5 gr. (101°); wt. 8st. 4lbs.

Gastric symptoms greatly improved.

Knew her to be well in December, 1907.

33

ii.04. 49. 7st. 8½lbs.

1 R. I—II, L. I?
2 →
3 1 year.
4 X. Loss of wt.
5 Y.
6 80.
7 N.
8 No Tb. ·001 Old T. 101°.
9
10 Dyspepsia; bilious attacks.
11
12 Treated with T.E.

22.vi.04. ·002 gr. T.E. 102°; later reactions, 100°, 100·6°; T.E. up to ·15 gr.; then T.R. up to 1·2 gr.
15.vi.04. 8st. 3½lbs.
18.vii.05. 9st. 1¼lbs.

Gained 20¾lbs.; feels quite well and able to do anything.

1907. Still well.

34

x.04. 20. 9st. 7lbs.

1 R. I—II.
2 ⌒
3 Six months.
4 X.
5 Y.
6 100—110 (cardiac trouble).
7
8 No Tb. Reacted.
9 Treated with T.E. up to ·4 gr.; then P.T.O. up to 1 gr.

Pt. improved, but there was also cardiac trouble in this case; I saw patient in 1906.

Well so far as lungs were concerned.

1905

35

vi.05. 24. 9st. 13lbs.
Sister $\theta\phi$ 4½ years with her.

1 R. II.
2 →
3 Cough for 18 months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·002 gr. Old T. 102·6°; severe symptoms.
9 Hæmorrhages.
10–11
12 P.T. up to ·15 gr.; severe pains in neck and left flank after doses. Improved greatly and hæmorrhages checked; seen late in 1907, then well.
Advised to have more treatment.

36

ix.05. 18. 8st. 1¼lbs.

1 R. I—II.
2 →
3 Few months.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. Old T. 101°.
9–11
12 Treated with T.E. to 4 gr.
1.iv.06. 2·1 gr. T.E. 101°; 8st. 3¼lbs.
Treated while she was at work; engaged all day long in making shirts.
1908 quite well and strong.

37

i.05. 28. 6st. 3lbs.
Sister in Stage III always with her.

1 R. I—II.
2 →
3 Few months.
4 X.
5 B.
6 N.
7 N.
8 Tb. in sputum G. 3; ·003 gr. Old T. 102°.
9–11
12 Treated with T.E. to ·03 gr.; very sensitive. Reactions, several 102° and 101°; still gained wt. 6st. 9lbs.; then P.T. up to 85 gr.; wt. 6st. 10lbs. Very great improvement; lost cough, no phlegm. Gained 8lbs. under treatment.
Went back to home in New Zealand.

38

—— 20. 9st. 7lbs.
Other doctor same diagnosis.

1 R. I—II.
2 →
3 6 months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 101°; severe in arm and severe symptoms.
10 Nervous, felt doses very much.
12 Treated with T.R. up to 2 gr. Moderate reactions, but severe subjective symptoms, especially severe pains.
xii.05. 10st. 9lbs.; no cough or phlegm.
Gained 16lbs. under treatment. Married in 1906, and has had a child.
1908 well.

39

—— 37. 9st. 8¾lbs.
Gardener.

1 R. I—II; ordered to country.
2 →
3 Cough for years.
4 X. Lost 3 stone.
5 Y.
6 N.
7 N.
8 No Tb. Reacted.
9–11
12 P.T. up to ·05 gr.; gained 4lbs. Said he felt strong and able to work, and in 1908 was apparently well. 9st. 12¾lbs.; gained 4lbs. in two months.
Heard of as well late in 1907.

40

i.05. 27. 8st. 12lbs.
Sister in Stage III.

1 R. I—II.
2 →
3 6 months.
4 X.
5 B.
6
7
8 No Tb. ·001, mistake in recording; ·005—·01, 103·2°; headache, vomiting, herpes, severe cough.
12 Treated with T.E. to 3·5 gr. Moderate reactions; wt. 10st. 1lb. In spite of reactions quite well. All Wright's ideas disregarded. Gained 17lbs.
Well.

41

—— 27. 8st. 11½lbs.
Dr. O'N., same diagnosis.

1 R. II, L. I.
2 →
3 Cough often?
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001? ·004 gr. T.A. 103°; shivering, cough, vomiting, pains in limbs.
9–11
12 Treated by Dr. O'N. with creasote, caused vomiting each time, gave it up. Treated with T.R. ·0015 gr. T.R. 103; 8st. 13lbs.; T.R. up to 1·75 gr.; lost cough and phlegm; plenty of energy; never tired now. Pains in same knee after each dose and knee swollen, also in ankles at times; slipped, and twisted knee, and since then pain after each dose.
Seen Dec., 1907, quite well.

42

vi.05.

1 R. I—II.
2 ⌒
3 6 months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001—; ·005—; ·01,98, 100°, 100·2°, 100°; symptoms.
12 Treated with T.E. to ·01 gr.; then T.R. to ·6 gr. Dose affected sight, saw rings and blurs.
Left off treatment on account of private troubles. Actress.

43

—— 34. 11st. 6lbs.
No family history.

1 R. I—II.
2 ⌒
3 6 months.
4
5
6
7
8 ·001 gr. Old T. (T.A.) 100·4°; ·001, 99°; ·005, 101·3°.
9
10
11
12 Treated with T.E. up to 4 gr. Recovered energy and felt quite well.
Well in 1908.

44

ix.05. 21. 8st. 4lbs.

1 R. I—II.
2 →
3 6 months.
4 X.
5 Y.
6 92·100.
7 *f.*
8 Tb.? Reacted ·003 Old T. (T.A.) 101·8°; severe symptoms.
9 Pleurisy.
10–11 *c.*
12 Treated with T.E.; some severe reactions in hospital; 102°, 102°, and afterwards 101°, 102°, but lost cough; recovered strength and gained 28lbs. T.E. up to 2·4 gr.
vi.06. wt. 10st. 7lbs.; *gained* 31*lbs.*
Have not seen since.

45

v.05. 8. 3st. 5lbs.
Father 3 years ago θφ.

1 R. I—II, and dulness into interscapular space (bronchial tuberculosis).

2 →
3 5 months ; cough and wheezing.
4 X.
5 Y.
6 100—110.
7 N.
8 No Tb. ·0002 gr. T.A.—·0005—·001, 103°, 99°, 100·6.
9 Bronchial tuberculosis.
10 Bronchitic signs.
11 *b.*
12 Treated with P.T. up to ·125 gr. ; vomiting and pains in stomach after doses ; after ·02, ·03, ·04, ·1 and ·125 gr. reactions 100°—101°.
xii.05. Wt. 4st. 5¾lbs. ; also tested with T.A. ; did not react to ·02 gr. T.A.
xi. 1907 quite well ; wt. 5st. 3lbs. ; *gain of 26lbs.*, no doubt some due to growth.

46

ii.05. 20. 8st. 3lbs.
Sister θφ, father and mother well.

1 R. I—II.
2 →
3 6 months.
4 X.
5 Y.
6 N.
7 *f.* 99·99·6° for days.
8 No Tb. ·001 gr. T.A. 99·8°; ·005 gr. T.A. 100·4° ; arm much swollen and râles at right apex ; cough worse.
9–10 Nil.
11 *b.*
12 T.E. up to ·75 gr. ; several severe reactions, ·75 gr. T.E. 103° ; still wt. incr. to 9st. 6lbs. ; then P.T. up to ·325 gr. ; at end, wt. 9st. 10¾lbs. ; gained 21¾lbs. Had always lived in country (*Dubbo*).
1907 well.

47

vi.05. 30. 6st. 5lbs.

1 R. I—II> L. I.
2 ⌒ well-marked signs.
3
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A.—; ·005—; ·01, 99·8°, 100°.
9
10 Asthma and insomnia from cough.
11 *b.*
12 Treated with P.T. up to ·45 gr. ; gained 1½lbs. but feels much better ; sleeps very well now and looks better ; used to cough through the night, now does not cough.
1907. Well.

48

vii.05. 34. 9st. 8lbs.

1 R. I—II, also scattered foci of pleurisy.
2 →
3 3 to 6 months.
4 A.
5 B.
6
7
8 No Tb. ·0005gr. T.A.—; ·0018—; ·007, 103° for three hours ; severe sympts.
9 Pleurisy over half right lobe.
10
11
12 Began with ·004, very severe reactions.
2.viii. 3 A.M. 103°.
3.viii. 2 P.M. 105°.
4.viii. 5 P.M. 103°.
Then treated with T.R. up to 2 gr. ; wt. 11st. 5½lbs. ; lost pain and cough Jan. 1908 ; well ; severe reactions did no harm ; gained 25½lbs. ?
1908. Well.

49

vi.05. 30. 8st. 8lbs.
No family history.

1 R. I—II.
2 ⌒
3 Pleurisy in 1900.
4 X ; discoloration of skin.
5 Y ; anæmia.
6 120.
7 N.
8 No Tb. ·001 gr. T.A. 99·6° ; ·003, 100·2°.
9
10 Splenic anæmia, enl. spleen (Banti's disease).
11
12 Treated with T.E.
7.vi.05. ·003 gr. T.E. 103°.
17.vi.05. Began with T.R.
24.vi.05. ·0005 gr. T.R. 101°.

14.vii.05. ·01 gr. T.R. 100·2°.
29.vii.05. ·1 gr. T.R. 100°.
26.viii.05. ·2 gr. T.R. 101°.
2.xi.05. ·25 gr. T.R. 103·4°; wt. 8st. 10lbs.
18.xi.05. ·9 gr. T.R. 101°.
1.xii.05. Used P.T. up to ·325 gr.; wt. 9st. 10¾lbs.
Became pregnant during treatment, but aborted; treated by local physician.
Died in vi.1907 of Banti's disease or splenic anæmia.

50

iv.05. 17. 8st. 9lbs.
No family history.

1 R. I—II.
2 Sympts. of asthma.
3 For some months.
4 X.
5 Y.
6 76.
7 N.
8 No Tb. ·001 gr. T.A.; said to be 104°; more likely 100·4°.
9 Repeated. ·001 gr. T.A. 101·4°.
10 Asthma and bronchitic râles.
11 *b*.
12 Treated with T.E.
27.iv.05. ·1 gr. T.E. 102°.
22.v.05. ·5 gr. T.E. 101°.
29.v.05. ·55, 102°.
5.vi.05. ·55, 102°.
12.vi.05. ·02 P.T. 101°.
17.vi.05. ·0275 P.T. 99·6°.
1.vii.05. ·2 P.T. 103°.
10.vii.05. ·2 P.T. 99·2°.
16.vii.05. ·25 P.T. 101·4°.
23.vii.05. ·25 P.T. 101·4°.
2.viii.05. ·2 P.T. 100°.
7.viii.05. ·3 P.T. 100°.
14.viii.05. ·35 P.T. 99·2°.
21.viii.05. ·4 P.T. 99°.
27.viii.05. ·5 P.T. 99°.
10.ix.05. ·75 P.T. 98·4°; wt. 9st. 4lbs.
Sileat Wright.
1907. Tested, negative.
1908. Well.

51

—— 42.

1 Stage I.
2 ⌒
3 4 months ago Pleurisy.
4 Thin.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100·2°.
9–11
12 Treated with P.T. up to ·65 gr.; early dose caused temp. of 101·2°, otherwise no trouble; lost cough and felt well.
Believe him to be well, though I have not seen him.

52

i.05. 38. 6st. 5lbs.

1 Stage I—II.
2 →
3 ?
4 X; 6st. 5lbs.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100·4°; 002, 100°.
9–11
12 Treated with P.T. up to ·05 gr.; then T.E. up to 4 *gr.*
vi.06. "Can walk splendidly and "do anything now—could not "walk when I came." Wt. 7st. 2¾lbs.; gained 11¾lbs.
Seen since, quite well.

53

x.05. 17. 8st. 11lbs.

1 Stage I.
2 →
3 Influenza? five months ago, temp. 100—102°; for five weeks cough, phlegm, sweats, short of breath.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. Old T. 99·5°; ·002 gr. Old T. 99·8°; ·005 gr. Old T. 101°.
9
10 Fits of coughing, due to enlarged lingual tonsil.
11 *b*.
12 Previous treatment: for three months treated for cough and colds, and patient got gradually

worse till she came to me and Tuberculin was used.
Tuberculin treatment: treated with P.T. up to ·65 gr.; she gained wt., recovered health and energy and has no cough or phlegm; wt. 9st.
Seen in 1908, quite well.

54

xii.05. 22. 10st. 3lbs.

1 Stage I.
2 ⌒
3 Cough for 2 years.
4 X.
5 Y; indigestion.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100°; ·002, ?; ·005, 101·2°.
9–11 Nil.
12 Treated with P.T. up to ·65. 10st. 6½lbs.
22.viii.06. 11st. 1½lbs. Never felt so well in her life; lost cough, and no indigestion.
viii.06. Tested up to ·03 gr. T.A.——

54a

xi.05. 24. 8st.
Brother θφ (galloping φ).

1 R. I—II.
2 ⌒
3 Recent.
4 X.
5 B.
6 N.
7 *f* 99·2°.
8 No Tb. ·01 gr. T.A. 100·6°.
9
10
11
12 Treated with T.E. up to ·35 gr.; gained 5lbs. and lost cough.

55

xi.05. 26. 7st. 7lbs.
No family history.

1 Stage I.
2 →
3 6 months to a year.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 99·2°; ·002, 99·8°; ·005, 100·4°.
9 Hæmorrhage, small for some days.
10–11
12 Treated with P.T. up to ·7 gr.; gained 3lbs.; never so heavy in life; looked very well; no cough.
Tested vi.06. up to ·03——
1908. Well.

56

1905. 50. 7st. 13lbs.
Bad history in cousins.

1 I—II.
2 ⌒
3 1 year.
4 X.
5 Y.
6 80—96.
7 N.
8 No Tb. ·001 gr. T.A. 101°.
9
10 Neurotic; injections very painful.
11
12 T(reated) with T.E. up to ·03 gr.; reactions, 100·2°, 100·8°, 101·6°, 102°, 101·6°; still, wt. 8st. 8lbs.; then T.R. up to 1·2 gr.; lost 3 or 4lbs.; then few doses of P.T.O. (·2 gr.) and T.E. (·75 gr.); wt. 8st. 7lbs.; then P.T.
i.07. P.T.O. up to ·75 gr.; then P.T. up to ·5 gr.
iv.07. 8st. 11¾lbs; "never so heavy "before"; gained 12¾lbs.
1908. Well.

57

viii.05. 22. 9st. 4lb.
Sister θφ.

1 R. I—II. L. II.
2 →
3 1 year.
4 X.
5 B.
6
7
8 Tb. 9·5—3j.
9 Hæmorrhages.
10–11
12 Treated with P.T. up to ·6 gr.; 9st. 13lbs.; many reactions, 100°, 101°, 102°.
29.xii.05. ·5 P.T. 103·4°.
8.i.06. ·5 P.T. 101°.
15.i.06. ·55 P.T. 101°; wt.

9st. 13½lbs.; great improvement in health and physical signs; no cough and no phlegm; gained wt.; few clicks now; before crackling and bubbling râles.
Not seen since.

58

xii.05. 16 10st.
Brother in Stage II—III.
1 Stage I (R. I—II).
2 ⌒
3 Few months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·00750 gr. T.A. 103°; very severe in arm and sympts.
9
10–11
12 Treated with P.T., sensitive.
2.i.06. ·002 P.T. 102·4°; previous smaller doses also ·0004 gr. P.T. 100·6°; ·0004 gr. P.T. 100·4°; then P.T.O. up to ·65 gr.
Apparently well, but have advised more treatment if test be positive.

59

vi.05. 21. 12st. 5lbs.
Mother θφ.
1 Stage I; pronounced healthy by four doctors; passed for A.M.P. Society and passed by railway doctor.
2 →
3 Short of breath for 6 months, and loss of energy.
4 A.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 102·2°.
9–10
11 *b.*
12 Treated with P.T.; after, a few smaller doses.
3.vi.05. ·005 gr. P.T. 102°.
10.vi.05. ·005 *gr. P.T.* 100°.
13.vi.05. ·0075 *gr. P.T.* 99°.
20.vi.05. ·015 *gr. P.T.* 98°.
3.vii.05. ·05 gr. P.T. 103°; no further reaction above 100°; P.T. continued up to ·9; patient was tested in May, 1907, wt. 12st. 13lbs., and did not react to ·045 gr. T.A.; he has regained all old energy; can do anything and is very strong; he had hard work in the railways and continued at work throughout treatment *in spite of reactions*; I arranged larger doses so that reactions occurred when he was off duty.

1906

60

iv.06.
No family history.
1 Stage I.
2
3 Cough 3 years; phlegm 2 years.
4 X.
5 B.
6 N.
7 N.
8 No Tb. Reacted.
9-11
12 P.T.O. few doses; then treated with P.T. up to ·25 gr.; wt. 11st. 6lbs.
Feels quite well, had to return to country.

61

iv.06. 25. 9st. 4lbs.
Brother θφ vii.06.
1 R. I—II; crackling râles.
2 →
3 Cough for 14 days.
4 Lost a stone.
5 B.
6
7
8 No Tb. Reacted.
9–11
12 Treated with P.T.O. to ·1 gr. with reactions; then T.O.A. up to ·075 gr.; then P.T.O. up to 1 gr.; 9st. 12lbs.; then P.T. up to ·85 gr. (ix.06); tested 1·07 up

to ·035 gr. T.A. (Old T.); wt. 10st.; gained 10lbs., and feels and looks well.

62

—— 24. 7st. 11lbs.

Pregnant.

1 R. I—II.
2
3 Recent.
4 X.
5 Y.
6
7
8 No Tb. ·001 gr. T.A. ·003, 99·6°; ·006, 100·6°, 100·8°.
9
10–11
12 Treated with P.T.O. up to 1 gr.; then P.T. up to ·7 gr.; gained weight and strength.

63

ii.06. 23. 9st. 8lbs.

Mother *aet.* 54, *θφ*.

1 R. I—II.
2 →
3
4 A loss of strength.
5 B.
6 N.
7 N.
8 No Tb. ·002 gr. T.A. 102°; phlegm, half an ounce, yellow.
9–11
12 Treated with P.T.O. up to 1 gr.; then P.T. to ·35 gr.; 10st. 6lbs.; had to go off to shearing; felt well and strong, gained 12lbs.

64

—— 24. 8st. 5lbs.

Spent much time with boy who died of *φ*.

1 R. I—II. Pleurisy and râles.
2 →
3 Recent.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·0015 gr. T.A. 100·4°; swollen arm, headache, nausea, sleepy.
9–11
12 Treated with P.T. up to ·75 gr.; 8st 11¼lbs.; gained 6¼lbs., and felt quite well; no physical signs.

65

ix.06. 33. 8st. 12lbs.

1 Stage I—II.
2 →
3 Six months. Pneumonia six months ago, cough ever since, much blood in expectoration.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·01 gr. T.O.A.—; ·04—; ·1 T.O.A. 103°.
9
10 Pn. in iii.06; ill six weeks, cough ever since.
11 C.
12 Treated with P.T.O. up to ·45 gr.; then T.O.A. up to 1 gr.; reactions 101° (·025 gr.); 102° (·125); then P.T. up to ·75 gr.
Lost cough, looks very well, infinitely better, and feels quite well. Went back to New Zealand.

66

iii.06. 30. 8st. 6lbs.

Advised by doctor to see me. Brother *θφ* and sister *θφ*.

1 R. I—II.
2 →
3 Cough and hoarseness for 2 years.
4 X. Lost a stone in 10 months.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 101°; arm swollen, cough and headache.
9
10
11 *a*.
12 Treated with P.T.O. to ·7 gr. In 3 months no cough, and wt. 8st. 13½lbs.; working all the time; afterwards P.T. up to ·775 gr.; wt. 9st. 1lb; tested 9 months later; ·005, ·015, ·03, ·045, gr. T.A. all negative.
Since quite well and looks very well.

67

—— 32. 12st. 4lbs.
Sister $\theta\phi$, also husband has ϕ.

1 R. I—II.
2 →
3 Cough for 18 months.
4 A.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 101·4°; very severe in arm.
9-11 Nil.
12 Treated with P.T.O.
18.vii.06. ·003 P.T.O. 100·6°.
23.vii.06. ·003 P.T.O. 102·6°.
29.vii.06. ·0015 P.T.O. 101°; then T.R.
8.viii.06. ·0002 T.R. 99°.
11.viii.06. ·0003 T.R. 100·4°.
24.ix.06. ·015 gr. T.R. 102°.
2.x.06. ·015 gr. T.R. 99·2°.
7.x.06. ·02 gr. T.R. 99°.
10.x.06. ·03 gr. T.R. 98°.
12.xii.06. 1·2 gr. T.R. 98°.
20.xii.06. 1·45 gr. T.R.
27.xii.06. 1·79 gr. T.R.
Sister's death interrupted treatment.
Well ever since.—Sileat Wright.

68

viii.06. 35. 8st. 5lbs.
Eldest sister $\theta\phi$.

1 R. I—II.
2 ⌒
3 Very liable to colds for years.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·002 gr. T.A. 100·5°.
9 Catarrh ?
10 Frequent colds.
11
12 Treated with P.T.O.
8.viii.06. ·003 gr. P.T.O. 103°.
9.viii. 100°, 102°.
15.viii.06. ·001 gr. P.T.O. 101°.
21.viii.06. ·001 gr. P.T.O. 99·2°; no further reactions (Sileat Wright); P.T.O. up to ·8 gr.; then T.O.A. up to 1 gr.; then P.T.
20.xii.06. ·175 gr. P.T. 100·8°.
27.xii.06. ·225 gr. P.T. 98·4° (Sileat Wright) on to ·525 gr. P.T. 98·4°.
vii.06. Tested, ·02 Old T. 100·5°; further course with T.R. up to 2·2 *gr.*; no reactions; wt. 8st. 7¾lbs.
Dec. 1907. Very well.

69

viii 06. 17. 8st. 12lbs.
Mother in Stage III.

1 Stage I.
2 →
3 Recent (3 months).
4 A.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A.—; ·0025—; ·005, 100·2°, 101°, 101·4°, 101°, 98·98° 97·2°.
9–11. Nil.
12 Treated with P.T.O. up to 1 gr.; then T.O.A. ·8 gr.; then P.T. up to ·5 gr.; lost cough, no phlegm, appetite good; feels quite well. 1907.

70

viii.06. 16. 7st. 8lbs.
Father $\theta\phi$ in 1900; also half-brother and sister $\theta\phi$. Other father no ϕ.

1 R. I—II. Well marked.
2 →
3 6 months.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100°; ·002, 101°.
9–11. Nil.
12 Treated with P.T.O.
13.viii.06. ·003 gr. P.T.O. 100°.
15.viii.06. ·003 gr. P.T.O. 103·2°.
22.viii.06. ·c015 gr. P.T.O. 99°.
28.viii.06. ·002 gr. P.T.O. 99·4°.
5.ix.06. ·003 gr. P.T.O. 99°.
9.ix.06. ·005 gr. P.T.O. 99·4°.
16.ix.06. ·01 gr. P.T.O. 103·2°.
24.ix.06. ·01 *gr. P.T.O.* 98°.
30.ix.06. ·02 gr. P.T.O. 98°; no further reaction.
30.xii.06. ·85 gr. P.T.O. 100°.
6.i.07. 1 *gr. P.T.O.* 98·4°; then P.T.
17.i.07. ·03 gr. P.T. 100°.
22.i.07. ·04 *gr. P.T.* 99°.

10.iii.07. ·375 gr. P.T. 101°.
20.iii.07. ·375 *gr. P.T.* 99°; and afterwards no more reactions.
5.v.07. ·7 *gr. P.T.* 98·4°.
Tested x.07, ·005, ·015. ·025, ·04; throughout patient was at work all day long from 9 till 6, except for first 2 months of treatment; not a day out of Sydney.
1908. Well.

71

x.06. 26. 9st. 8lbs.
Brother *aet.* 46, *θφ*.

1 R. I—II, L, I.
2 ⌒
3 7 months ill.
4 Usually 8st.; after rest, 9st. 11lbs.
5 B.
6 N.
7 N.
8 No Tb. ·01 T.O.A.—; ·03 T.O.A., 99·4°; ·01 T.O.A., 101·2°; severe reaction in arm.
12 Treated with P.T.O.; very sensitive.
30.x.06. ·005 gr. P.T.O. 102·4°.
5.xi.06. ·0025 gr. P.T.O. 100·8°.
8.xi.06. ·0025 gr. P.T.O. 99°.
13.xi.06. ·003 gr. P.T.O. 99·6°.
16.xi.06. ·005 gr. P.T.O. 101·6°; then T.R.
30.xi.06. ·002 T.R. 100·2°.
25.i.07. ·2 gr. T.R. 100·5°.
4.ii.07. ·25 gr. T.R. 100·2°; no reaction till
10 iii 07. 1·4 gr. T.R. 102·8°.
18.iii.07. 1·3 gr. T.R. 98·4°.
18.v.07. 2 gr. T.R. 99·8°.
Felt well.
Well late in 1907.

72

viii.06. 27.
Husband in second stage *φ*.

1 R. I—II.
2 →
3 Few months.
4 X.
5 Y.
6 N.
7 *f.* 99·4°; 99·6°; E.
8 No Tb. Reacted; ·001 gr. T.A. 101°?
9—11 *b.*
12 Treated with P.T.O.
1.ix.06. ·002 gr. P.T.O. 101·6°.
8.ix.06. ·002 gr. P.T.O. 99°.
12.ix.06. ·003 gr. P.T.O. 100°.
21.x.06. ·02 gr. P.T.O. 100°.
23.xi.06. ·08 gr. P.T.O. 99·6°; then T.R.
18.i.07. ·1 T.R. 99·4° up to 1·85 gr. 100°.
1908. Apparently well, but should be *tested.*

73

iii.06. 30. 10st. 6lbs.

1 R. I—II. R. I.
2 —
3 Since Christmas 1905 (4 months), hæm. in ii.06.
4 X.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 102·5°.
9 Hæmorrhages moderate.
10 Influenza? and sweating.
11 *b—c.*
12 Treated with P.T.O. up to ·9 gr.; T.O.A. up to ·5 gr.; wt. 11st. ½lb.; then P.T. up to ·125 gr.
1.vi.06. T.E. up to 1 gr.; *wt. 11st. 7lbs.* Pause.
Then:
1.ix.06. ·175 P.T. 100·2°.
8.ix.06. ·2 gr. P.T. 100°.
16.ix.06. ·25 gr. P.T. 101·2°.
23.ix.06. ·25 gr. P.T. 100·2°.
30.ix.06. ·3 gr. P.T. 99·8°.
7.x.06. ·35 gr. P.T. 99·8°.
14.x.06. ·4 gr. P.T. 101°. Pause. Then P.T. again up to ·85 *gr.*; wt. 11st. 5½lbs.; later, 11st. 13lbs.; gained 21lbs.; working as tram conductor all the time; now looks and feels well.

74

iii.06. 18. 8st. 4lbs.
Cousin *θφ*.

1 R. II. L. I—II.
2 →
3 1 year.
4 X.
5 B.
6 N.
7 *f.*
8 Tb. G. 3. ℥ij sputum.
9-10-11. Nil.

12. Treated with P.T.O. ; gained 7lbs.
11.iv.06. ·85 gr. P.T.O.
So much improved in every way that he did not think he needed any more treatment. Have not seen him since.

75

ix.06. 8. 2st. 13lbs.
Hospital case.

1 Tuberculous adenitis ; enlarged glands in both submandib. regions, esp. right.
2 →
3 3 months.
4 A. 2st. 13lbs.
5 B.
6 72.
7 *f.*
8 No Tb. ·00025 Old T. 101° ; typical reaction in arm and in right knee, some pain.
9 Tuberculous disease of knee a year ago.
10
11
12 Treated with P.T.O. up to ·325 gr. ; wt. 3st. 6½lbs. ; then P.T. up to ·1 gr. ; wt. 3st. 6lbs.
Glands much smaller ; knee feels quite well ; great improvement.

76

xi.06. 18. 10st. 12lbs.
Sister $\theta\phi$.

1 Stage I—II.
2 →
3 3 months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·01 gr. T.O.A. 102°.
9–11
12 Tried P.T.O. but reacted ; then treated with T.R. At first lost weight and was very pleased ; down to 10st. 4lb., but gained again and went steadily forward.
26.xi.06. ·01 gr. P.T.O. 101° ; then T.R.
4.xii.06. ·001 T.R. 102°.
10.xii.06. ·0015 *gr. T.R.* 98·4°.
13.xii.06. ·002 gr. T.R. 99°.
21.i.07. ·2 gr. T.R. on to 2·1 gr. T.R.
In February (ii.07), few reactions of 100° ; looked and felt well ; 11st. 5lbs. ; gained 7lbs.

77

v.06. 36. 11st. 12lbs.
P.A. Hospital.

1 Pain and swelling with much effusion in right ankle ; movements limited ∴ of pain ; pain also in mid-tarsal joints ; diagnosed as gonorrhœal rheumatism, and treated as such by Dr. M. Then Dr. M. asked me to test patient.
2 →
3 10 months.
4 Pale, flabby.
5 B.
6 70.
7 N.
8 No Tb. ·001 gr. Old T. 100·4° ; ·00125, 99·8° ; ·004, 103°.
9 Perineal sinus leading to prostatic abscess (tub.).
10–11
12 Treated with P.T.O.
22.vi.06. ·02 gr. P.T.O. 101°.
30.vi.06. ·03 gr. P.T.O. 101·8°.
7.vii.06. ·02 gr. P.T.O. 99·4° ; *wt. 12st. 9lbs.*
26.vii.06. ·065 gr. P.T.O. 103°.
4.viii.06. ·065 gr. P.T.O. 103°.
23.viii.06. ·1 *gr. P.T.O.* 98·4°.
30.viii.06. ·2 *gr. P.T.O.* 98·4°.
3.ix.06. ·3 *gr. P.T.O.* 103·4°.
10.ix.06. ·35 *gr. P.T.O.* 100°.
15.ix.06. ·4 *gr. P.T.O.* 99°.
24.ix.06. ·5 gr. P.T.O. 98·4°.
3.x.06. ·9 gr. P.T.O. 100° ; 13st. 10lbs. Feels well ; no pain ; swelling much diminished and sinus almost healed ; *gained 26lbs.*

78

xi.06. 30. 8st. 6lbs.
Brother and sister have ϕ.

1 R. II L. I—II ; numerous râles.
2 →
3 2 years ago, hæmorrhage, lost 2 quarts.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. Reacted.

9 Hæm. 2 years ago and 1 year and 5 months ago.
10
11
12 Treated with P.T.O.
29.xi.06. ·0075 gr. P.T.O. 101·8°.
7.xii.06. ·0075 *gr. P.T.O.* 98·4°.
10.xii.06. ·01 gr. P.T.O. 98·4°.
14.xii.06. ·02 gr. P.T.O. 98·4°.
2.i.07. ·1 gr. P.T.O. 102·6°.
10.i.07. ·1 *gr. P.T.O.* 98·4°.
20.ii.07. ·65 gr. P.T.O. 101°; wt. 9st. 3½lbs.; gained 9lbs.
28.ii.07. ·75 *gr. P.T.O.* 100°.
17.iii.07. 1·1 gr. P.T.O. 98·4°; then P.T. after 3 doses.
5.iv.07. ·05 gr. P.T. 103°.
12 iv 07. ·05 gr. P.T. 101°.
22.iv.07. ·05 P.T. 103·4°; advised pause.
10.v.07. Wt. 9st. 8½lbs.; gained 16lbs. and lost cough; and looks picture of health. Another course up to x.07; wt. 10st. 2lbs.; all râles have disappeared.
1.xi.07. P.T.O. up to ·9 gr.; then P.T. up to ·225 gr.; wt. 9*st.* 13*lbs.* never more than 8st. 7lbs. before illness; gained 19lbs.; at one time 22lbs.
1908. Well.

79

iii.06. 36. 8st. 10lbs.
Sister θφ.

1 Stage I.
2 →
3 Few weeks.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. Reacted.
9–11
12 Saw Dr. S. S., who said right lung was affected and told her to spend a year in the country and sleep on balcony. She did neither but had tuberculin, with result appended.
Treated with P.T.O. to 1 gr.; gained 17lbs.; then T.O.A. 1 gr.; wt. 10st. 1½lbs.; then P.T. to ·35 gr.; wt. 10st. 4lbs.; *gained* 22¾*lbs.*

80

vi.06. 54. 9st. 9lb.

1 Stage I.
2 ⌒
3 ?
4 A.
5 Y; indigestion; lives chiefly on vegetables.
6 N.
7 *f* 99°, 99·4°.
8 No Tb. ·001 gr. T.A. 99·2°; ·005, 99·2°; 0·1, 100°, 101°, 102°.
9 Hæmorrhage 7 years ago ℥ss.
10 Indigestion.
11 *b*
12 Treated with P.T.O. up to 1·1 gr.; T.O.A. up to 1 gr.; then P.T. up to ·15 gr. (4.xi.06).
19.xi.06. ·15 gr. Old T. 102·6°.
28.xi.06. ·15 gr. P.T. 101·2°; *although on* 4.xi.06 ·15 *gr. P.T. did not raise temperature at all*; wt. 9st. 10¾lbs.
Temperature gradually fell to normal, and now patient can lie on right side; could not do so before treatment.

81

vi.06. 30. 11st. 1¾lbs.
Wife in Stage III, since dead.

1 Stage I.
2
3 Two or three months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 98·2°; ·005, 99·2°; ·006, 103·3°. Herpes, headache, cough worse.
9–11
12 Treated to P.T.O. to 1 gr.; then P.T. to ·65 gr.; no reactions of any degree; wt. 11st. 8¾lbs.; gained 7lbs.; lost cough and looks well.
Advised testing but he thought it unnecessary.

Control Case.

L. N. 21. Tested in 1902 by me, in P.A. Hospital and reacted; Stage I, physical signs slight, would not have treatment; came to see me privately and told her own history exactly; was in Stage III four years later, hopeless.
1906.

1907

82

i.07. 30. 7st. 4lbs.
No family history.

1 Stage I—II (R).
2 →
3 Since Christmas 1906.
4 X; lost few pounds.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A.—; ·002, 100°; ·005, 102°.
9
10 Very severe pains after doses; inside from hip down; had this pain before doses; "pains in chest and back, and in all joints, fingers and thumbs" after dose.
11 *b*.
12 Treated with P.T.O. up to ·95 gr.; gained 3lbs.; then P.T. up to ·675 gr.; no reaction; gained 6¼lbs. In 1908, tested and did not react.
1908. Feels quite well, and able to do everything.

83

v.07. 21. 9st. 6½lbs.
Brother under treatment also.

1 Stage.
2 ⌒
3 Recent.
4 A.
5 B.
6 N.
7 *f* 100°—100·6°.
8 No Tb. ·001 gr. T.A. 99·5°; ·002, 99·4°; ·005, 100·5°, for 24 hours.
9–11
12 Treated with P.T.O. up to ·8 gr.; wt. 9st. 12½lbs.; then P.T. up to ·95 gr., 10st. 0¾lbs. Improved in every way; temperature gradually fell to normal.
1908. Well.

84

i.07. 17. 7st. 8lbs.
Uncle had hæm. of lung.

1 R. I—II; and enlarged glands in neck.
2 →
3 Few months.
4 A.
5 B; app. good.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 103°, lasting 24 hours.
9 Enlarged cervical glands.
10–11
12 Treated with P.T.O.
22.i.07. ·003 P.T.O. 103·2°; cough, swelling and pain in glands of neck, lasting two days; headache and vomiting.
31.i.07. ·002 gr. P.T.O. 102°.
7.i.07. ·0025 gr. P.T.O. 102°.
14.ii.07. ·004 *gr. P.T.O.* 100° up to ·75 gr.; then P.T.
27.iii.07. ·02 gr. P.T. 103·2°.
5.iv.07. ·015 gr. P.T. 99°.
10.iv.07. ·02 *gr. P.T.* 98·4° up to ·55 gr.
Quite well.

85

ii.07. 30. 7st. 11lbs.
Father *aet.* 56 θ acute pleurisy.

1 R. II L. I—II; many râles.
2 →
3 2 or 3 months ago.
4 X.
5 Y.
6 N.
7 N.
8 No Tb.
9–11
12 Treated with P.T.O. up to 1 gr.; 8st.; but lost 4lbs. after Alexander operation, &c., while under treatment; then P.T. up to ·3 gr.; wt. 8st. 6lbs. on to 95 gr.; was treated for some months at a distance; doses sent regularly and injected by sister. Wrote to say she felt quite well; never better in her life; no cough, no sputum, able to do anything now.
1908. Well.

86

iii.07. 26. 9st. 4lbs.

1 R. I—II.
2 ⌒
3 *Aet.* 5, Pleurisy.
4 A.

5 B.
6 N.
7 N.
8 No Tb. ·001—; 005—; ·01, 100·5; ·01, 102·6°.
9
10 Asthma.
11 *b*.
12 Treated with P.T.O. up to ·1 gr.; then P.T. to ·9 gr.; 9st. 8½lbs. tested 6 months after ·005, ·015, ·03, ·045 gr. T.A. Can now throw off colds, feels and looks well.
1908. Well.

87

iv.07. 28. 7st. 5lbs.

1 Stage I.
2 ⌒
3 Small hæmorrhage in xi.06.
4 X; lost weight.
5 B.
6 100—104; pulse excitable, also systolic murmur at apex.
7 N.
8 No Tb. ·001—; ·005—; ·01, 102·65°, for 24 hours, and stiff neck, never before.
12 Treated with P.T.O. up to 1 gr. ·005 P.T.O 101°; in a month "felt grand," "treat to feel so well"; 7st. 12½lbs.; then P.T. up to ·75 gr.; "aching in limbs," rather severe after doses.

88

v.07. 42. 13st. 7lbs.

1 Stage I—II.
2 →
3 Severe hæmorrhage 3 years ago; colour in sputum since.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·002 gr. T.A. 101·2°; lasting 24 hours.
9–11
12 Treated with P.T.O. to ·1 gr.; and then P.T. to ·9 gr.

89

v.07. 22. 10st. 8½lbs.
Brother and sister had *ϕ*.
1 R. I—II.
2 →
3
4
5
6
7
8 No Tb. ·001 gr. T.A. 101°; marked symptoms.
9–11
12 Treated with P.T.O. to ·75 gr.; then P.T. up to 1 gr.; 10st. 10lbs. Looks well, no cough now, before troublesome; says he feels quite well and strong.

90

vi.07. 18. 9st. 6½lbs.
Four brothers and one sister *θϕ*.
1 Stage I—II.
2 →
3 Cough for fortnight.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100°; very great swelling in arm, œdema, redness; also headache, loss of appetite, vomiting.
9–11
12 Treated with P.T.O. to 1 gr.; wt. 10st.
14.vii.07. ·003 gr. P.T.O. 101°.
21.vii.07. ·003 gr. P.T.O. 100°.
25 vii.07. ·006 *gr. P.T.O.* 100°.
30.vii.07. ·0075 gr. P.T.O. 99·4°.
3.viii.07. ·01 gr. P.T.O. 100·8°.
10.viii.07. 01 gr. P.T.O. 100·4°.
16.viii.07. ·01 gr. P.T.O. 99·4°.
22 viii.07. ·015 *gr. P.T.O.* 98·4°; no more reactions on to 1 gr.; then P.T. up to ·95 gr.; wt. 10st. Great improvement; gained 7½lbs.

91

vi.07. 17. 9st. 1½lbs.
Sister *θϕ* this year, and mother *θϕ* two years ago.
1 Stage I—II.
2 →
3 Cough for a month.

4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100·3°.
9–11
12 Treated with P.T.O.; very sensitive.
16.vi.07. ·0075 gr. P.T.O. 101°.
20.vi.07. ·0065 gr. P.T.O. 98·4°.
23.vi.07. ·0075 gr. P.T.O. 98·4°.
27.vi.07. ·0125 gr. P.T.O. 101°.
5.vii.07. ·0125 gr. P.T.O. 101°.
15.vii.07. ·01 gr. P.T O. 100°.
19.vii.07. ·01 gr. P.T.O. 98·4°.; no further reactions up to ·9 gr. P.T.O.; wt. 9st. 7¾lbs.; then P.T. up to ·175 gr.
Felt so well that he did not think he needed more treatment.

92

iv.07.
Mother in Stage II. Tb. G. 6.

1 Stage I.
2
3 Recent.
4 X. Anæmic and listless.
5 Y.
6 N.
7 N.
8 No Tb. ·00025 gr. T.A. 100°, 100°, 100·4°; arm swollen, red and tender.
9—11
12 Treated with P.T. up to ·2 gr. Has now healthy colour, full of energy, never tired; apparently in best of health.
Should be tested.

93

iv.07. 29. 10st. 12lbs.
No family history. Public school teacher.

1 Stage I.
2 ⌒
3 Cough for 2 years.
4 A.
5 B.
6 N.
7 *f.* 99·4°.
8. No Tb. ·002 gr. T.A. 102°, 102·4°; 100°, 99·4°, 101·1°, 101°; 98·4°, 99·4°, 98·4°, 99·4°; severe headache; cough worse; severe local reaction in arm.
9—11
12 Treated with P.T.O. up to 1 gr.; then P.T. up to ·725 gr.; 11st. 3lbs.
Tested in Dec. 1907, ·025 gr. T.A.—

94

iv.07. 24. 8st. 11lbs.
Sister has had tub. glands in neck, and brother reacted to tuberculin.

1 Stage I—II.
2 ⌒
3 Cough for 4 months.
4 Tall and thin; X.
5 Y.
6 N.
7 N.
8 No Tb.
9—11
12 Treated with P.T.O. up to 1 gr.; wt. 9st. 3½lbs., gain of 6lbs.; then P.T. up to ·6m. Had much worry and trouble, and lost wt. 8st. 5lbs.; but felt well and lost cough altogether.
Should be tested.

95

vii.07. 25. 9st.

1 Stage I.
2 ⌒
3 Cough for 4 months.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 99°; ·003, 98°; ·005, 99·8°, 101°, 103°; 101·6°, 101·2°; cough worse and more nasal discharge.
9
10 Nasal catarrh and obstruction; turbinates cauterised.
11 *b.*
12 Treated with P.T.O. to 1·1 gr.; then P.T. up to ·95 gr.; feels quite well.
Should be tested this year.

96

viii.07. 25. 9st. 10½lbs.
Brother of 91.

1 Stage I; *no physical signs.*
2
3 Cough for years.
4 A.
5 B.
6 N.
7 N.
8 No Tb. Finding no physical signs was inclined not to test him. However, patient, knowing that his sisters (2) had benefited by treatment, *asked* to be tested to make sure, and to my surprise reacted energetically, both locally and generally; severe reaction in arm; headache; fever.
9—11
12 Treated with P.T.O. up to 1 gr.; gained 5lbs.; 10st. 1¼lbs.; then P.T. up to ·95 gr.
In splendid health and no cough.

97

iv.07. 8st. 6lbs.
Sister of another patient.

1 Stage I.
2
3 Suffered from "asthma."
4 X. Loss of wt. and dyspnœa.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 99·8°; very severe reaction in arm; ·003 P.T.O. 100·4°.
10 Asthma.
11 *b.*
12 Treated with P.T.O. up to 1·1 gr.; wt. 9st. 1½lbs., gain of 9½lbs.; then P.T. up to ·8 gr.; wt. 9st. 4lbs. Appearance quite changed; was pale, face drawn, and dyspnœic, now rosy, round face and healthy appearance; gained 12lbs.
Should be tested during this year (1908).

98

viii.07. 22. 12st.
No family history.

1 Stage I.
2 →
3 In Oct. 1905, low fever, night sweats and evening temp.; lost weight; short of health.
4 X.
5 Y. App. capricious.
6 N.
7 N.
8 No Tb. ·0015 gr. T.A. 99°; 97·2°, 99·2°, 99·4°, 100·4°, 10?·4°; 99·8°, 99·4°, 99·8°, 99·5°, 99·8°; 98·8°, 98·8°.
9
10
11
12 Treated with P.T.O. up to ·75 gr.; then P.T. up to ·325 gr. Greatly improved; wt. 12st. 4½lbs.
Still under treatment.

99

v.07. 36. 7st. 1½lbs.

1 I—II, definite.
2 →
3 1 year.
4 X. 7st. 1lb.
5 B.
6 64
7 99°.
8 No Tb. Reacted. Aching pains in back and legs, and aching all over.
9
10—11
12 Treated with P.T.O. up to 1gr.; then P.T. up to ·65 gr.; gained 14lbs.; wt. 8st. 1lb.
Very well; no signs of active disease; no cough.

100

v.07. 23. 10st. 4lbs.
Brother θφ saw him in Stage III, sent him home.

1 Stage I—II, with gastric symptoms.
2 →
3 6 months.
4 X.
5 Y. No appetite, and pains in stomach after food.
6 N.
7 N.
8 No Tb. ·002 gr. T.A. 100°; ·0045, 101°; caused pains in stomach.
9 Gastralgia, &c.
10
11

12 Treated with P.T.O. up to 1 gr.; wt. 10st. 10lbs., gained 6lbs.; then P.T. up to 1 gr.; wt. 10st. 11½lbs. "Feels splendid," and regained old energy, and lost gastric symptoms. I sent doses to this patient living 400 miles from Sydney. Taught him how to give himself doses, no trouble.

Brother is *control* case. *Died in 2 years of pulmonary tuberculosis.* Sister treated by me quite well.

101

iv.07. 33. 10st. 2lbs.

1 Stage I—II.
2 →
3 Cough for 8 months; pleurisy 3 months ago; could feel "grating"; much pain.
4 X.
5 Y. Indigestion.
6 N.
7 N.
8 No Tb. Reacted.
9 Pleurisy.
10 Influenza? in ix.06; sleeps badly.
11 *b.*
12 Treated with P.T.O. up to 1 gr.; gained 5lbs.; then P.T. up to 1 gr.; 10st. 10½lbs., gained 8½lbs.; "feels splendid," eats and sleeps well; got married on strength of it.

Remained at work throughout treatment.

102

—— 27. 9st. 3lbs.

1 Stage I.
2 ⌒
3 4 months ago vomited basin of blood.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100°; *severe swelling in arm.*
9—11
12 Treated with P.T.O. up to 1 gr.; in 3 weeks gained 5lbs.; then P.T. up to ·8 gr.

Feels quite well and strong; no cough.

103

ix.07. 36. 11st.

No family history.

1 Stage I—II or II; marked signs, râles, &c.
2 →
3 Cough for 6 months; 13 years ago operated on for tuberculous disease of iliac crest, large scar left.
4 X.
5 Y.
6
7 *f.* 99·6° to 99·8°.
8 Tb. G. 2·3 sputum $\bar{3}$ gr. (½)
9 Hæmorrhages; several $\bar{3}$
10—11
12 Treated with P.T.O. ·01 gr. P.T.O. 101°; gained 5lbs. in a month; in two months temperature gradually fell to normal; then P.T. sensitive; several reactions 100° to 101° up to ·1 gr. (xii.07). Dr. Griffiths continued treatment for 2 months, and then gave a rest because reactions continued. No cough and no phlegm. Had a rest at Manly on sea-coast for first month, but afterwards went in daily to work in his warehouse. He could not afford to do otherwise.

Sent two other cases for treatment.

104

ix.07. 26. 6st. 10lbs.

1 brother *aet.* 28 θφ, ill 8 months. Father and father's brother θφ.
1 R. I—II. Diagnosed by other doctors.
2 →
3 No pulm. symptoms.
4 X.
5 Y.
6 110—120.
7 *f.* 99·6.
8 No Tb.
9—11
12 Treated with P.T.O. up to ·8 gr.; in six weeks, *wt.* 7*st.* 4*lbs.*; then P.T. up to ·18 gr. Dr. Griffiths continues treatment. Temperature gradually fell to normal and remained so; pulse also improved greatly.

104a

—— 18. 9st. 9lbs.

1 L.. I—II.
2
3
4
5
6
7
8
9—11
12 Treated with Old T. up to ·1 gr.; gained 3lbs. Feels better, looks better, and has hardly any cough.

105

xi.07. 25. 10st. 1lb.
Got a first-class life the day before he saw me and Dr. H.; said "nothing the matter."

1 R. I—II.
2 →
3 Cough for 6 months.
4 X.
5 B.
6 N.
7 N.
8 No Tb. Reacted to ·001—100·2°; but patient was *sceptical* .·. gave ·005; temp. 103·4°; no more scepticism; vomiting, headache, and herpes instead.
9—11
12 P.T.O. up to ·95 gr.; gained 6½lbs. in a month; and then P.T. up to ·025 gr.; wt. 8st. 8½lbs.
Treatment continued by Dr. Griffiths.

106

—— 19. 8st.
B. (hospital case).

1 Stage I—II.
2 →
3 ?
4 X.
5 Y. See 10.
6 104.
7 F.
8 No Tb. ·001 old T. 98·4; ·004 old T. 103·4°; yet gained 3lbs.
9
10 Acute lead poisoning.
11
12 Treated with P.T.O. up to ·225 gr.; wt. 8st. 8lbs.
Pt. feels very well.

107

vii.07. 21
E. (hospital case).

1 Sent in as case of *cerebral tumour*. Frontal headache, vomiting; not dull, no fits; sight good; pupil reactions N; no optic neuritis; muscular power good; sensation unimpaired; reflexes normal; temp. normal; pulse 72; no signs in lungs.
Tested ·00125 old T. 99·8°; local reaction in arm; ·00125 old T. 101°; gained 8lbs. in 2 months.
Improved greatly under tuberculin.

108, 109, 110 } Still under treatment by Dr. Griffiths, but though all are improving, they have not yet had treatment enough to do much good.

111
112, 113, 114 } Reacted, and 113 and 114 are to have treatment.

115—See notes.

116—Error in diagnosis.

Case 115 is one of the most puzzling cases I have ever seen. She came from Tumut. There two doctors diagnosed tuberculous disease of kneejoint; and patient then came to Sydney and saw a surgeon, who confirmed diagnosis. I then saw patient and was of the same opinion, and proceeded at once to treat her, thinking that the nature of the case was certain. However, after a few doses of P.T.O., which, though rapidly increased, produced no effect at all, I began to be sceptical as to the existence of tuberculosis. I therefore gave some doses of Old T. up to ·015 *gr.*, with no effect in arm or in joint or in system. I then applied Calmette's ophthalmic reaction, which was also absolutely negative. Patient is still having tuberculin, though I fear the possibility of myeloid sarcoma of the lower end of the femur. It may be a chronic inflammation of knee-joint, not tuberculous. There was no history of injury, and no pain at first.

In case 116 I made a mistake in the diagnosis, because I did not use enough

tuberculin, and perhaps also the patient did not know how to read the thermometer. At the first visit I diagnosed pulmonary tuberculosis in Stage I, and tuberculous ulcer on one of vocal cords. But in testing with tuberculin the patient said that temperature throughout was normal. I gave as third dose ·005 gr. Old T. There being no reaction, I made an appointment for patient to come, so that I might remove a portion of ulcerating mass in larynx for microscopical examination. Patient did not come again, but went to another specialist, who removed a portion of mass, and in this mass giant cells were found. The subsequent history of the case I do not know. I did not see the patient again, but I heard that she was having tuberculin treatment by Wright's method.

Eight are still under treatment.

Eighteen cases reacted to test dose, but had no or less than 2 months' treatment.

I have herein presented 115 cases in the first stage of pulmonary tuberculosis (including in Stage I. all cases of Stage I.–II.), so that this classification agrees with the system recently adopted at the Vienna Congress of 1907, and includes rather more advanced cases than those of Stage I. in the scheme of the Imperial Board of Health of Berlin. From 1902 to 1908 20 cases in the first stage which reacted to tuberculin have not been treated, and three cases had only two months' treatment. Two cases reacting to tuberculin in 1902 and 1903, respectively, would not have treatment. One is dead, dying a year later, the other was dying in 1906. These are "control" cases.

Of the 110 cases treated fully from 1902–1908, I know of the death of three cases: 1 of acute malaria, 1 of splenic anæmia, and 1 the day after laparotomy for symptoms suggesting intestinal obstruction or acute pancreatitis. This last case died in New Zealand. I have done my best to keep in touch with all my cases, and I have not yet heard of a single case dying of pulmonary tuberculosis. In short, I know of two deaths among 23 early cases that refused tuberculin treatment, and I know of no deaths among the 110 that have had treatment. I know also that 90 out of these 110 are well and strong, and I would lay a wager about the others, giving odds. *At the worst I can claim success for tuberculin treatment in* 80 *per cent. of cases treated from* 1902 *to* 1908.

Another case, No. 52 Stage II., went to sanatorium in first stage, and in less than three months after being discharged as "cured" and "one of their best cases," he came to me when the disease was in the late second stage. This patient was

in the first stage when he went to the sanatorium. The examining doctor told me so (see No. 52, Stage II.). I was able to help this poor fellow with tuberculin. Another case in the first stage refused to have tuberculin treatment and went to the sanatorium. There he developed acute pleurisy and nearly died. He had to leave the sanatorium, and then again came to me. He was nearly in the third stage, and I declined to attempt treatment with tuberculin. These are further "control" cases treated at the sanatorium which may be contrasted with my results.

STAGE II.

1901

1

ix.01. 34. 9st. 4lbs.

1 Stage II (R. II, L. I—II, C. pleurisy).
2 →
3 2 years.
4 X.
5 Y.
6 80—84.
7 *f* 99·4°—99·6°.
8 Tb. G. 3 ; sputum ℥j.
9 Pleurisy, extensive.
10–11 *b*.
12 Treated with T.R. up to 2 gr.; several reactions (100°), one 101°.
20.xii.01. 2 gr. T.R. 102° ; several times pains in stomach and over liver, with vomiting, six hours after doses ; cough much less, with phlegm ; "was free from cough "and phlegm for a fortnight."
ii.07. Wt. 9st. 2lbs.; great retraction of right side of chest ; then P.T.O. to ·1 gr. and P.T. up to ·175 gr.; had intercurrent attack of pleurisy and wt. fell to 8st. 4lbs.; again (1908) under treatment.
Has so far survived 7 years.

2

iii.02. 28. 8st.

1 R. I—II ; L. II ; severe pleurisy also over cardiac area.
2 →
3 2 years.
4 X.
5 Y.
6 110—120.
7 *f* and F. 100°—101°.
8 Tb. G. 5.
9 Pleurisy on left side.
10 Severe cardiac weakness and tachycardia.
11 *c*.
12 Treated with T.R. up to 1·7 gr.; when he left me I suspected that he had also tuberculous pericarditis.
Died 1907 ; *survived 6 years.*

1902

3

—— 28. 6st. 13lbs.
Only one left of six all $\theta\phi$.

1 R. II ; L. I—II.
2 →
3 2 years.
4 X.
5 Y (poor eater).
6 84—90.
7 *f* (99—100°).
8
9
10 Faintings and anæmia.
11 *b—c*.
12 Few doses of T.E.; then T.R.; lost cough and recovered strength and energy ; sensitive, therefore pause ; ·03 T.E. up to 3·4 gr. *in* 1907 ; five years after was still well ; gained 17lbs.; *8st. 2lbs.*
1907. Well.

4

viii.02 34. 8st. 4lb.
Father *θφ*.

1 R. I—III ; L. I—II.
2 —
3 8 years ; "gave up all hope."
4 X.
5 Y.
6 86—92.
7
8 Tb. G. 3.
9 Typical plastic bronchitis and hæmorrhages.
10 Severe hysterical sympts.; fainting and tremors after a dose.
11 *b*.
12 Treated with T.R. up to 2·1 *gr.*; 8st. 8lbs.
v.03. wt. 9st. 4lbs.; cough and other symptoms practically gone ; feels grand.
7.viii.03. Wt. 9st. 4lbs.; no cough ; one small hæm.; appetite very good.
In 1904 T.E. up to 4 gr.; reactions (101°, 102°)
1908. Feels quite well ; no symptoms ; 10st.

5

v.02. 30. 7st. 11lbs.

1 R. II ; also pl. L. I—II ; effusion at right base.
2 →
3 Six months at least.
4 X ; very weak.
5 Y.
6 96—100.
7 99°.
8 Tb. later.
9 Pleurisy.
10
11 *c*.
12 Treated from May to Sept., 1902, with T.R., T.O., and T.E.
2.vi.02. ·425 gr. T.R. 101·8°.
4 vii.02. ·07 T.O. 101°.
16.vii.02. ·07 T.O. + 2 T.R. 101·8°.
22.vii.02. ·07 T.O. + 3 T.R. 103·4°; 8st. 1¾lbs.
31.vii. 7.viii. and 15.viii. severe reactions (103·2°, 103·4°, 103°) with ·4 gr. T.R. + ·1 gr. T.O., ·2 gr. T.E. and ·275 gr. T.E.
24.viii. ·3 gr. T.E. 102·4°.
1.ix. ·4 gr. T.E. 102·4°.
8.ix.02. ·45 T.E. 101·8° ; pause for one month.
20.x.02. 8st. 3lbs.
20.x.02. ·1 gr. T.E. 103·4° ; ·075 gr. T.E. 100·4° up to ·5 gr. T.E. ; then T.R. up to 1·5 gr.
v.03. Wt. 8st. 4½lbs.
13.vi.03. 8st. 9¾lbs.
In 1904 11st.; *gained* 45*lbs.*
Returned *in February*, 1906 ; temp. 100·4°—102°, and mixed infection with Tb. P.T.O. up to 1 gr.; then P.T. to ·125 gr.; 7st. 6lbs.
24.vii.06. 8st. 3lbs.
20.viii.06. 8st. 9lbs.; under P.T.; then again sensitive.
7.x.06. ·1 gr. P.T. 103°.
19.x.06. ·125 gr. P.T. 103° ; pause till xi.06.
3.i.07. ·159 gr. P.T. 102° ; pause for 2 months.
13.iii.07. P.T.O. up to 1 gr. ; normal temperature ; then P.T. with reactions up to ·125 gr.; under ·225 gr.; *loss of wt.*

1903

6

iii.03. 46. 9st. 3lbs.
Sent by Dr. M.

1 R. I—II ; L. I—II ; pleurisy with effusion.
2 →
3
4 X.
5 Y.
6 N.
N.
No Tb. ·001 gr. T.A. 100·6.
11 Pleurisy.
12 Treated with T.R. up to 6 gr.; then T.E. up to 3 gr.; wt. 9st. 3¾lbs.
x.03. No cough, no phlegm, "breathing good now," "tight feeling gone," able now to walk 20 miles, have done it several times ; before could not walk at

all without distress, now working hard in office and feeling well.
1908. Quite well but should be tested.

7

'03. 52.
1 R. II ; L. I—II.
2 →
3 12 months.
4 A.
5 B.
6 N.
7 N.
8 Tb. G. 2—3.
9–11 *b*
12 Treated with T.E. for few weeks only ; sensitive ; reactions ; improved so much that he thought he did not need any more treatment.
Have not seen or heard of him since.

1904

8

ix.04. 26. 9st. 10lbs.
No family history.
1 R. II ; L. I.
2 →
3 1 year.
4 X.
5 Y.
6 Excitable (60 to 80) to 110°.
7 *f* and F. 101°, 100°, 102°, 103·4°.
8 Tb. G. 1·2.
9 Hæmorrhage every morning, ℥ij—℥ss.
10 Hoarseness.
11 *b*.
12 Treated with T.E. ; in 14 days hæmorrhage stopped but returned though much less.
3.xi.04. ·8 gr. T.E. 101·6°.
12.xi.04. ·9 gr. T.E. 101·6°.
18.xi.04. 1 gr. T.E. 101·8°.
1.xii.04. 1 gr. T.E. 101·8°.
9.xii.04. 1 gr. T.E. 100·4°.
15.xii.04. 1·4 gr. T.E. 100·4° ; pause for 3 weeks.
5.i.05. 1 gr. T.E. 101°.
12.i.05. 1·25 gr. T.E. 101·2.
19.i.05. 1·5 gr. T.E. 100·6°.
27.i.05. 1·8 gr. T.E. 100·4°.
6.ii.05. 2·25 gr. T.E. 101·6°.
20.ii.05. 2·5 gr. T.E. 101·2°.
11.iii.05. 2·7 gr. T.E. 101°.
25.iii.05. 3 gr. T.E. 100·6°.
8.iv.05. 3·5 gr. T.E. 101°.
3.v.05. 4 gr. T.E. 102° ; hæmorrhage ceased ; went up country, and was very well for about a year.
9.vii.06. Slight hæmorrhage after severe exertion ; then P.T.O. up to ·9 gr. and P.T. up to ·9 gr.
1908. Has been well ever since ; in this case, although hæmorrhages occurred, even large doses with reactions did not cause but prevented hæmorrhages.
Four years after. Well.

9

viii.04. 26. 9st. 10lbs.
Sister, mother, and brother, *θφ* (see No. 17, Stage I).
1 R. I ; L. II.
2 →
3 6 months or more.
4 X.
5 Y.
6 N.
7 N.
8 Tb. later ·001 gr. T.A. 100·2°.
9
10 *b*.
11 Treated with T.E. up to 3·5 gr. ; steadily gained wt.
x.04. Tested ·004, 100·2° ; treated further with P.T.O. ; and then P.T. up to ·35 gr. ; wt. 10st. 8lbs.
24.viii.06. P.T. ·8 gr. ; 10st. 7½lbs.
9 months later :
4.v.07. P.T.O. to ·2 gr. ; then P.T. up to ·5 gr. ; "feels ever so much better after the doses."

10

v.04. 18. 8st. 6lbs.
No family history.
1 R. I—II ; L. II ; extensive pleurisy at base.
2 →
3 2 years spinalgia.

4 X.
5 Y.
6 90.
7 *f* 100°—101°.
8 No Tb. Reacted.
9 Pleurisy.
10 First diagnosed as impacted renal calculus and sent to hosp. for operation.
11 *c.*
12 Treated with T.E. up to 1·2 gr.; gained 18lbs.; wt. 9st. 10¾lbs.; then on to 2 gr. T.E.
December, 1907. 3½ years after, quite well and got well; I advised testing, but patient was satisfied; married in 1907.

11

vii.04. 26. 7st. 8½lbs.
Nursed husband for 1½ years who died 3 months ago.

1 R. II; L ?
2 →
3 3 months.
4 X.
5 B.
6
7 *f* and F. 100°—101°, 102°—103°.
8 No Tb. Reacted.
9–11
12 Sent by her doctor to Bathurst; cough worse; sent away again but refused to go and came to me; treated with T.E.
21.vi.04. ·03 gr. T.E. 104°.
27.vi.04. ·03 gr. T.E. 99°.
1.vii.04. ·05 gr. T.E. 104°; then T.R. up to ·45 gr.; reactions 101°, 101°, 101°, and 104°); in September, although no dose was given, temp. 105°, with rigor, headache, vomiting, diarrhœa, abdominal pains, and menstruation, *no dose had been given for 9 days*; lost 5lbs. but soon regained in spite of reactions; wt. 8st. 6lbs.
iii.05. Wt. 9st. 1½lbs.; gained 21lbs.
20.iii.05. ·2 gr. T.E. 100°.
24.iii.05. ·3 gr. T.E. 100°.
29.iii.05. ·4 gr. T.E. 101°.
6.iv.05. ·4 gr. T.E. 99°.
10.iv.05. ·55 gr. T.E. 99°.
15.iv.05. ·7 gr. T.E. 100°.
19.iv 05. ·9 gr. T.E. 100°.
24.iv.05. 1·1 gr. T.E. 100°.
1.iv.05. 1·1 gr. T.E. 100°.
While in hospital acquired a mixed infection; signs of active trouble over upper R. lobe esp. in interscapular region; temp. 104° in evening, normal in morning, for 10 days; given an intravenous injection of collargol (1 c.cm. of 1% solution) and effect was dramatic; in 12 hours temp. fell to normal and did not rise again.
1908. Patient quite well.

12

ix.04. 24. 8st. 12lbs.
No family history.

1 R. II; L. I.
2 →
3 6 months; influenza ?
4 X; 8st 12lbs.
5 13.
6 84.
7 *f* (100°—101°).
8 No Tb.
9 Severe hæmorrhage, 1 *quart*; second 1½ pints.
10
11
12 Treated with T.E. up to ·3 gr.; gained 10lbs. in 20 days; then T.E. up to 1·1 gr.; many reactions (101°, 101·6°, 102°), yet gained 14½lbs.
6.iv.05. ·45 gr. T.E.
4.v.05. 1·5 gr. T.E. 101·6°.
29.v.05. 1·9 gr. T.E. 104°; pause.
15.xii.05. Tested: ·001—; ·005—; ·01, 100·2°; then treated with P.T. up to ·4 gr.
Sept., 1906, 10st. 1lb., gained 17lbs.
7.vi.07. P.T.O. up to 7·5 gr.; wt. 10st.
1908. Well. Has been working hard and travelling much since first course in 1904.

13

x.04. 14. 4st. 2½lbs.
Sent to hospital by doctor as typhoid fever.

1 R. I; L. II; with acute pleurisy.
2 →
3 2 months.

4 X ; 4st. 2½lbs.
5 Y.
6 100, 110, 120.
7 *f* and F. (100°—103°).
8 No Tb.; reacted.
9 Pleurisy.
10
11 *c*.
12 Treated with P.T. up to ·25 gr.
vii.05. 7st.
ix.05. 7st. 2½lbs. ; gained 42*lbs. in one year*, though growth at puberty accounts for part of this increase.
iii.06. Reacted slightly to Old T., though she feels and looks quite well ; in the hospital alone she gained 23lbs., from 4st. 2lbs. to 5st. 11lbs.

14

xi.04. 21. 7st. 11lbs.
Also sent to hospital as typhoid fever by doctor.

1 R. I—II ; L. II ; *c* pleurisy.
2 →
3 Ill 3, 4, or 5 months ; fever for weeks.
4 X.
5 Y.
6 90
7 *f* 100—100·2 and F.
8 Tb. G. 3 ; sputum ℥iij ·001 gr. old T. 101°.
9 Hæmorrhages ; pleurisy.
10 Rheumatic pains ; neurotic also.
11 *b*.
12 Treated for 4 or 5 months by doctor for rheumatism ; then sent to hospital as "typhoid fever" ; *great loss of energy* ; treated with T.E. up to ·45 gr. ; then reactions, 102°, next dose 104°, one reaction 105°.
23.x.05. No phlegm and no cough, feels well ; wt. 9st. 5lbs. ; gained 22lbs. ; then P.T. up to ·35 gr. ; seen afterwards and was well ; should be tested.

15

ix.04. 37. 8st.
Sister and husband θφ.

1 Stage II ?
2 →
3 Cough and hæm. a year.
4 X ; suckling child.
5 Y.
6 100—114.
7 N.
8 Tb. G. 2·3.
9 Hæmorrhage Oj.
10
11
12 Treated with T.E. to ·05 gr.
18.x.04. ·05 gr. T.E. to 102° ; still wt. 8st. 10lbs. ; then P.T.O. up to ·6 gr. ; wt. 8st. 11¼lbs. ; could not leave home to have more treatment ; not seen since ; improvement very marked in every way.

16

ix.06. 38. 10st.
No family history.

1 Stage I.
2 →
3 3 months.
4 X.
5 Y ; lost strength.
6 N.
7 N.
8 No Tb. ·002 gr. T.A. 101·1° ; awful headache, loss of appetite, pains in back, cough worse, sputum ℥ss.
9
10
11
12 Treated with T.E. ; sensitive.
8.x.04. ·025 gr. T.E. 102·8°.
13.x.04. ·025 *gr. T.E.* 98·4°.
16.x.04. ·05 gr. T.E. 101·6° ; gave up treatment because his friends told him that reactions were dangerous ; gained 1½lbs.

16a

xi.04. 39. 9st. 13½lbs.
Entered hospital, 9st. 3lbs.

1 Stage II.
2 →
3 11 weeks.
4 A.
5 B.
6 60.
7 N.
8 Tb. ; also ·001 gr. T.A. 99·2° ; ·003, 102° ; after test doses gained 7lbs. in 26 days.
9
10

11
12 Had few doses in hospital and later P.T.O. up to ·03; wt. now (1907), 11st. 10lbs.; gave up treatment; gained 21lbs. or from entrance to hospital 35lbs.

17

iii.04. 30. 8st. 6lbs.

1 Stage II.
2 →
3
4 X.
5 Y.
6 84.
7 N (99°).
8
9
10 Severe rheumatic pains in r. knee, elbow, and shoulder, after doses.
11
12 Treated with T.E. for one month, then went to country; gained 3lbs.; not seen since.

18

Control Cases.

30.

1 Stage II.
2 →
3 6 months.
4 A.
5 B.
6 N.
7 N.
8 Tb. 9·5.
9
10
11
12 Decided to have tuberculin and had a month's treatment with T.E.; gained 3lbs.; then persuaded by friends to have sanatorium treatment instead. Went to the sanatorium and was exhibited within a year as a case of arrested pulmonary tuberculosis. *Within 3 years he died of pulmonary tuberculosis.*

1905

19

ix.05. 50. 10st. 12lbs.
Mother, *aet.* 49, $\theta\phi$.

1 R. II. L. II.
2 →
3 Cough 2 years, came out from England for "weak chest" 25 years ago.
4 A.
5 B.
6 N.
7 N.
8 No Tb. ·001 gr. T.A.—; ·002—; ·005, 101°; sputum ℥j.
9
10
11
12 Sensitive to tuberculin. First 3 doses of P.T. 101°. *Still went on with P.T. and reactions ceased.*
26.i.06. ·375 gr. P.T.
Felt quite restored to health and did not think he needed more treatment; wt. 10st. 12¾lbs.
Has kept well.

20

ix.05. 48. 10st. 10lbs.

1 Stage II.
2 →
3 More than a year.
4 A.
5 Y.
6 N.
7 N.
8 Tb. G. 3.
9
10
11
12 Treated with P.T. up to ·9 gr. Lost a few pounds (10st. 6lbs.), but there was great improvement, and cough and phlegm ceased. "Felt so well."
Believe her to be well.

21

iv. 5. 21. 8st. 11lbs.
Father $\theta\phi$, 1 brother $\theta\phi$.

1 R. II. L. I—II.
2 →

3 1 year and 9 months.
4 X.
5 B.
6 72·84.
7 N.
8 Tb. G. 2·3.
9 Pleurisy.
10 Antral abscess.
11
12 Treated with P.T.O. to ·2 gr.; then T.O.A. to ·4 gr.; then P.T.O. to 1 gr.; then P.T.; very sensitive.
21.v.05. ·025 gr. P.T. 99°.
24. ·0375 gr. P.T. 104°.
4.vi. ·035 gr. P.T. 103·6°; then pause till iii.06; then P.T.O. and P.T. to ·175 gr.; wt. 8st. 12lbs.
14.07. T.R. up to 2 gr.
xi.07. Wt. 8st. 9lbs.
Feels better, looks better, and has lost cough.

22

xii.05. 33. 8st.
No family history.

1 R. II >. L. II.
2 →
3 10 years' history.
4 X. Deadly weariness.
5 Y. No appetite.
6 104.
7 *f.* 101°—102°; gen. 99·4, 100°.
8 Tb. 9·4.
9
10
11
12 Treated with P.T.O. to 1·2 gr.; many reactions at first and very sensitive; 4 months for course of P.T.O. to 1 gr.
1908. Extremely well.

23

ii.05. 31. 7st. 9lbs.

1 Stage II.
2 ⌒
3 2 years.
4 X.
5 Y. Ruined digestion with creasote, which he took for 2 years.
6 N.
7 N.
8 No Tb. ·001—; ·002—; ·005, 103°.
9
10 Albuminuria (Tub. ?).
11
12 Had open-air treatment and creasote *ad nauseam*. Doctor said "stick to creasote." Treated with T.E. up to 2·4 gr. No cough or phlegm. Used to get up two or three times in the night. Since treatment, has not to get up at all.
1908. Still well.

24

vi.05. 36. 7st. 8¼lbs.
Father *aet.* 49, *θφ*.

1 R. II >. L. II.
2 →
3 3 years.
4 X.
5 Y. Indigestion.
6 N.
7 N.
8 Tb. G. 2·3.
9
10
11
12 Treated with P.T. up to × ·1 gr.; gained 14lbs. in a month.
3.viii. ·1 gr. P.T. 102°; then treated with P.T. up to ·75 gr. The doctor who sent her to me can attest to the result in this case.
1908. Well.

25

xi.05. 22. 7st. 13lbs.
Mother suffers with chest.

1 R. II. L. I—II—II.
2 →
3 2 years. Night sweats for a year.
4 X.
5 B.
6 N.
7 *f.*
8 Tb. G. 2·3. ℥iij.
9
10
11 *b.*
12 Treated with P.T. to ·125 gr.; gained 5lbs.; wt. 8st. 4lbs. Went away for a month.
26.ii.06. P.T.O. to 1 gr.; then P.T. up to ·1 gr. Great improvement, lost cough and phlegm. Went up country and lost sight of.

26

xii.05. 25. 8st. 6lbs.

1 Stage II. (R. II. L. I—II).
2 —>
3 Two years' cough. Three years ago treated by Dr. A. for pleurisy.
4 X.
5 Y.
6 100.
7 N.
8 Tb. G. 6. Clumps. ℥ss.
9—11
12 Treated with P.T. up to ·05 gr.; then pause for 3 months.
1.iii.06. P.T.O. to ·8 gr.; T.O.A. to 5 gr.; then 3.v.06. P.T. again up to ·2 gr., and, finally, T.E. up to ·35 gr.

Well, when seen in 1907.

27

xii.05. 28. 9st. 1lb.

Husband δφ.

1 Cystitis—probably Tub.
2 —>
3 Since birth of child 2 years ago.
4 A.
5 Y.
6 86·96.
7 *f.* (100°—101°).
8 No Tb. ·001 gr. T.A. 101·4°; prolonged reaction for 24 hours.
9
10 Neurotic.
11 *b.*
12 Very sensitive to tuberculin; reactions 101°—102°; then no reaction above 100·4°; then was persuaded to trust to general hygiene. Had only a few doses—should not be included.

History: known to be fairly well a year later.

28

—— 22. 8st. 4lbs.

No family history.

1 4 years ago enlarged cervical glands, removed suppuration and large scar-circles, wide over and above and below clavicle; discharged for 2 years.
2 —>
3
4 Wt. 6st. after abscess; now 8st. 4lbs.
5
6
7
8 No Tb. Reacted severely to ·006 gr. Old T.

29

viii.5. 15½. 8st. 10lbs.

1 Enlarged glands of neck; glands of r. side were removed extensively; now large and numerous glands on l. side, as large as small hen's egg.
2 >
3 Years.
4—7. Nil; very fat.
8 No Tb. ·001 gr. T.A 102·4°.
9—11
12 Treated with T.A. up to 1 gr.; at first with P.T., but very sensitive.
22.xiii.05. ·0005 gr. P.T. 101°.
29.ix. ·015 gr. P.T. 100·6°.
15.x. ·03 gr. P.T. 100·6°; then old T. less reaction.
4.xi. ·1 Old T. 99°.
2.i.06. ·2 Old T. 99. Pause, then,
vi.06. P.T.O. up to ·9 gr.; T.O.A. up to 9 gr.; then P.T. up to 1 gr. Had glands then removed by Dr. B.

Control Case.

—— 40. 11st. 3lbs.

1 Stage II.
2 —>
3 2 years.
4 A.
5 B. Dyspepsia.
6 N.
7 N.
8 Tb. G. 5.
9 Hæmorrhages.
10
11 *a.*
12 He felt well, and said he could not afford time to take treatment. Meeting him again in street, urged him to be treated. He did not yield to my entreaties, and died *within a year* of φ.

1906

30

iv.c6. 38. 12st. 2lbs.

1 Stage II. (R. II > L. II).
2 →
3 2 years.
4 A.
5 B.
6 N.
7 N.
8 Tb. G. 3. ℥j.
9 Small hæmorrhages.
10
11
12 Previous treatment failed. Treated with P.T.O. to 1·1 gr.; wt. 12st. 7½lbs.; then T.O.A. to ·4 gr.; then P.T. up to 1 gr.
x.07. Doubtful if arrested; slight reaction. T.R. up to 2·1 gr. from November 13 to December 18 without any reaction; wt. 12st. 9¾lbs.; no cough or phlegm or râles.
Feels and looks strong and well.

31

iii.06. 44. 7st.
Eldest brother θφ; was much with him.

1 R. II. L. II.
2 →
3 18 months.
4 X.
5 B.
6 N.
7 N.
8 Tb. G. 3. ℥j.
9 Pleurisy.
10—11
12 Treated with P.T.O. to ·65 gr.; then T.O.A. to ·35 gr., and two doses of P.T. Improved; gained weight; but had trouble with her husband, and could not continue.

32

v.06. 32. 10st. 4lbs.
No family history.

1 R. II. L. II>.
2 →
3 Since Feb. 1904. Dr. Percy Kidd treated for throat in Sept. 1905.
4 A.
5 B.
6 N.
7 N.
8 Tb. G. 3·4. ℥j.
9 Hoarseness and hæmorrhage.
10
11 *b*.
12 Treated for throat in 1905. Treated with P.T.O. up to ·95 gr.; then T.O.A. to ·6 gr.; then P.T. to ·625 gr.: 10st. 4½lbs. (normal wt. 9st. 9lbs.).
vi.07. T.E. up to 2·5 gr.
Two reactions with each dose of 2·5 gr.; 10st. 4½lbs.
Feels well and looks well. July 1908.

33

iv.06. 9st. 2lbs.
Fruit dealer.

1 R. II. L. I.
2 →
3 For years.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001—; ·005—; ·01, 102°.
9
10 Rheumatic arthritis (lame).
11
12 Treated with P.T.O. to 4 gr.; then P.T. up to ·25 gr.
Much better; no cough. "Hardly feels anything in lungs now."

34

ix.06. 29. 9st.

1 R. II > L. I—II.
2 →
3 Pleurisy in 1903; severe hæmorrhage five weeks ago.
4 X.
5 Y.
6 90—96—120.
7 N.
8 No Tb. Severe reaction.
9 Hæmorrhage, Oj.
10 Typhoid fever and appendicitis.
11 *c*.

12 Sent by Dr. M. to Lithgow in 1903 or 1904.
Treated with P.T.O. up to 1 gr.; then T.O.A. up to ·75 gr.; gained 14lbs. in two months; then P.T.; very sensitive.
5.xii.06. ·03 gr. P.T. 104·6°, *a week after* 1 *gr. P.T.O. had no effect.* Yet he says he "can walk more easily and breathe more freely since reaction." ·03 gr. P.T. repeated 3 times, always 102°. Pause for 5 months; then P.T.O. to 1 gr. and P.T. without any reaction or trouble up to ·4 gr.
1908. Was very well.

35

x.06. 50. 9st. 10lbs.

1 R II. L. II or II—III.
2 →
3 Cough, 4½ years.
4 A.
5 Y.
6 80.
7 *f* 99·2°—100°—100·2°.
8 Tb. G. 3. Sputum ℥j.
9 Hæmorrhages.
10
11
12 Treated with P.T.O. up to ·9 gr.; then ·015 P.T 101°; ·02 gr. P.T. 102°.
iii.07. Much less phlegm and no blood. Now no râles over R. lung. L. lung also drier; wt. 9st. 13½lbs.
Not seen since.

36

viii.06. 25. 7st. 12lbs.
No family history. (N. 8st. 12lbs.)

1 R. II (râles). L. II (râles and friction).
2 →
3 8 months. Influenza 9 months ago.
4 X.
5 Y. Bad.
6 90—100.
7 *f* 100°.
8 Tb. G. 5—6-
9 Hæmorrhages ℥i.
10 Insomnia.
11 *c.*
12 Treated with P.T.O. up to ·05 gr.; gained lbs. Then on to 1 gr. P.T.O. and ·75 gr. T.O.A.; then P.T. up to ·6 gr.
During my absence in Tasmania, patient had a peculiar attack: severe headache and nausea without vomiting. No stiffness at back of neck; had funny sensations; lost her senses as soon as she lay head on pillow, and on opening eyes was giddy and staggered. For two days could not walk; would topple over; could not lift head from pillow. Patient is deaf, and there is loss of bone conduction. There is sometimes pain in the ears (Meniere's disease).
1908. Patient is greatly improved; *no cough or phlegm* or *râles.*

37

ix.06. 31. 8st. 8lbs.
No family history (N. 10st.)

Worked in Telegraph office with a man who died of ϕ, and used to expectorate all over the place.

1 R. II. L. I—II.
2 →
3 Hæmorrhage 5 years ago; second, 10 weeks ago; third, 14 days ago.
4 X.
5 Y.
6 N.
7 N.
8 Tb. G. 3. Sputum ℥ss.
9 Hæmorrhages ℥vi.—viii.
10 Pleurisy.
11 *b*
12 Treated with P.T.O. up to ·85 gr.; then T.O.A. up to ·9 gr.; then P.T. up to 4 gr. Reactions:—
14.ix.06. ·01 P.T.O. 102°.
18.ix.06. ·01 *gr. P.T.O.* 98·4°.
23.ix.06. ·015 gr. P.T.O. 98·4°.
16.xi.06. ·025 *gr. P.T.* 104°; within a week of ·85 gr. P.T.O. and ·9 gr. T.O.A.
1908. Heard of him as well and back at work, having gained many pounds in weight and recovered old energy.

38

v.06. 28. 8st. 5lbs.
Brother and sister reacted to T.

1 Dr. MacC. removed tub. glands of neck in August, 1905, now other glands enlarging on same side under sterno-mastoid and over scapula; also lungs R. I—II; cough, phlegm, short of breath.
2 —→
3 See above.
4
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 101°.
9 Pulm. T. and T. glands.
10
11 *b.*
12 Treated by Drs. M. L. D., who found nothing in lung. Treated with P.T.O. up to 1 gr.; then T.O.A. up to ·5 gr.; then P.T. up to ·45 gr.; moderate reactions. Went to Tasmania, did far too much in spite of advice, walked up Mt. Wellington; glands began to enlarge again.
iii.07. T.R. up to ·85 gr.; wt. 8st. 6½lbs.
In v.07 enlarged gland, softened, and was removed by Dr. MacC.
1908. Seems well, but has had much worry.

39

i.06. 25. 12st. 8lbs.
No family history.

1 R. II.
2 —→
3 Pleurisy 10 months ago; temp. 103°; rigor; 9 weeks in bed; cough ever since.
4 A.
5 B.
6 N.
7 N.
8 Tb. G. 3. Sputum ℥iij.
9 Hæmorrhages.
10—11
12 Before had 6 months open air by Dr. S. I., yet now active second stage; treated with P.T.O. up to 5 gr. Greatly improved, cough much less and much less sputum; looks to be in splendid health.
Said he could not afford time for more treatment; more's the pity.

40

ii.06. 29. 10st. 8lbs.
No history in past, but sister and brother reacted to T., and were treated.

1 R. II; L. I.
2 —→
3 6 months.
4 X.
5 Y.
6 84.
7. *f.* 99·4°.
8 Tb. G. 4.
9—10
11 *b.*
12 Treated with P.T.O. and P.T. up to ·475 gr.
25.viii.06. ·475 *gr. P.T.* 101·2°.
3.ix.06. ·475 *gr. P.T.* 99·2°.
10.ix.06. ·55 *gr. P.T.* 98·8°.
17.ix.06. ·65 *gr. P.T.* 99°.
22.x.06. ·9 gr. P.T. 100·4°.
Tested in iii.07. ·03 gr. Old T. 99·8°; advised more treatment.
iv.07. Treated with T.R. up to 2 gr.
30.vi.07. 1·5 gr. T.R. 100°, and ten days after this dose, opsonic index, 1·5.
After treatment, opsonic index, 1·78.

41

iii.06. 24. 9st. 2lbs.

1 R. II.
2 —→
3 Hæm. Oj. Christmas, 1904.
4 A.
5 B.
6 N.
7 N.
8 Tb. G. 2.
9 Hæmorrhage.
10
11 *b.*
12 Previous treatment: 3 years in New England, also at Yass.
Treated with P.T.O. to ·9 gr.; reactions, 100°—100·4°; then T.O.A. to ·7 gr.; then P.T. up to ·95 gr.; few reactions (100·8).
12.vii.07. Tested ·005 gr. T.A.—·015,—; ·035,102°. Further course, P.T.O. to 1 gr.; then P.T. to 1 gr., without reactions.
Very well; no cough or phlegm; at work throughout treatment.

42

ii.06. 26. 9st. 6lbs.
No family history.

1 Stage II.
2 →
3 2 years.
4 A.
5 B.
6 N. (78–80 or 72).
7 N.
8 Tb. G. 3.
9—11
12 Previous treatment for 9 months by creasote and inhalations, no better. Then P.T.O. to ·9 gr., T.O.A. to ·65 gr., and P.T. up to ·4 gr.; reactions, 1.
3.vii.06. ·25 gr. P.T. 100·4°.
9.vii.06. ·3 gr. P.T. 101°.
15.vii.06. ·3 gr. P.T. 99°.
21.vii.06. ·4 gr. P.T. 102°.
30.vii.06. ·4 gr. P.T.
iv.07. Tested ·03.
1908. Apparently well; wt. 10st. 6lbs., gained 14 lbs.

43

viii.06. 28. 8st.

1 R. II; L. II>
2
3 3 years ago.
4 A.
5 B.
6 N.
7 *f.* 99—99·4.
8 Tb. G. 3.
9– 11.
12 Treated by open air for two years, yet disease progressed; then P.T.O. up to 1 gr.; reactions slight (99·6—100·6°); then T.O.A. to 1 gr. (101·4°); then P.T.
xi.06. I sent two graduated doses for patient to inject; she gave stronger dose first; temp. 102°.
5.i.07. ·25 gr. P.T. 101·6°.
20.iii.07. ·7 gr. P.T. 99°; 7st. 13lbs. Lost a pound in weight, but greatly improved in health and physical signs.
Hardly any cough or phlegm.

44

vii.06. 31. 9st.

1 R. II.
2 →
3 Dating from childhood, 15 years old; numerous extensive chronic discharging sores which have left huge scars in skin, arms, neck, and nose. Hæmorrhage from lungs in 1900.
4 X.
5 B.
6
7 *f.* 100°, esp. after meals.
8 No Tb.
9 Hæmorrhage.
10 Liable to diarrhœa and dysentery.
11 Had to give up work 4 years ago.
12 Treated with P.T.O.; reacted to 99·6°—100° after each dose, but increased nevertheless; in a month, ·1 gr. P.T.O. 99°; then:
24.ix.06. ·45 gr. P.T.O. 100°.
28.ix.06. ·6 gr. P.T.O. 98·4°.
1.x.06. ·8 gr. P.T.O. 98·4°.
13.x.06. 1·1 gr. P.T.O. 98·4°; gained 20lbs.; wt. 10st. 4lbs.; then P.T.
20.x.06. ·02 gr. P.T. 101°.
26.x.06. ·02 gr. P.T. 100°.
30.x.06. ·03 gr. P.T. 99·2°.
8.xii.06. ·25 gr. P.T. 100°; then pause, went home to Armidale, got a very severe attack of dysentery, lasting some weeks, lost much weight; came to see me, sent him to Tasmania, where he gradually recovered health and weight.
v.07. Further course P.T.O. to ·9 gr. and P.T. up to ·875; 10st. 10lbs.; never so heavy before; bowels now regular; before severe diarrhœa alternating with constipation (Tub. ?); now got better of constipation and no diarrhœa for 6 months.
Looks picture of health.

45

i.06. 28. 8st. 11lbs.
Sister $\theta\phi$ in 1906.

1 R. II; L. I—II.
2 →
3 Pleurisy, Jan. 1905.
4 A.
5 Y.
6 N.
7 N.
8 Tb. G. 2.
9—11
12 Treated with P.T.O. to 1 gr.;

then T.O.A. to ·85 gr.; then P.T. to ·1 gr.; 3 reactions (101·2°).
4.ix.06. ·325 gr. P.T. 100°.
10.ix. ·35 gr. P.T. 101°.
17.ix. ·4 gr. P.T. 100°.
2.x. ·45 gr. P.T. 101°.
11.x. ·45 gr. P.T. 101°.
Tested iii.07, ·005 gr. T.A.—·015—·03 gr. Old T.; ·05 gr. Old T. 100° for an hour.
1908. Well since.

46

vi.06. 27. 8st. 6lbs.

1 Stage II.
2 →
3 Cough for 8 years; losing a lb. a week till treatment with tuberculin, then put on 3lbs.
4 X.
5 Y.
6 84.
7 *f.* 100°, 101·2°, 101·6°.
8 No Tb.
9—10
11 *b.*
12 Treated with P.T.O. Sensitive.
12.vi. ·002 gr. P.T.O. 100·8°; ·002 gr. P.T.O. 100·2°; ·0025 gr. P.T.O. 99·8°.
12.vii. ·005 gr. P.T.O. 100·6°.
20.vii. ·005 *gr. P.T.O.* 103°.
3.viii. ·006 gr. P.T.O. 99·6°; wt. 8st. 9¼lbs. No further reactions with P.T.O. up to ·9 gr.; 8st. 11lbs.; then P.T. up to ·6 gr.
Lost all the rheumatic pains from which she had suffered so much before tuberculin treatment.

47

v.06. 35. 10st. 11lbs.

1 R. I—II; L. II.
2 →
3 Since Jan. 1905.
4 A.
5 B. Large eater.
6 N.
7 N.
8 Tb. G. 3·4. Phlegm ℥ss.
9 Hæmorrhages severe.
10—11
12 P.T.O. up to ·9 gr.; T.O.A. up to ·6 gr.; then P.T. up to ·8 gr.
10.xii.06. ·575 gr. P.T. 104°.
22.xii. ·5 gr. P.T. 100°.
2.i.07. ·55 gr. P.T. 98·8°.
11.i.07. ·6 gr. P.T. 99·2°.
22.i.07. ·7 gr. P.T. 100°.
1.ii.07. ·8 gr. P.T. 99°.
iii.07. Severe hæmorrhage occurred; gave T.R. up to 1·7 gr.; no reactions.
vi 07. No râles; used to be coarse and moist; gained 14lbs.; wt. 11st. 12lbs.
1908. Was very well, but fear recurrence of hæmorrhage.

48

xi.06. 33. 9st. 12lbs.
No family history.

1 R. II; L. I—II.
2 →
3 Cough 6 months; pleurisy, 2 years ago.
4 X.
5 Y.
6 N.
7 *f.* 99°—99·6°.
8 Tb. G. 2·3; ℥ss.
9 Hæmorrhage Oj. 4 months ago.
10
11 *b.*
12 P.T.O. up to ·7 gr.; then P.T.; very sensitive.
25.i.07. ·01 gr. P.T. 102·6°.
1.ii. ·0075 gr. P.T. 100°; then T.R.
5.iv.07. ·1 gr. T.R. 102°.
11.iv.07. ·1 gr. T.R. 99·6°.
6.v.07. ·45 gr. T.R. 102·6°.
13.v.07. ·45 gr. T.R. 102°; loss of wt.; wt. 9st. 7lbs.; over sensitiveness; extreme to all forms.
xii.07. 8st. 13lbs.
Ordered rest; pt. could not afford to rest; poor.

49

iii.06. 18. 7st. 7lbs.
Sister θφ in 1906; father had many hæm. ?

1 R. II; L. II.
2 →
3 1½ years
4 X.
5 Y.
6 N.

7 *f.* 100°—101°.
8 Tb. not found ; ℥ss.
9 Hoarseness, diarrhœa, and griping pains since Christmas ; later double ischiorectal abscess.
10
11 *c.*
12 P.T.O. to ·7 gr. ; T.O.A. to ·6 gr. ; then P.T. up to ·05 gr. ; several reactions 101°.
12.viii.06. ·05 gr. P.T. 102·6° ; pause for two months ; then P.T. to ·15 gr. ; reactions, 100°, 101°, 102·6° ; but temperature by slow steps fell to 99°.
vi.07. 8st. 13lbs. ; P.T.O. again to 1 gr., and P.T. up to ·7 gr. ; *gained 21lbs.* ; wt. 9st. ; another ischiorectal abscess.
xii.07. Very well indeed.

50

iii.06. 25. 10st. 12lbs.
Father θφ and brother θφ (galloping).

1 R. II.
2 →
3 2 years ago hæmorrhage and pleurisy.
4 X.
5 B.
6 N.
7 N.
8 Tb. G. 7·8 ; sputum ℥ss.
9 Hæm. and pleurisy.
10–11
12 Treated with P.T. up to ·95 gr. ; few reactions, 101°, 102°, 100°.
ix.06. 11st. 0½lb.
Seen late in 1907 ; was very well and full of energy ; no cough and no phlegm.

51

v.06. 30. 8st. 4lbs.
Father θφ.

1 Stage II.
2 →
3 Hæmoptysis *aet.* 15.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100·2° ; great swelling of arm.
9–11 *b.*
12 Previous treatment by Dr. H. creasote.
Tuberculin ; sensitive.
25.v.06. ·003 gr. P.T.O. 101·2°.
1.vi. ·003 gr. P.T.O. 101°.
10.vi. ·003 gr. P.T.O. 101·2°.
19.vi. ·002 gr. P.T.O. 98·4°.
21.vi. ·003 gr. P.T.O. 98·4°.
24.vi. ·006 *gr. P.T.O.* 98·4°.
28.vi. ·01 gr. P.T.O. 101°.
4.vii. ·01 gr. P.T.O. 98°.
8.vii. ·015 gr. P.T.O. 101°.
15.vii. ·015 gr. P.T.O. 99·6°.
20.vii. ·02 gr. P.T.O. 99·4° ; then rapid increase without reaction till
17.viii. ·2 gr. P.T.O. 100°.
1.ix. ·4 gr. P.T.O. 101°.
7.ix. ·45 gr. P.T.O. 99°.
7.x. 1 gr. P.T.O. 99° ; then T.O.A. up to 1 gr. (98·4°) ; then P.T. up to ·2 gr.
25.xi.06. ·2 gr. P.T. 101·8°.
3.xii. ·2 gr. P.T. 99·6°.
1908. Feels and looks quite well ; at work during whole time.

52

v.06. 22. 9st. 6lbs.
No family history.

1 R. II > active L. I—II.
2 →
3 Cough for 10 months.
4 X.
5 Y.
6 N.
7 N.
8 Tb. G. 5.
9 Cords red and interarytenoid area rough and velvety ; hoarse.
10–11 C.
12 Previous treatment. This is a splendid *control case.* In July, 1905, Dr. G. sent to sanatorium at Wentworth Falls. Dr. G. in note says, "No physical signs and no Tb." He had been ill 5 weeks and lost 3 stone. Went to sanatorium, gained 28lbs., was discharged "cured" ; "one of the "best cases they had ever turned "out" ; advised to go to sea ; went to sea and in 5 weeks lost 22lbs.
Came to me and was treated with tuberculin, *living in Sydney all the time.* On coming to me he was in an advanced second stage, Tb. G. 5 and wasted, pale and

weak. I gave him P.T.O. up to ·45 gr., gained 3lbs.; then T.O.A. up to ·2 gr.; then P.T. up to ·475 gr.; wt. 9st. 10¼lbs.
He had to take work again on a ship and at intervals he came to see me. There was an immense improvement; he had hardly any cough; his appetite was very good. There was no relapse.
One has but to look on this picture and on that, and then explain the facts in the light of logic and reason.
Unfortunately he had lapsed into a late second stage before I treated him, and one cannot be sure of results in such cases.

53

vi.06. 36. 10st.
Brother *aet.* 55 θφ 12 years ago, and lost two children of φ, *aet.* 14 mos. and 5 years.

1 R. II>; L. II.
2 →
3 Hæmorrhage 8 years ago; in 1898 and in 1903 (Oj.)
4 X.
5 B.
6 N.
7 *f.*
8 Tb. G. 4. >℥j. sputum.
9 Hæm. and pleurisy.
10–11
12 (Had open air in New Zealand); P.T.O. up to 1·1 gr., gained 10lbs.; 10st. 10lbs.; then T.O.A. up to ·75 gr.; then P.T. up to ·75 gr. During treatment had very acute attack of pleurisy with high fever (105°) in July (viii.), 1906, passed off in 10 days; afterwards no fever except with reactions.
Went back to N.Z. apparently well.

1907

54

viii.07. 24. 7st. 1lb.
One brother θφ laryngis.

1 Stage II.
2 →
3 1 year; pleurisy 8 years ago.
4 X.
5 Y; fair.
6 80—90.
7 *f.*
8 Tb. G. 4.
9 Hoarseness.
10 Asthma as a child, always a cough.
11 *c.*
12 Very sensitive to tuberculin; first P.T.O. ·003, 101·6° three times, *yet gained 6lbs.*; cough better, less phlegm; increased to ·025 gr. P.T.O.; then T.O.A. up to ·35 gr.; wt. 7st. 8lbs.; reaction 102° went on to ·65 gr. T.O.A. *Eyes sore after doses.* Marked improvement, can get right tone in voice now, better in every way.
Still under treatment.

55

vi.07. 24. 9st. 4lbs.
No family history.

1 R. II; L. II>; extensive pleurisy.
2 →
3 Cough, six months.
4 Lost six pounds.
5 B.
6 N.
7 N.
8 Tb. G. 5–6 ℥ss>.
9 Fistula in ano with huge excavation.
10
11
12 Treated with P.T.O. to 1 gr.; 9st. 8lbs.; then P.T. up to ·575 gr.; now very little cough, except after dose; phlegm less every day; greatly improved.
Still under treatment.

56

iii.07. 58. 10st. 6lbs.
Hotel-keeper, no family history.

1 R. I—II; L. II>.
2 →
3 Pleurisy in Oct. 1904; hæmorrhage in Ap. 1907. ℥ij.
4 X.
5 B.
6 80.

7 N.
8 Tb. G. 4.
9 Extensive pleurisy.
10 Insomnia.
11 *b.*
12 Treated with P.T.O. to ·85 gr.; 11st. 2lbs.; then P.T. up to 1 gr.; 11st. 6¾lbs.; no serious reaction throughout. "No cough all the "week, though everyone else has "a cold;" can now sleep all night from 12 to 8; used to sleep three or four hours and then long for day. This man has been busy throughout with his hotel.
1908. Very well. *Gained* 13*lbs.*

57

vii.07. 32. 8st. 3lbs.
Father *θφ.*

1 R. II; L. I.
2 →
3 Pleurisy in 1899; hæmorrhage one month ago.
4 X.
5 Y.
6 N.
7 N.
8 Tb. G. 2.
9 Hæmorrhages.
10 Pains in joints and back after doses; night sweats.
11 *b—c.*
12 P.T.O. up to ·6 gr.; then P.T. up to ·175 gr.; several reactions: 103°, 101°, 100·6°, 100°, 8st. 4lbs.; much better, but will need careful treatment for some time; hardly any cough or phlegm.

58

ix.07. 22. 10st. 2lbs.

1 Severe tuberculous lesion of wrist with soft raised red fluctuating mass, no doubt containing caseous material, tender and painful on movement.
2 →
3 Many months.
4 X; fat and flabby, lost weight and looked better.
5 Y.
6 N.
7 N.
8
9
10
11
12 P.T.O. up to ·95 gr.; then P.T.
xi.07. ·1 gr. P.T. caused two reactions; sent home to Tasmania to rest; great improvement; the fluctuating mass has been absorbed and the joint is much improved.
Went home with instructions for further treatment.

59

vi.07. 30. 10st. 7lbs.

1 R. I—II.
2
3 Cough 8 or 9 weeks; took creasote; "nearly killed me; vomited it."
4
5
6
7
8 No Tb.
9–11 Hæmorrhages.
12 Went to mountains, weight fell to 10st.; P.T.O. to ·3 gr.; she felt doses very much; then P.T. up to ·1 gr.; gave up treatment on account of pain.

60

v.07. 24. 11st. 8lbs.

1 R. II; L. I.
2 →
3 2 or 3 months.
4 A.
5 Y.
6 104.
7 *f* 99·6°.
8 Tb. G. 3.
9 Laryngeal catarrh?
10 Pleurisy; several hæmorrhages, ℥ij.
11 *b.*
12 Treated with P.T.O. to ·9 gr.; 11st. 12lbs.; then P.T. to ·175 gr.; great improvement, but sensitive; pause.

61

vii. 07. 25. 8st. 7lbs.

1 R. II; well marked.
2 →
3 Sent to me by physician as anæmic, possibly pernicious anæmia.

4 X ; bloodless.
5 Y.
6 99—108.
7 *f* 99·8°—100°.
8 No Tb.
9 Marked pallor.
10
11 *c*.
12 P.T.O. to ·1 gr., in 6 weeks gained 13lbs.; in 2 months, pulse normal and pallor gone ; P.T.O. up to ·9 gr.; then P.T. up to ·75 gr.; 9st. 3lbs.; lost all symptoms, and looks splendid ; feels as well as ever in life.

62

i.07. 43·

This case has a special interest, at any rate from my point of view. The patient had been treated for Hodgkins disease, and possibly he had this disease, but under the peculiar circumstances I had no opportunity of examining his blood. When I examined him, there were enlarged glands in the neck on the right side and also under the clavicle and in the axilla. But I found also the apex beat of the heart displaced to the left. At once examining the right pleura I found fluid, and made a rough sketch of dull area in my note-book. Although fluid in the pleura may occur in lymphadenoma, I was disposed to make the diagnosis of tuberculosis provisionally. At any rate I proposed to give a test dose of tuberculin, to which he readily acquiesced. There and then I gave him ·001 gr. of Old T.; he recorded the following temperatures: 100·8°, 101°, 102°, 100°, and next day 97°, 98·4°, 98·4°. He certainly had tuberculosis and probably tuberculous pleurisy. Before seeing me, he had consulted four other doctors (Dr. M., Dr. J., Dr. F. and Dr. R.). After seeing me, he was advised to try another doctor (Dr. F.), who denied existence of fluid, in spite of dulness, displacement of heart and friction. He then returned to Dr. M., who also stoutly denied that there was fluid. Thinking that the truth must be with the many, he gave me up, and I did not see him again till the day before I left Sydney, when it chanced that Dr. Griffiths was in my consulting room. He then narrated the sequel. Within two months of his first visit to me, Dr. M. proposed to tap the right pleura ; in that time the fluid had increased so as to be recognised by symptoms as well as signs. The patient awoke to the truth, and went to another doctor (Dr. H. M.), who drew off a large quantity of fluid ; he came back to me and told the whole story of his own accord. I have not had any news of him, but I imagine Dr. Griffiths has been treating him with tuberculin, as I wished to do six months before ; if he had remained with me, I have little doubt that there would have been no cause for tapping. However, I have not heard from Dr. Griffiths what the blood examination showed. There was, however, little room for disagreement about the diagnosis ; such mistakes can be avoided without using tuberculin.

63

viii.07. 33· 7st. 1lb.
No family history.

1 R. II.
2
3 Treated by Dr. L. for 1 year.
4 X.
5 Y.
6 N.
7 *f* 100° for days.
8 No Tb.
9
10
11 *c*.
12 Treated with P.T.O. up to 9 gr.: ·15 gr. P.T.O. 102°; then P.T. up to ·65 gr.; 7st. 3lbs. Gradual fall of temperature to normal in 3 weeks ; thereafter temperature absolutely normal.

Patient should be tested again this year.

64

—— 20. 8st. 13lbs.
No family history, an athlete.

1 R. II; L. I—II.
2 →
3 3 months; *pleurisy* a year ago; attended by Dr. C.
4 X.
5 B.
6 N.
7
8 Tb. G. 3.
9 Hæmorrhages. ℥ij.
10
11 *b.*
12 Treated with P.T.O. sensitive up to 1 gr.; then P.T. up to 2·75 gr.; improved steadily, gained weight, strength and energy.
1908. Still under Dr. Griffiths.

65

v.07. 40. 6st. 7lbs.

1 Copious chronic effusion into left pleura, Oiij, withdrawn by aspiration.
2 →
3 Insidious onset.
4 X; 6st. 7lbs.
5 Y.
6 88.
7 *f.*
8 No Tb; tested, ·004 gr. T.A. 100°.
9–11 *c.*
12 Treated with P.T.O. up to 1 gr.; wt. 8*st.* 3*lbs.*; then P.T. up to ·55 gr.; 8st. 6½lbs.; *gain of* 27*lbs.*; feels much better, breathes very well, appetite very good. pain still felt on left side on deep breathing.
I also tested daughter of patient, but ·01 Old T. ——.

66

iv.07. 53. 9st. 13lbs.
(Hospital).

1 Bronchitis.
2 →
3 Many years.
4 X.
5 Y.
6 80.
7 N.
8 No Tb. ·001—; ·005, 99·6°; ·005, 99·4°; ·01, 101·4°.
9
10 Trace of albumen.
11
12 Treated with P.T.O. up to ·25 gr.; *wt.* 10*st.* 12*lbs.*; feels very well, appetite very good, no signs; against my advice left hospital and went to the country.
xii.07. Returned to hospital with definite pleurisy and *f* (100°–101°); treatment continued by my assistant.
This is a control case and shows *small effect of small doses.*

67

iv.07. 18. 7st. 12lbs.
A case of morbus cæruleus.

1 R. I; L. II.; I lectured upon this case, making diagnosis (provisional) of patent foramen ovale and perhaps patent ductus Botalli; very marked thrill over pulmonary artery and diastolic murmur. At lecture I had sputum examined, because I felt sure there was well-marked pulmonary tuberculosis. Moreover, I was determined to show that tuberculin was not contraindicated in cases of heart disease. The boy had also had pulmonary hæmorrhage.
2 →
3 4 months.
4 X.
5 B.
6 80.
7 *f.*
8 Tb. G. 4.
9
10 Congenital heart disease; treated with P.T.O. up to ·03 gr. (100·4°); he had a hæmorrhage followed by temp. 103·4°, when no P.T.O. was given; in two months he gained 9lbs.; and in another two months, another 10lbs.; wt. 9st. 3lbs. Thus even in this bad form of heart disease, tuberculin did good.

68

iv.07. 41. 10st. 5lbs.
Mother, *aet.* 40 θφ, also sister.

1 R. II, I.
2 →

3 Was sent to Australia by Sir A. Clark twenty years ago; then broke down again, went to New Zealand; each year had an attack, got rid of cough and put on weight. This is the first case in Stage II in which disease has been long at a standstill, though he has had no tuberculin treatment.
4 A.
5 B.
6 N.
7 N.
8 No Tb.
9–11
12 Treated with P.T.O. up to ·9 gr.; then P.T. up to ·6 gr.

69

v.07. 22. 6st. 12lbs.
Hospital case.

1 Sent in as "gastritis or gastric ulcer." *R. I*, well-marked signs; also enlarged gland in neck in front of and under sternomastoid. L.
2 →
3 2 years.
4 X.
5 Y; anorexia, frequent and severe vomiting after food-like symptoms of nervous dyspepsia, constipation. Dr. W. had diagnosed *appendicitis* and had operated, but no better.
6 72.
7 *f* to N.
8 No Tb. ·001 Old T. 98·4°; ·005 gr. Old T. 100·2°; swelling in arm and *cervical gland*.
10 Lienteric diarrhœa; irregular menstruation; from 13 to 17, regular, from 17 to 18, amenorrhœa; since 18, every five months till tuberculin was given; menses in October 1907, and again in November —*first time for four years*.
1 *c*.
2 Treated with P.T.O. up to ·75 gr.; gained 26lbs.; 8st. 10lbs.; no vomiting, appetite much better; then P.T. up to ·175 gr.; remarkable improvement, looks now rosy and robust; mother very pleased.

70

24. 9st. 8½lbs.

1 R. I—II, L. II.
2 →
3 Cough 8 or 9 months; ending in vomiting.
4 X; lost 6lbs.
5 Y.
6 100; intermittent.
7 *f* 99·6°.
8 Tb. G. 8; sputum ℥j.
9 Hæmorrhages.
10
11
12 Not seen any doctor before; yet advanced disease; treated with P.T.O. up to ·9 gr.; lost 1½lbs.; probably would have lost more if he had not had tuberculin; says he feels much better, indeed quite well; cough much better and phlegm less.
Still under tuberculin treatment by Dr. Griffiths.

71

42. 10st. 2lbs.

1 Stage II.
2 →
3 Began as influenza Oct. 1907; pleurisy 11 years ago, recovered and passed first-class life for A.M.P. Society.
4 A.
5 B.
6 N.
7 N.
8 Tb. G. 3.
9–11
12 Had been sent to country; put on weight and recovered appetite; is now under tuberculin treatment; doing well.

72

—— 44.

1 R. II >.
2 →
3 Cough 8 months.
4 X.
5 Y.
6 80.
7 99°.
8 Sputum ℥j.
9
10 Chr. fœtid rhinitis.
11
Under treatment; has improved.

73

—— 44. 7st. 6lbs.

No family history.

1 R. II. Râles.
2 →
3 Cough for some time.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. ·001 gr. T.A. 100°; ·001?; ·005, 102·2°; cough worse.
9
10
11
12
Under treatment.

74

v.07. 27. 6st. 6¾lbs.

1 Stage I. ? Tuberculous peritonitis. Pain in abdomen, esp. right side; gastric pain; vomiting, flatulence, dirty tongue, constipation, abd. distension.
2 →
3 2 years.
4 X. 6st. 6¾lbs.
5 Y.
6 76.
7 *f.*
8 No Tb. ·0015 Old T. 100·8°; ·005, 99·8°; ·005, 100·4°; thereafter normal for 4 days, morning and evening.
9
10 Hysterical and unsatisfactory.
11
12 Treated with P.T.O. up to ·35 gr.; gain 8¾lbs.
Troublesome in hospital. Went up the country. No news since.

STAGES II—III.

1902

1

—— 28. 8st.

1 R. I—II ; L. II—III. Extensive pleurisy with signs over cardiac area.
2 →
3 2 years.
4 X.
5 Y. Poor app. ; sweats.
6 110–120.
7 *f.*
8 Tb. G. 5.
9 Pleurisy.
10 Cardiac weakness.
11 *c.*
12 Treated with T.R. up to 1·7 gr.
Greatly improved ; but very weak and persistent tachycardia. I did not expect him to live another year, but he lived till middle of 1907. His brother (also Stage II, late) is still alive and having further treatment. He has had no treatment since 1902—*eight years ago.*

2

v.02. 13. 7st. 11lbs.
Mother $\theta\phi$, only brother $\theta\phi$, only sister reacted (1906).

1 R. I—II ; L. II—III.
2 →
3 10 months.
4 X.
5 B.
6 116–130.
7 F. 100°–102°–103°.
8 Tb. G. 6. M. s.
9 Hæmorrhage.
10
11 *b.*
12 Treated at first with rest, then T.R. up to 2 gr. ; wt. 8st. 9½lbs. Great improvement, no cough or phlegm. Tb. disappeared from sputum.
iv.03. Went to Show, got a severe chill and relapse, and mixed infection (S.).
vi.03. Gave T.E. up to 1·5 gr. Many reactions, 102°, 103°, yet pulse 90 and wt. 9st. 0¾lb.
vii.04. Gave T.E. up to 2·3 gr. ; wt. 9st. 8lbs.
xii.05. Wt. 9st. 12lbs. No cough, no phlegm.
January 1908. No cough, no sputum ; fat and strong.
April 1905. Tested with Old T. up to ·04 gr. No reaction.
Again, in 1907, when question of his going to a public school had to be settled, I tested him again. Old T. up to ·03 gr., no result.
Dr. M., the family doctor, and an utter sceptic, wrote to me in 1903, and described the grave condition of his lungs—"litera scripta manet" in my pocket. In this case also we have the "controls" in the mother and brother.

1903

3

—— 34. 7st. 12lbs.

1 R. II—III; L. I—II.
2 →
3 Pleurisy, 8 years ago; consumption for 1¼ years.
4 A.
5 B.
6 80. Good.
7 N.
8 Tb. G. 5·6.
9 Pleurisy.
10
11 *b*.
12 Treated with T.R. up to 1·5 gr.?; no reactions, but severe with T.E.; 1 gr. T.R. had no effect; yet ·075 gr. T.E. raised temp. to 103°; pt. was much improved, but could not remain away from home any longer.

Had no news of her since—good or bad. Certainly should have had more treatment.

4

iv.03. 31. 7st. 10lbs.

1 R. II; L. II—III.
2 →
3 2 years.
4 X. 7st. 10lbs.
5 Y.
6 76–84.
7 N.
8 Tb. G. 5. >℥j sputum.
9 Hæmorrhage.
10
11 *b*.
12 Treated with T.R. up to 1 gr., then T.E. Reactions began with ·4 gr. (103°, 102°, 101°, 102°). Pause for 6 weeks.

iii.04. T.E. up to 1 gr.
13.iv.04. 2 gr. T.E. 102°.
22.iv.04. 2·4 gr. T.E. 102·2°.; very little cough or phlegm—*yesterday none.*
7.v.04. 2·6 gr. T.E. 103·4°; wt. 8st. 10½lbs.
20.v.04. 2·3 gr. T.E. 103°; wt. 8st. 11lbs.
31.viii.04. Wt. 9st. 1lb.; *gained* 19*lbs.*
1907. Was well. Worked in office during treatment and ever since.

5

vii.03. 30. 8st. 3lbs.

No family history.

1 R. II—III; L. I—II.
2 →
3 2 years.
4 X.
5 Y.
6 84-96.
7 N.
8 Tb. G. 5·6. Sputum ℥iv.
9 Infiltration of one vocal cord; lost voice.
10
11 *b* or *c*.
12 Treated with T.R. up to 2 gr.; *in spite of severe reactions* (103°, 102·6°, 103°, 103°, 103°) and *great sensitiveness*; *gained* 14lbs.; no cough or expectoration, appetite good, and now plenty of energy; able to take severe walk.

i.05. Reacted to tuberculin; gave P.T.O. up to 1 gr., and
iii.05. T.E. up to 3.15 gr. (vii.05).
viii.07. Reacted; then P.T. up to ·85 gr. (x.07); wt. 9st. 2½lbs. Looks well and feels well. Needless to say, before he came to me, he had been treated by several doctors, but none of them by open-air methods, &c., did any real good.

1904

6

vi.04. 22. 10st. 4½lbs.
No family history, potus.

1 R. II—III ; L. II.
2 →
3 3 years.
4 X.
5 Y ; potus.
6 N.
7 N.
8 Tb. G. 8 clumps M.S.; sputum ½ cupful.
9 Tuberculous laryngitis ; pleurisy.
10 Potus.
11 *b*.
12 Treated with T.R. up to ·6 gr.; then T.E. up to 1·75 gr.; pause advised because 1·75 gr. T.E. caused 102·2° ; during treatment recovered voice, which he had lost completely for two months before he came to me. At first he gained 6lbs. but then lost 8lbs. In a few months very little cough ("not once a day"), and sputum a dessert-spoon instead of half a cupful. Three months before he came to me, he saw Dr. S. J., who sent him up the country. Dr. S. J. said he was in an early stage, that there was "nothing to worry about." In two months he certainly gained 10lbs., but he lost his voice from tuberculous laryngitis, and the disease advanced from an early stage to a late second stage. I advised him to come to me again in three months, but instead he gave way to his old habit, and went downhill. He was alive late in 1907.

7

xii.04. 20. 7st. 3lbs.
No family history.

1 R. II, L. II—III, or III (O).
2 →
3 Said to be 5 months.
4 X.
5 Y ; poor eater.
6 100—120.
7 F. 103°—103°.
8 Tb. G. 8. M.P. sputum ℥ij.
9
10 Diarrhœa (? T.) for six weeks and since.
11
12 At first treated with three intravenous injections of collargol (1% 1 c.cm.); then P.T. up to ·15 gr.; in three months temperature normal. Improved enormously. Then sent her to mountains for a rest. Here she was informed *by nurse*—or at least by her mother—that I considered her case as hopeless : her brother came to see me and asked me if I had told the nurse so. I had told the nurse that she was in a late stage, so that she should adopt the proper precautions. I have not seen the patient or her parents since, professionally, though I passed the patient in the street late in 1907.

8

x.04. 26. 9st. 2lbs.

1 Stage II—III or III.
2 →
3
4 X.
5 Y.
6 90—100 ; even 120 without tuberculin.
7 *f* and F. (100°—102°).
8 Tuberculous laryngitis.
9
10
11 *c*.
12 Treated with T.E. for few doses ; then T.R. up to ·3 gr.
26.x.04. ·325 gr. T.R. 105·4° ; probably into veins.
4.x.04. ·2 T.R. 98·4°.
10.x.04. ·3 gr. T.R. 101·4°.
18.x.04. ·3 gr. T.R. 103·8° ; but later temperature rose to 104° for no reason ; then P.T.O. up to ·4 gr.; gave up treatment.
Was alive in 1906.

9

ix.04. 23. 9st.
Mother, *aet.* 37, *θφ*.

1 R. II; L. II—III.
2 →
3 3 years ago hæmorrhage.
4 A.
5 B; good appetite.
6 N.
7 N.
8 Tb. G. 2.
9
10
11 *a*.
12 Treated with T.E. up to 4 gr.; 8st. 13lbs.; lost wt.; very hot weather (105°) and bush fires everywhere; tested six months after; reacted ·0025 Old T. 100°; then gave P.T. up to ·8 gr.; wt. 9st. 1½lbs.
2.vii.06. Tested again, ·003, ·01, ·02, *all negative*; can play tennis now all the afternoon; no cough and no phlegm.
Came to see me late in 1907. Quite well.

10

—— 30. 7st. 13lbs.
No family history.

1 Stage II—III.
2 →
3 For a year; 6 months night sweats.
4 X; 7st. 13lbs.
5 Y.
6 N.
7 N.
8 Tb. G. 5—6; phlegm ℥iij (½ cupful).
9 Hoarse.
10
11 *c*.
12 Treated with P.T. up to ·65 gr.; some severe reactions, 103·6°, 102°, 102°, 103°, 103°, 102°; still went on gaining weight and improving till he weighed 9*st.* 3*lbs.*; gained 18lbs.; looked a different man after treatment in spite of reactions; and has been at work ever since.
Well at end of 1907.

11

i.04. 39. 7st. 11½lbs.
No family history.

1 Stage II. With tuberculous testis.
2 →
3 Two years.
4 X.
5 Y.
6 N.
7 N.
8
9 Very severe hæmorrhage; hoarseness.
10–11 *b*.
12 Had been sent up country by Dr. F.; had a severe hæmorrhage; testis still discharging; treatment with P.T. up to ·14 gr.; abscess of testis healed completely; then T.E. up to ·55 gr. (101·8°); sensitive but gained wt.; gained 6½lbs.; 8st. 3lbs.; then P.T. up to ·05; patient left off coming for no reason that I can understand. His brother wrote to testify to the great improvement in his brother.
Heard nothing since.

Control.

v.04. 30. 9st. 0½lb.
No family history.

1 R. II—III; L. II.
2 →
3 Cough 2 years.
4 X.
5 Y.
6 N.
7 N.
8 Tb. ℥j.
9 Many hæmorrhages.
10
11
12 Saw Drs. W. and L. in Hobart, sent to Sydney; saw Dr. S. J., sent to country; saw Dr. H. at Orange; then Armidale, Glen-innes, Tenterfield; also Dr. O. W., Dr. R., and Dr. P.; met one of my patients and came to me; I decided to try tuberculin. However, he went away and in a few weeks was advertising virtues of a quack remedy, "Eureka," which had "cured" him. *Within 9 months he was dead.*

1905

12

i.05. 32. 8st. 11lbs.

1 R. II ; L. II—III.
2 →
3 12 years ago pleurisy and pneumonia ; cough for five years ; hæm. in 1903.
4 X.
5 Y.
6 100—120.
7 N.
8 Tb. G. 2 ; much phlegm.
9 *Asthma.*
10 Appendicitis ; severe pneumonia.
11 *c.*
12 Treated with few doses of T.E. and T.R. (·075 gr.) ; then P.T. up to ·7 gr. (8st. 10lbs.) ; no asthma at all, greatly improved, appetite better, and sleeps better, looks far better.

13

x.05. 42. 8st. 11lbs.
Mother, *aet.* 33, θφ.

1 R. II ; L. II—III.
2 →
3 1¼ years.
4 X.
5 Y.
6 84—100 ; 112 *c.* reaction.
7 N.
8 Tb. G. 4.
9 Hæmorrhages.
10
11 *b—c.*
12 Before treatment went to Narrabri and gained 10lbs. ; yet now stage II—III ; treated with P.T.O. up to ·9 gr. ; then P.T. up to ·9 gr.
x.06. Reacted ; further treated with T.O.A. up to ·4 gr. ; then P.T.O. up to ·9 gr. ; then P.T. up to 1 gr.
vi.07. Tested, ·005, ·0125, ·025, ·045 all negative.
Dec. 1907. Wt. 10st. 6lbs. ; seems and feels quite well. Gained 23lbs.

14

iii.05. 34. 8st. 2lbs.

1 R. II—III ; L. I—II.
2 →
3 3 years.
4 X.
5 Y.
6 N.
7 N.
8 Tb. G. 3. Sputum ℥ij.
9 Hæmorrhages.
10
11 *b.*
12 Treated with P.T. up to ·375 gr. Many reactions, no cough for 2 nights, no sputum ; wt. 8st. 13lbs. ; pause on account of severe reactions.
1906. Moderate relapse ; treated with P.T.O. to 1 gr., T.O.A. to 7·5 gr., and P.T. to 7 gr. ; wt. 8st. 7½lbs. ; sputum now about a teaspoonful in 24 hours ; no hæmorrhages ; can walk up hill ; before treatment could hardly walk any distance ; appetite very good.
Heard of patient late in 1907 as being very well and having gained weight.

15

i.05. 26. 8st. 5lbs. (N. 9st.)

1 R. II—III ; L. II (moist crackling râles in both lungs).
2 →
3 Cough for 6 months.
4 X.
5 Y.
6 84—100—120 *c* reactions.
7 *f* 99·6° to 100·2°.
8 Tb. G. 3.
9 Pleurisy.
10
11 *b.*
12 Treated with P.T.O., very sensitive, even to small doses (102°, 102°, &c.) ; then P.T., also sensitive.
1.xi.05. ·002 gr. P.T. 102°.
6.xi.05. ·002 gr. P.T. 98·4° (Sileat Wright).
9.xi.05. ·004 gr. P.T. 100·2°.
14.xi.05. ·006 gr. P.T. 99.4°.
Again :—
5.xii.05. ·025 gr. P.T. 103°. *Wt. 9st. 4lbs.*
12.xii.05. ·025 gr. P.T. 103°.
Pause :—
19.i.06. ·015 gr. P.T. 98·4°.
·025 gr. P.T. 100·4°.

25.i.06. ·03 gr. P.T. 99°.
30.i.06. ·04 gr. P.T. 103°.
Began again with small doses of P.T.O., and went forward without reaction to 1 gr.; then P.T., with moderate reactions (100°—101°) up to ·5 gr. Enormous improvement in physical signs, no cough or phlegm, looks well, wt. 9st. 8lbs.

16

iv.05. 33. 7st. 9lbs.
First child died of tuberculous meningitis; second has had large tuberculous glands of neck removed. Patient's mother, *aet.* 32, *θφ*.
1 R. II; L. II—III.
2 →
3 1 year.
4 X.
5 Y. Bad appetite.
6 90—96.
7 *f* 99·4°—100°.
8 Tb. G. 3—4.
9
10
11 *b*.
12 Previous treatment by open air—got worse; treated with P.T. up to ·7 gr.
24.v.05. ·03 gr. P.T. 104·2°. Since severe reaction no cough at all, hardly any phlegm, feels splendid, and appetite good.
In June two reactions of 101°.
In July reactions 100·2°—101·8°.
In August reactions 99·2°—101°.
9.x.05. ·65 gr. P.T. 101·8°.
11.xi.05. ·7 gr. P.T.
24.iii.06. 8st. 2lb.
Tested v.06. ·005 Old T., ·01, ·02, and ·03, all negative.
xi.06. Went to Melbourne Cup; a terribly wet day; feet wet all day; got a chill and relapsed.
9.xii.06. P.T.O. up to ·8 gr.; then T.O.A. to 8 gr.; then P.T. up to ·675 gr.; seems quite well again; no cough or phlegm and feels quite well; *wt.* 8*st.* 12*lbs.* In spite of advanced stage of disease (II—III) in 1905, 2½ years later no symptoms, gain of 15lbs. in weight, and best of all. Tested in December 1907 with negative result.
Such a result demands the confidence and patience of the patient, but it is worth it.

17

xi.05. 35. 10st. 12lbs.
No family history. Pt. is 9th in family.
1 R. II—III; L. II.
2 →
3 3 years. Hoarse for 1½ years. Many doctors.
4 X.
5 Y.
6 80—86.
7 N.
8 Tb. G. 4. Sputum ℥iv.
9 Hoarseness.
10
11 *b* or *c*.
12 Previous treatment; took creasote for 6 months, did him no good. In Queensland for open air treatment for 6 months, no better for it; slowly lost ground; had to give up work of any sort.
Treated with P.T.O. up to 1·2 gr. Many reactions (102°, 102°, 103°); then T.O.A. up to 1 gr. Then P.T. up to ·4 gr.
x.07. Reacted. P.T. again up to ·9 gr., not the slightest trouble and no reaction; feels and looks well; returned to work a year ago and at work ever since.

18

iii.05. 24. 10st. 12lbs.
1 Stage II—III.
2 →
3 2 or 3 years.
4 X.
5 Y.
6 106—100.
7 *f* 100°—101°.
8 Tb. G. 4—5 M.
9
10 Dengue. Diarrhœa.
11 *b*.
12 Treated with P.T. up to ·125 gr.; wt. increased to 11st. 9lb., feels vigorous, no cough. I then advised a pause because pt. was very sensitive (102°, 104°, 103—4°). He should have returned in 3 months. He waited 8 months, and meanwhile by travelling in trains hundreds of miles every week he got a severe mixed infection. Temp. 101; pulse 110. He had four doses and did not come again.

This case is also a control case, and the want of success was his own fault. He felt so well that he was over-confident and lost his life. Died few months after.

19

ii.05. 50. 10st. 2½lbs.

1 R. II—III ; L. II.
2 →
3 3 years ago cough and expectoration, yet pt. says "6 weeks ago."
4 X.
5 Y.
6 N.
7 *f.*
8 Tb. G. 5—6. ℥iij (half cupful).
9 Tub. laryngitis ; ulceration, hoarseness ; ulcer healed ; then, later, ulcer between arytenoids, which cicatrized, causing fixation of cord and aphonia.
10
11
12 Treated with T.E. up to 3·5 gr., with hardly a reaction ; *very rapid course.*
26.vi.05. 3 gr. T.E. 101°.
15.vii.05. 3·5 gr. T.E. 101°.
19.viii.05. 11*st.* 3*lbs.*, gained 15lb.
xi.05. Tested. ·02 old T. ——
In ii.06 discharging Tb. Treated with P.T.O. up to 1 gr. and P.T. up to ·175 gr.
Then ii.07 P.T. up to ·015 gr. 104° ; then T.O.A. up to 1 gr. and P.T. again to ·04 (100·6°) ; lost wt. ; 10st. 11½lbs.

20

x.05. 22. 10st. 2lbs.

1 R. II—III ; L. II.
2 →
3 Pleurisy 3 years ago.
4 X.
5 Y.
6 74–84 ; reaction, 101°, 90 ; 102°, 100.
7 N.
8 Tb. G. 3 ; ℥ij. sputum.
9 Pleurisy.
10
11 *b.*
12 Treated with P.T. up to ·75 gr. ; some severe reactions (103°, 101·8°, 102°), but most moderate ; gained 15lbs. ; wt. 11st. 3½lbs. ; even 11st. 7lbs. Went up country to station, and looked very ill on return. Treated again, P.T.O. up to ·85 gr., and T.O.A. up to 1 gr. ; then treated with P.T. up to ·45 gr. (103·4°) ; pause for six months.
1908. Very much better ; June 1908 heard from his brother, a doctor, that he had gained more weight and was very well.

21

—— 24. 8st. 7lbs.

1 Stage II—III ; also tub. disease of ear.
2 →
3 3 years.
4 A.
5 B.
6 80.
7 *f.* (100°).
8 Tb.
9 Tuberculosis of ankle and mastoid.
10
11 *b.*
12 Treated for 3 weeks ; 2 severe reactions ; gained 2lbs. ; but gave up treatment because she was a monthly nurse and could not afford to give up this work. There should be a law to prevent this kind of thing—a nurse, in advanced consumption, attending mother and infant. I said so plainly, and so she went away.
I do not know the sequel, but can guess it.

22

iii.05. 44. 7st. 13lbs.

1 Stage II—III.
2 →
3 1 year.
4 X.
5 Y.
6 N.
7 N.
8 Tb. G. 5. Sputum ℥iv.
9
10 Diarrhœa ? tub.
11 *b.*
12 Treated for a month with P.T. up to ·125 gr.
Cough better, has not coughed for 3 nights, much less phlegm ; gained 2lbs. ; wt. 8st. 1lb.
Left off treatment.

1906

23

i.06. 36. 8st.
Father, *aet.* 65, *θφ.*

1 R. I—II ; L. II—III.
2 →
3 Cough for 5 months.
4 X.
5 Y.
6 N.
7 N.
8 Tb. G. 9–10. M. streptococci thousands of colonies on plates. Sputum ℥j.
9 Hæmorrhages.
10
11
12 Treated with P.T.O. up to ·9 gr. ; then T.O.A. up to ·5 gr. ; then P.T. up to ·1 (103°) ; 8st. 2½lbs. ; pause for 2 months.
On return (3.ix.06) ·005 P.T. 103° ; began again with small doses of P.T.O. Pt. left off coming and died within 6 months. She had not had enough tuberculin to do much good, and she could only die without it.

24

—— 20.

Tb. G. 3–5. Sputum ℥ij.
Stage II—III.
Had 4 doses of P.T.O. and then left off coming.

25

—— 20. 7st. 6lbs.
No family history.

1 R. II ; L. III ; cavity at base (bronchiectasis).
2 →
3 2 years.
4 A. Not lost weight.
5 Y.
6 N.
7 *f.* 99°–100°.
8 No Tb. Reacted. Sputum ℥vj.
9 Hæmorrhage and pleurisy.
10 Clubbed fingers (bronchiectasis).
11 *b.*
12 Adenoids and naval spine removed. Dr. T. Y. sent to Dublo for 3 months.

Tuberculin.—Sensitive at first (102°, 103°, 101°, 101°) ; P.T.O. up to 1 gr. and P.T. up to ·1 gr. ; gained 12lbs. ; wt. 8st. 4lbs. Cough much better ; phlegm ℥ss. ; and feels and looks much better. Left off coming, I know not why. Do not know what happened after.

26

06. 62. 8st. 12lbs.
No family history.

1 R. II—III or III ; L. II.
2 →
3 Cough for 12 months.
4 X. Lost 2 stone (28lbs.)
5 Y.
6 80. Very *hard pulse.*
7 *f.*
8 Tb. G. 5–6. Sputum ℥jv.
9 Hæmorrhages ℥j + >
10 Albuminuria and chronic interstit nephritis.
11 *b.*
12 P.T.O. up to ·45 gr. ; then P.T. up to ·03 gr. During my absence one dose (·15 gr. P.T.) caused temp. of 105°, but slowly recovered ; then T.R. up to ·6 gr. ; wt. 9st. 3lbs. He also gave up coming ; any accident was possible.
He certainly improved under treatment.

27

—— 25. 8st. 10lbs.
No family history.

1 R. II ; L. II—III.
2 →
3 *Pleurisy*, 1904 ; 2 years ago treated by Drs. W. and S. I.
4 X.
5 Y. Poor eater.
6 72–80.
7 N.
8 Tb. G. 4. Sputum ℥j.
9 Hæmorrhage and pleurisy.
10 Hysterical.
11
12 For 2 years "plenty of food and fresh air" ; Katoomba for 2 summers, yet stage II–III.

Tuberculin: P.T.O. up to ·95 gr.; 9st.; then P.T. up to ·6 gr.; 9st. 11lbs.
Very unsatisfactory and disobedient patient, yet very great improvement.

28

x.06. 28. 8st. 7lbs.
11 years ago 1 brother $\theta\phi$, 10 years ago mother $\theta\phi$.
1 R. II.; L. II—III.
2 →
3 6 months.
4 A.
5 B.
6 N.
7 N.
8 Tb. G. 4. Sputum ℥j.
9 Hæmorrhages.
10
11
12 Treated with P.T.O. up to 1 gr.; then T.O.A. up to ·75 gr.; *in less than 2 months no cough or phlegm*; then P.T. ·015 gr. P.T. 101°.
9.i.07. ·015 gr. P.T. 101°.
16.i.07. ·015 gr. P.T. 98·4°.
20.i.07. ·025 gr. P.T. 101°.
25.i.07. ·025 gr. P.T. 100°; no further reaction till
4.iii.07. ·125 gr. P.T. 100°.
11.iii.07. ·15 *gr. P.T.* 99°.
18.iii.07. ·2 gr. P.T. 100°.
24.iii.07. ·25 gr. P.T. 102°.
2.iv.07. ·25 gr. P.T. 99°; no further reaction till
2.v.07. ·6 gr. P.T. 100°.
13.v.07. ·7 gr. PT.; 8st. 9lbs.
Feels and looks well; no cough and no sputum.
During whole of treatment went *every day* to work as clerk in office.

29

v.06. 21. 8st. 2lbs.
1 II—III or III.
2 →
3 Cough for a year.
4 X.
5 Y.
6 90.
7 N.
8 Tb. G. 6. Clumps; sputum ℥jv.
9 Pleurisy; laryngitis.
10
11 *b* or *c*.
12 Treated with P.T.O. to ·8 gr.; gained 8lbs.; wt. 8st. 10½lbs.; then P.T. up to ·075 gr.; pause; then P.T. again up to ·15.
22.xii.06. ·15 P.T. 101·4°.
28.xii.06. ·15 P.T. 101·6°.
4.i.07. ·15 P.T. 100°.
10.i.07. ·175 gr. P.T. 99·2°
17.i.07. ·225 gr. P.T. 100°; up to ·4 P.T. 99°.
vi.07. Had severe influenza.
vii.07. T.E. up to 1·8 gr.; lost wt.; wt. 7st. 11⅝lbs.; sputum reduced from 4 ounces to ½ ounce. In very poor circumstances, can hardly get food.

30

vi.06. 22. 8st. 4lbs.
1 R. II. L. II.
2 →
3 2½ years. Hæmorrhage, lost a quart; second Oj.
4 X.
5 Y.
6 84–96.
7 N.
8 Tb. G. 3. ℥j.
9 Pleurisy; hæmorrhage.
10
11 *b*.
12 Treated with P.T.O. up to ·9 gr.; then T.O.A. to 1 gr.; then T.R. up to ·4 gr.; then T.E. up to 4 gr. Cough and phlegm ceased. Subsequently a small hæmorrhage. When she came to me she could hardly walk a mile. After treatment she went up and down Federal Pass at Katoomba—a good task for me.

31

vi.06. 27. 8st. 10¼lbs.
1 R. II. L. II—III.
2 →
3 2 years.
4 X. Very poor health.
5 Y. No appetite at all.
6 90–100.
7 *f.* 99·4°, 100°, 101°.
8 Tb. G. 3. ℥ij.
9 Hæmorrhage; diarrhœa (prob. T.).
10
11

12 Sent to country ; no good.
Treated with P.T.O. up to 9 gr. ; then P.T. up to ·1 gr. ; gained weight and improved, but I suspect *abdominal (intestinal) tuberculosis* ; gained 8lbs. ; weight 9st. 4lbs. ; relapsed and treated again with P.T. up to ·15 gr. ; then pause for 5 months and again P-T. up to ·15 gr. (102°). From the outset I took a grave view of this case, and do not hope for any good result.

32

vi.06. 32. 10st. 12lbs.

1 R. II. L. II—II.
2 →
3 3 years and more.
4 A.
5 Appetite fair.
6 80–100.
7 N.
8 Tb. G. 3. Sputum >℥iv.
9
10
11 C.
12 Treated with P.T.O. to ·95 gr. ; then T.O.A. up to ·45 gr. ; then P.T. up to ·45 gr. No reactions of any account till September, then occasionally 100°. Very great improvement ; cough much better ; much less phlegm ; feels ever so much better.
1908. Know that she is still well.

33

v.06. 48. 7st. 11lbs.

1 R. II—III. L. I—II.
2 →
3 Since Jan. 1906?
4 X. 7st. 11lbs.
5 Y.
6 84–100–110. Even temp, of 104° only increases rate to 106–110.
7 *f* and F (104° and 105° apart from tuberculin).
8 Tb. G. 4. ℥iv sputum.
9 Hoarse for 4 months ; lost voice.
10
11
12 Previous treatment : Dr. P. did no good ; sent her to Dr. A. P. in Sydney, who said he "could do no more, and did not expect her to come back." Certainly a very grave case when I first saw her.
Tuberculin. 6.v.06. P.T.O. up to 1 gr., sensitive ; T.O.A. up to ·6 gr. ; then P.T. up to ·02 gr. In Oct. 1906 two reactions of 105°. Yet much better, and wt. 9st. 9lbs., *a gain of 26 lbs.* Went home for rest ; relapse ; came back to Sydney, 24.i.07. P.T.O. up to ·05 gr. ; wt. 8st. 9lbs. ; very sensitive ; ·05 gr. P.T.O. 104·6° ; then T.O.A. up to ·8 gr. ; then P.T.O. to ·65 gr. ; then T.R. up to 0·6 gr. ; wt. *9st. 4lbs.* ; then ix.07. P.T. to ·03 gr. ; very sensitive.
Dr. Griffiths has reported this case in *Aust. Med. Gazette*, 1907.

34

v.06. 42. 11st. 4lbs.
No family history.

1 R. II—III. L. I—II.
2 →
3 >1 year. 14 days after operation by Dr. M. for fistula, had a hæmorrhage from lung.
4 A.
5 Y. Bad eater.
6 96–100.
7 *f*. 99·4°, 100·6°.
8 Tb. G. 3. Sputum ℥ij.
9 Fistula 12 months ago.
10–11
12 "Heavier than ever in his life ; though bad eater, has gained wt. but got worse." Treated with P.T.O. to ·75 gr. ; gained wt. ; wt. *11st. 13lbs.* ; then P.T. up to ·175 gr. ; wt. 12st. ; temperature also fell by slow degrees to normal ; P.T. continued up to 1 gr. (28.x.06) ; very great improvement, which patient himself admitted, though at the outset he was a sceptic and pessimist. He merely came to me because he had seen the marvellous result in Case 2, Stage III. Further, he brought his wife to me to be tested. ·01 Old T. had no effect upon her.

35

iii.06. 33. 8st. 4lbs.
1 brother, 3 years ago, $\theta\phi$

1 R. II—III. L. II.
2 →
3 9 years.
4 X.
5 Y.
6 100–110°.
7 *f* 100°.
8 Tb. G. 4. Sputum ℥iv.
9
10 Pruritus vulvae.
11 *b—c*.
12 Treated with P.T.O. to 9 gr. ; then T.O.A. up to ·75 gr. ; then P.T. up to ·15 gr.; sensitive (102°, 101°, 101°, often 100°). After 14 days' treatment the temp. gradually fell to normal ; pause from xi.06 to xii.06 ; then P.T. up to ·5 gr. Wt. 8st. 8¾lbs.
This was a grave case, and the improvement was greater than I expected ; she looked very well.
April, 1608. — Heard from Dr. Griffiths that she had been to see him, and state of lung was quiescent.

36

iii.06. 40. 9st. 4½lbs.
Mother $\theta\phi$. 2 sisters $\theta\phi$.

1 R. II L. II—III.
2 →
3 Pleurisy 5 or 6 years ago ; fluid drawn off ; 16 years ago hæmorrhage. Oj.
4 X.
5 Y.
6 100—110.
7 *f* 101°—102°.
8 Tb. G. 6. M. Sputum ℥ij >.
9 Hæmorrhage.
10
11
12 Treated with P.T.O. to 1 gr. ; then T.O.A. up to ·5 gr. ; then P.T. up to ·175 gr. Gained 5½lbs., and temperature gradually fell to 99°. No other treatment. Man was able to come to me from Manly-at-Sea. Very great improvement, much more than I expected or promised. He went back to country. No news since.

1907

37

v.07. 47. 10st. 7lbs.
No family history.

1 R. II—III (cavity or bronchiectasis at base). Great retraction. L. II.
2 →
3 20 years ago pleurisy.
4 X.
5 Y.
6 90—100.
7 *f* 100·2°—101°.
8 Tb. G. 4. Sputum ℥iij.
9 Hæmorrhage (Oij).
10
11 *c*.
12 Previous treatment ; took creasotol for 1 year ; open-air treatment at mountains every summer. Tuberculin, P.T.O. up to 1 gr. and P.T. up to ·4 gr. (2.i.08). Temperature gradually fell to 99°–98° between doses ; sensitive. For months this patient gave himself the injections into his leg without any trouble after three weeks' instruction by myself. Many other cases in Queensland, New Zealand, Tasmania, and Victoria have been able to do the same without any trouble.

38

i.07. 42. 11st. 7lb.
No family history.

1 R. II L. I—II. Extensive and destructive ulceration of epiglottis, and infiltration of cords ; almost no voice.
3 Began as hoarseness and pain in throat 7 months ago ; sent to Dr. K. Then saw old friend, Dr. McK., who sent him to me. Dr. K., a specialist, said nothing could be done, and advised him to to go back.
4 X.
5 B. Good.

6 N.
7 N.
8 Tb. G. 5. Sputum ℥ij.
9 Ulceration of epiglottis and infiltration of vocal cords; some hæmorrhages.
10
11 *b.*
12 Previous treatment: gained weight, but disease advanced.
Tuberculin, P.T.O. up to 1 gr.; then P.T. up to ·8 gr. Extraordinary improvement. *Epiglottis* healed to all appearance; no cough or phlegm; *gained* 21*lbs.*; wt. 13st. Dr. McK. can vouch for these facts. Further, tested with Old T. in *Dec.* 1907, gave no reaction to ·05 gr. Old T. One of the most extraordinary cases in all my experience. He also gave himself most of the doses of P.T.

39

i.07. 18. 6st. 13lbs.
Always lived in country. Aunt, 14 years ago, caught ϕ.
1 R. II—III. L. II.
2 →
3 Cough since 1906. Pleurisy since April 1906.
4 X.
5 B. Appetite good, but patient very pale.
6 Pulse good, 80–88.
7 N.
8 Tb. G. 6. Sputum > ℥i.
9 Pleurisy since April.
10 Hoarseness for 3 weeks.
11 *b.*
12 Treatment in country of no avail. The patient was brought to me because the doctor held out no hope. Then tuberculin. P.T.O. up to 1 gr. In three months gained 16lbs.—8st. 1lb.; then P.T. up to ·1 gr. At this stage (May, ·07) a very sharp attack of measles. No injection from May 20 to June 8. Lost weight—7st. 13lbs. Then (8.vi.07) ·03 P.T. 102°; ·03 P.T. 99°; ·045 P.T. 99°; ·055 P.T. 99°; ·075 gr. P.T. 102°, up to ·9 gr. Wt. 9st. 2lbs. Immense improvement. No cough or phlegm; appetite very good, and *gained* 31lbs. It seemed like a miracle.

40

iv.07. 29. 7st. 10lbs.
1 R. II L. II—III.
2 →
3 9 months.
4 X.
5 Y.
6 96—106, 110.
7 *f* and F.
8 Tb. G. 4. Sputum ℥ii.
9
10
11 *c.*
12 Treated by Dr. H. in Wellington, N.Z., who sent her to me that "I might advise her to go to Dubbo," &c. Her history was very bad: seven acute attacks with temp. 103, 104; then abatement; then relapses. *Tuberculin* P.T.O. up to ·1 gr.; in 5 weeks pulse 84—86, wt. 8st. 6lbs.; P.T.O. up to ·1 gr., wt. 8st. 13lbs.; then P.T. up to ·1 gr.; *in* 3 *months gained* 20*lbs.*, 9st. 1½lbs.; P.T. continued to ·7 gr. (101°), pulse 78, wt. 9st. 6lbs.; extraordinary improvement, in 6 months gained 24lbs., lost cough and phlegm, and looks a fresh, active, healthy woman. I have Dr. H.'s own serious view of her case without tuberculin treatment.

Other cases (41, 42, 43, 44, 45, 46, 47, 48, 49 and 50) have been under treatment for but a few months, and I can assert that in every one of these cases, improvement began with tuberculin treatment, and will continue so long as the patients have patience and do their best to help themselves in their dire misfortune. Most of these cases are young girls: 41 *aet.* 31, 42 *aet.* 29, 43 *aet.* 25, 44 *aet.* 26, 46 *aet.* 17,

47 *aet.* 21, 49 *aet.* 30, 50 *aet.* 19. In all tubercle bacilli were present; but it would serve no useful purpose to do more than say that they are all under tuberculin treatment, and in many of these cases I anticipate a good result. Without tuberculin they would be inevitably doomed—for sanatorium methods and open-air methods do not pretend to be able to do much for such cases. The striking results in these late stages by means of very large doses is surely a complete answer to men who, following Wright, use homœopathic doses, and to men like Weicker, the great pioneer of the People's Sanatoria, who will, I hope, be encouraged by my experience to try large doses of tuberculin. I was much disappointed with Weicker's latest publication (1908), because one can see that he is still afraid of tuberculin. I think, on the contrary, that tuberculin is the only remedy for all cases of tuberculosis; if tuberculin fails, other methods must also fail. But one must not speak of the failure of tuberculin until it has been tried in large doses. It is not likely that my experience has been singular, or that other factors, such as the climate of Sydney, *which has for years been so heartily condemned by the medical profession*,[1] have played a great part in the successful results. My results have been so uniform and striking that I can recommend tuberculin in carefully graduated doses up to large doses at proper intervals of time with the utmost confidence. If such results can be obtained in a late stage (Stage II.–III.) may one not reasonably hope to cure all early cases of pulmonary tuberculosis with certainty by means of tuberculin? Dr. Weicker describes certain forms of toxæmia which resist treatment. I have rarely seen any cases of this description. They do not form 5 per cent. of the early cases, and in such cases I have shrewdly suspected other conditions, such as miliary tuberculosis, intestinal tuberculosis, genito-urinary tuberculosis or even extensive tuberculous lesions in glands or special structures (Addison's disease). Thus Koch's original statement in 1891 is absolutely true, and because the truth was not then recognised, tens of thousands and

[1] See my article in *Austr. Medical Gazette*.

hundreds of thousands have been sacrificed to the captious and ignorant criticism of Koch's great discovery in 1891.

We now pass to the cases of the Third Stage, when all methods alike must fail in most cases. All admit that when the lung is extensively diseased, and large areas of the lung have softened, and communicate with the air passages, infected ulcerated surfaces result, and mixed infection is the cause of exhaustion and death. Nevertheless even in late stages the energy of tissue may make some sort of fight to protect the individual. Certain it is that even with extensive cavity formation there may be little or no fever and even the pulse may not be fast. One danger is always imminent, the danger of hæmorrhage. Even a large vessel may be exposed on the ulcerated wall and, being more or less diseased and weakened, may suddenly give way and drown the patient in his own blood. Such an accident may occur even when the tuberculous process is not progressing. It may be due to a violent effort; it may suddenly occur at rest or during sleep.

Let me give three illustrations of these severe hæmorrhages in the Third Stage and in Stage II. (No. 47).

1

DECEMBER, 1903.

28. 8st. 1lb.

1 R. II; L. III (cavities).
2 →
3 3 years.
4 X.
5 Y.
6 110—114.
7 F. later *f* (100°—100·6°).
8 Tb. G. 4. Abundant sputum, M.S.
9 Hæmorrhages.
10
11 *b* to *c*.
12 Previous treatment of no avail. Tuberculin T.R. up to ·3 gr.; then T.E. up to ·125 gr., very severe reactions; then T.R. up to 1·2 gr.
27.vii.03. 1·7 gr. T.R. 103·4°; wt. 8st. 2½lbs.
24.viii.03. 1·5 gr. T.R. 102°.
10.ix.03. 1 gr. T.R. 100°.
24.ix.03. 1·5 gr. T.R. 99·4°; wt. 8st. 8¼; very great improvement. Pause, then T.E. again.
1.iii.04. ·2 gr. T.E. 102·4.
8.iii.04. ·2 gr. T.E. 103°.
17.iii.04. ·2 gr. T.E. 103°.
Pause again. During this pause patient was suddenly called to his gold-dredging works, and on the impulse of the moment made a very heavy physical exertion to put the machinery in order. He brought on severe acute pleurisy. He went to the mountains on his own initiative, and hæmorrhage occurred. He went, in fact, to a private sanatorium, where he was "stuffed." No doubt the altitude and the increased amount of food raised blood pressure, and contributed to the hæmorrhage. He returned from the mountains "a perfect wreck," and did not improve. He died from a sudden and severe hæmorrhage. But for his violent physical effort, and his ill-advised visit to the sanatorium in the mountains, he might have lived at least 2 or 3 years. At least so I read from his history.

2

AUGUST, 1902.

—— 30. 10st. 3lbs.
1 R. II—III (cavity or bronchiectasis; and great retraction of whole side. L. I—II.
2 →
3 2 years.
4 X.
5 Y.
6 N.
8 Tb. G. 3. Sputum ʒi—ii.
9 Pleurisy, bronchiectasis, hæmorrhage.
10
11
12 Treated with T.R. up to 2 gr.; then T.A. to ·06 gr.; hæmorrhage stopped; sputum greatly diminished; wt. 10st. 8lbs. Tubercle bacilli disappeared from sputum and sputum almost ceased. He got married in 1902. In 1903 he began to lose ground and yet would not take my advice and have more treatment till 16.iv.04; wt. 9st. 3lbs.; then gave him T.E. up to 1·3 gr.; gained 4lbs., but gave up treatment. In 1906 he went to a sheep station in the country, and one day, getting excited, shouted out to a station hand. A severe hæmorrhage ensued, and he died within 48 hours.

Again I say that, if he had taken my advice, he would probably have lived several years longer. Half measures cannot help a man who is in the late stages of the disease.

The third case is No. 47, Stage II. He had had hæmorrhages before treatment, and after full treatment had two severe hæmorrhages, but not followed by any fever. Some days after this hæmorrhage I took another physician at his own wish to examine him, and neither of us could find a single sign of active disease. A hæmorrhage from the lungs in such a case may be post-tuberculous, so that it is conceivable that a man may die of hæmorrhage after he has been cured of pulmonary tuberculosis. There was no reaction such as one would expect from the breaking down of a tuberculous focus; there was merely hæmorrhage from the rupture of a superficial vessel (which might be relatively large) already permanently damaged and weakened by an old and even obsolete tuberculous deposit.

The fourth case of hæmorrhage occurred in:

M. 20.
1 L. III; R. II. Acute pneumonic fever.
2 →
3 2 months.
4 X. Much wasted.
5 Y.
6 120—130.
7 F. Temp. 103°—104°. Continuous.
8 Tb G. 5. M. (pneumococci).
9
10
11 *c*.
12 I was called in consultation to see this case. Signs of pneumonic consolidation over whole lung, especially upper lobe. Temp. 103°, 104°. Gave 3 or 4 intravenous injections of collargol (1 c.cm. of 1%) and the temperature came down to normal, and did not rise again. Nevertheless, lung began to break down. Had a few small doses of P.T.O., but I gave it up, because the case was hopeless. Six months after I first saw him, he had a severe and sudden hæmorrhage, which killed him.

While upon the subject of hæmorrhage I can only repeat what I wrote in 1901, " that so far from hæmorrhage contra-" indicating tuberculin, tuberculin is the best means of " arresting and preventing hæmorrhage." I have always used tuberculin in cases of hæmorrhages—rather more cautiously—and have never seen a harmful effect ; indeed, in every case I have seen the hæmorrhage become less frequent and then stop. I do not remember a case in which hæmorrhage occurred for the first time after tuberculin had been used. Except in the cases given above, I have had no deaths from hæmorrhage. I am therefore bound to conclude that tuberculin is the very best remedy for hæmorrhage in all stages of pulmonary tuberculosis ; if hæmorrhage occurs, it is the fault of the vessel and not the fault of the tuberculin.

Next, I know of one case only in which pneumothorax occurred in any of my cases treated with tuberculin. In this case I find in my notes on physical signs, " signs of " very superficial cavity at right apex." This pneumothorax occurred in Prince Alfred Hospital a year after tuberculin treatment, and patient recovered from it—a rare occurrence Spengler has noted also that the prognosis of pneumothorax is far better in cases in which tuberculin treatment has been adopted.

To return to a review of cases in the third stage. If it is admitted by all that cases in the third stage are " past " remedy," some interest attaches to such cases that have been verily snatched from the grave. Even cases in the third stage have to be treated, and I have seen too many of them, because when the methods that are in vogue fail, people are ready to try anything—even the methods of the heretic. I have for some years adopted the practice of refusing to treat these advanced cases until the patients have been already examined by well-known medical men and pronounced to be " hopeless." It must damage a man's reputation to treat cases in the third stage. Even with tuberculin I do not expect to get a satisfactory result in more than 10–20 per cent. of cases. This means of course 80–90 per cent. of failures. Thus for every one who can

speak of the benefit derived from the use of tuberculin, there are four to nine people ready to condemn it on their own experience. Only, then, on the understanding that at least two competent men have pronounced the case to be in the third stage, will I make an effort to save the patient from death, at any rate for a few years. It may happen, and it has happened to me, that the doctors do not rightly measure the gravity of the case. Thus one case of severe tuberculous disease of the larynx, which had been treated for a year by a throat specialist, was sent to me, and I greatly astonished the specialist when I told him that the lungs were in the third stage of the disease. In another case of severe laryngeal tuberculosis, two doctors, one a throat specialist and the other a general practitioner, said that there was not much the matter with the lung when the disease in the lungs was in the third stage. Death in both these cases occurred within four months. *It would have been very easy for these doctors, starting with the idea that the lungs were not very seriously involved, to suppose that the tuberculin hastened the end.* In neither case could tuberculin do more than give temporary relief, and this relief tuberculin can give even in desperate cases.

The cases which I proceed to record have all been pronounced by two—generally three—well-known doctors to be "hopeless."

Three of these cases date back to 1902. They have, therefore, survived six years. One of them got married last year; another is at work in an office and looking the picture of robust health—a case in which tuberculous ulceration of the larynx gradually healed under the influence of tuberculin in large doses; and the third is, in the words of the surgeon who sent him to me as hopeless, a living illustration of the value of tuberculin treatment.

STAGE III.

1

ii.02. 17. 9st. 5lbs.
Fat and breathless, flabby.

1 R. III ; L. I—II.
2 →
3 2 years ; had music lessons from a nun who had consumption and died of it a few months later.
4 X.
5 Y.
6 Pulse 110°—120°.
7 *f* (100°).
8 Tb. G. 5.
9 Lost her voice completely ; used to sing well.
10 Pleurisy ; night sweats.
11 *c*.
12 Treated with T.E. up to 2 gr. ; many reactions (102°, 103°, 101°, 101°). Lost 6lb. in weight : all the better for it. Lost expectoration and recovered energy. Two intravenous doses of T.O. Pause. *Pulse*, 72—80 ; *weight*, 8*st.* 2*lb.*
iv.04. Then T.E. again up to 4 gr. (101°). ·07. Suspected relapse ; slight reaction to T.A. Gave P.T.O. 1 gr. and P.T. up to 5 gr.
Patient looks very well ; can sing ; has no cough or expectoration. Against my advice she got married at end of 1907. The doctors said she could not live three months in 1902.

2

DECEMBER, 1902.

—— 32. 9st. 6lbs.

1 R. III. Signs of cavity ; L. I—II—II.
2 →
3 Two years (13 doctors).
4 X 9st. 6lb.
5 Y.
6 92.
7 N.
8 Tb. G. 6. Sputum abundant.
9 Hæmorrhages ; ulceration of larynx.
10
11 *b c*.
12 Treated with large doses of Old T., T.E., P.T.O., T.O.A., and finally P.T. up to 6 gr.
Extraordinary improvement ; no cough or phlegm ; ulcer at edge of vocal cord spreading on to the interarytenoid area has healed.
No sign of active disease in lungs.
Jan. 1908. In splendid condition and back at work ; five years after weight 12st. 3lb. *Gained* 39*lb.*

Control Case.

His sister became affected later ; tried open-air treatment for ten months and died a few months later in 1906.

3

NOVEMBER, 1902.

—— 50. 11st. 5lb.

1 Extensive tuberculosis of genito-urinary tract.
2 →
3 A very long history ; 25 years. Began 25 years ago as tuberculous testis ; abscess formed, burst, and healed.
4 X.
5 Y.
6 N.
7 N.
8 Numerous Tb. in urine in clumps.
9
10
11

12 Patient was sent to me by Dr. M. after a cystoscopic examination in 1902, which revealed ulcers on surface of bladder; urine on settling deposited about 2 inches of pus, in which numerous (millions) of tubercle bacilli; great dysuria; cannot hold water for an hour. Treated with T.R. Lungs also affected I—II.; always reactions; 100°—101°.

12.i.03. ·2 gr. T.R. 103·4° up to ·85 gr. T.R. 102°; then T.E. up to .075 gr. T.E. 101·8°

1907, four years after, found urine absolutely clear, no albumen, no pus; had some prostatic trouble, causing obstruction. Dr. M. opened bladder, and during operation saw the scars of the old ulcers. wt. 13st. "A living illustration of value of tuberculin" truly.

In 1904 very remarkable results were obtained in four cases:—

4

—— 16. 7st. 10lb.

No family history. Pigeon-breasted. "Paralytic chest."

1 R. III. L. II.
2 →
3 2 years under Dr. S. S.; obeyed all instructions.
4 X.
5 Y. "Could not sleep," "wished to die."
6 110°—120°.
7 *f* 100°—100·6°.
8 Tb. G. 6. Sputum ℥j and >.
9 "Terrible" hæmorrhage; severe pleurisy.
10 Voice much affected.
11
12 Previous treatment:—2 years ago only a cough, no phlegm; took pounds of Dr. S. S.'s medicine, cod liver oil and maltine; saw Dr. S. once a week for six months, travelling 40 miles each time; "*rest cure*"; sent to country; "got much worse."

Tuberculin.—At first T.E.; very sensitive; 100·6°, 100°, 101.4°, 102°; then P.T.O. up to 1 gr.; gained wt.—8st. 6lb., increase of 10lbs. "Not two coughs a day since last dose," not a bit of phlegm. "Oh, it was grand!"; then T.E. up to 3.2 *gr.*, *large doses* for boy of 16, yet gained 21½lbs.; wt. 9st. 3½lb. *Pulse* 90.

One evening, not after a dose, but two days later, was telegraphed for. He had a very extraordinary attack. I found him delirious and violent, noisy and aggressive. It was difficult to keep him in bed. He had had no sleep on previous night. Temp. 101°—102°. This attack gradually wore off, and later, in 1906, I tested him and he did not react.

I have heard of him since and know him to be well.

5

—— 37. 11st. 2lbs.

1 R. III. L. II.
2 →
3 1¼ years.
4 X.
5 Y.
6 86—96.
7 *f* and F. 100·5°, 101°, 102°.
8 Tb. G 3 M.S. Much sputum (℥ij).
9 Tuberculous laryngitis; cords swollen and red; infiltration between arytenoids; very hoarse, almost aphonic. Dr. K. said "ulceration of cords." Dr. S. S. ordered "cod liver oil and a squirt."
10
11 *c.*
12 Gave P.T. up to ·3 gr. Pause for a month; then P.T. up to ·15 gr. Pause for 3 months; then P.T. up to ·75 gr. (22.i.06) without any reaction. Gained 32lbs. Wt. 13st. 6lb. Appeared a hopeless case at outset, especially with fever and tuberculous laryngitis.

1908. Saw him in excellent health and spirits. No cough or sputum, and good voice. It is easier to describe such a case than explain it. In 1904 Dr. K. made a point of visiting patient's mother, in London, to tell her that he was dying.

6

—— 26. 9st. 3lbs.

1 R. III. L. II.
2 →
3 5 years.
4 X.
5 Y. Very short of breath.
6 90, 96, 106 ; then gradually fell to 72—78°.
7 *f* 102°—101°.
8 Tb. G. 6. Much sputum > ℥ij.
9 Otis med. supp. chr. (Tub.)
1900 pleurisy ; hæmorrhages.
1901, rectal abscess ; lost wt. as low as 8st. 6lbs.
10—11 *c.*
12 Previous treatment :—Sent to Australia from India. In New England 1½ years ; felt well. Then, in Brisbane, got rectal abscess and relapse. 1902 at Katoomba. 1903 England. 1904 Macedon ; came to me end of 1904.
Tuberculin.—Treated with P.T.O. up to ·15 gr. Two very severe reactions (104·4°, 103·6°) ; then P.T.O. up to ·95 gr. ; gained 12lbs ; wt. 10½st.
In 1906, P.T.O. up to 1 gr. ; then T.O.A. up to 1 gr. ; then P.T. up to ·6 gr. (101·4°).
1.v.07. Tested. Reacted to ·01 Old T. ; then T.E. up to ·8 gr.
1908. Had several attacks of pleurisy last year. Is having further treatment.

7

—— 36. 7st.

No family history.

1 Stage III.
2 →
3 1 year.
4 X.
5 Y. No appetite.
6 80°—90°.
7 N or subnormal.
8 Tb G. 2.
9
10 Night sweats.
11
12 Open-air treatment for many months, yet Stage III.
Treated with T.E. up to ·05 gr. ; then P.T.O. to ·35 gr., sensitive ; then P.T. to .05 gr., very sensitive ; then P.T.O. up to 1 gr. (102°, 101°, 101°) ; then T.O.A. up to ·75 gr. ; then P.T. up to ·125 gr.
General and local improvement ; diminished sputum ; signs of lung drying ; appetite much better ; in very good spirits ; was much depressed ; and some gain in wt. ; 7st. 2lbs.

8

—— 39. 9st.

Great cyclist. Under eight doctors ; three times in country for "open air." No benefit.

1 Stage III.
2 →
3 More than 5 years.
4 X.
5 Y.
6 92°—96°.
7 *f* 100°—101°.
8 Tb. G. 6. M. Abundant sputum.
9–11
12 Treated with T.R. up to 1.2 gr. Gained 14lbs. ; wt. 10st..
Very remarkable improvement ; hardly any sputum ; "used to be a pickle-bottle full." At this stage his father had an apoplectic fit (cerebral hæmorrhage), lingered for several weeks, and then died. Patient being eldest son had to attend to his father's affairs and gave up treatment. Great shock and great strain caused relapse. Even so, patient did not return to me for nearly a year, and then I told him and his wife that it was too late. He died within six months. This case also illustrates the futility of half measures and moderate doses.

1905

In 1905 the two following cases improved immensely, but one went to Orange and the other to New Zealand, and I have had no news of them since :—

8a

—— 33. 7st. 12½lbs.

No family history, yet her sister, who has been with her, has been infected. Tb.

1 Stage III.
2 →
3 3 years.
4 X.
5 Y.
6 80.
7 N.
8 Tb. G. 3. Abundant sputum.
9–11 *c.*
12 Treated with P.T. up to ·225 gr, Very great improvement : better appetite, better energy ; looks far better and has gained 13lbs. ; wt. 8st. 11½lbs. Patient had then to go to New Zealand, and I have had no news since.

9

—— 44. 10st. 6lbs.

Brother, *aet.* 30, *θφ*, and first wife *θφ*.

1 Stage III.
2 →
3 Cough for 14 years.
4 X.
5 Y.
6 N.
7 *f* 100°.
8 Tb. G. 2. Sputum ℥iv.
9 Ext. ulceration in mouth exposing molar teeth. (Tub.)
10 Tubercular testis removed 2 years ago, and general pleurisy over whole of left side.
11 *b.*
12 Treated with P.T. up to ·5 gr. General health greatly improved ; sputum much diminished ; gained 2lbs. Was himself abundantly satisfied with result. No news since he left me.

10

The most remarkable case of all. Her father, a wealthy man, brought his only and favourite daughter to me when she was in the third stage of the disease. Till then she had been treated for anæmia. I told her father plainly that I could not help him. He implored me to try. I refused, because the girl could hardly stand, was quite breathless, pulse 120–130, temperature 102°, and the physical signs were extensive and grave over both lungs. The father was importunate, and I agreed to do my best if he first took three doctors from Sydney to consult about her condition and report truly. The consultation took place at his house, because the girl was too weak and ill to venture out again. The verdict of the three doctors in the highest position was, "She is dying." I thought so, too, but yielded to the father's urgent entreaties and took her to my private sanatorium. I gave her three or four intravenous injections of collargol (1 c.cm. of 1%), and the temperature slowly abated. I then gave her tuberculin, and I saw her last December, looking a picture of health, though there was evidence of a slight return.

FEBRUARY.

The notes on this case are as follows :—

—— 14. 5st. 8lbs.

1 R. II ; L. III.
2 → (3 leading doctors said "dying.")
3 9 months ill ; treated for anæmia, and was having "physical culture."
4 X.
5 Y.
6 120—128.
7 F. and then *f.*
8 Tb. G. 7. M.S. a cupful of sputum.
9 Hæmorrhages.
10 Epistaxis.
11 *c.* Utterly weak.
12 Treated with intravenous injections of collargol, and then P.T. At the end of October, 9 months,

temperature fell to normal, and wt. was 7st.; *a gain of* 20*lb.* Further treatment in 1906; P.T. up to ·35 gr.; then pause, and P.T.O. to ·75 gr., followed by P.T. up to ·225 (June, 1906), and then to ·35 gr.; wt. 7st. 6lbs.; *gain of* 26*lb.*

xii.06. Tested. ·0025, ·006, ·009, 99·6°, ·016.

vi.07. Tested. ·04 gr. of Old T.; no reaction.

Yet x.07. A slight hæmorrhage; pulse 100; temp. 100.

Having further treatment.

11

Another case improved enough to disconcert the physician in attendance. The patient was so weak that she lay in bed and could not even lift her head to feed herself. The doctor told me and her father that she could not live two months. I used tuberculin to prove that even *in extremis* it does no harm, if properly used.

xii.05.

—— 16. Under 6st, a skeleton.

1 Stage III.
2 →
3 1½ years.
4 X.
5 Y, too weak to sit up, a terrible skeleton.
6 110—120.
7 F. 102°—104°, later *f* 101°.
8
9 Intestinal tuberculosis also.
10
11 *c.*
12 Treated with P.T.O. and intravenous injections of collargol. After three doses temperature came down to 100°, but went up again to 101°; P.T.O. up to ·75; then few doses of T.O.A. and P.T. up to ·05 gr.

In 4 months the patient was able to get up and do a little work about the house. Before treatment she had not been out of bed for months. At the end of 4 months I told the father I could do no more. He was very grateful for the little I had done. She died 4 months after.

12

—— 18. 4st. 8lbs.

Servant in the house of a popular doctor. At least seven other doctors had seen this case, and they have come round to the view, which I maintained from the first, that she had tuberculous broncho-pneumonia and pneumonia.

1 Stage III. Complete consolidation of upper lobe of r. lung, with typical signs of acute croupous pneumonia, but failing to end by crisis, and fever continuing for months. The case was considered hopeless by all, but I expressed my willingness to take her into the Prince Alfred Hospital, so that the doctor might be relieved of her.
2 →
3 12 *weeks.*
4 X. 4st. 8lbs.
5 Y.
6 100—106.
7 *f.*
8 No Tb. found. ·001 gr. Old T. 103°.
9 Nil.
10 Abscesses in l. arm and back.
11 *c.*
12 Treated with P.T.O. and P.T.; very sensitive, but in spite of reaction gained weight and improved. Went up the country, and in 1907 returned to Sydney, and is back again with her old friend "the doctor." Wt. 8st.; *gain of* 48*lbs.*; a record.

The doctor was an utter sceptic—now he is a firm believer—and sent me Case 38 (Stage II—III), which has also startled him.

13

—— 35. 7st. 5lbs.

Is an equally instructive and successful case.

Father and mother alive and well. Patient married. Her husband died in 9 months of phthisis. Her two brothers also died. They were all treated by Dr. S. J. Patient had also been treated by Dr. S. J. and Dr. W. S., and was in Stage III when she came to me.

1 R. III; L. I.—II.
2 →
3 1 year.
4 X.
5 Y.
6 66·72. With reactions 100° P. 90, 101° 100, 102° 108.
7 N.
8 Tb. G. 7. Much expectoration ℥i.
9 Small hæmorrhages.
10–11 *b.*
12 Treated with P.T.O. Very sensitive. Many severe reactions (100·6°, 100·4°, 101°, 102°); it took five months to reach ·25 gr. P.T.O.; pause for 10 weeks; then P.T. up to ·2 gr.
v.06. P.T.O. to 1 gr.; T.O.A. to 7 gr., and P.T. to ·7 gr.
Great improvement in every way, hardly any cough, phlegm less than ever. "No phlegm from 3 in the afternoon till morning." "Feels wonderfully stronger."
Dec. 19.7. No cough, hardly any phlegm, no blood. *Contrast result with Dr. S. J.'s results in case of brothers and husband.*

14

—— 28. 7st. 2lbs.

1 Stage R. II; L. III. Large cavity (dry) extending into axilla? Hydatid?
2 →
3 Several years.
4 X.
5 Y.
6 N.
7 N.
8 No Tb. Profuse expectoration—quite a pint in 24 hours of anchovy sauce (no liver symptoms or enlargement). No tubercle bacilli to be found, but beautiful long chains of streptococci; never seen such long and beautiful chains.
9 Pleurisy.
10
11 *c.*
12 Before coming to me had been operated on for floating kidney, and in Prince Alfred Hospital had been needled several times by Mr. C. P. B. C. Tested in hospital by me ·001 gr. T.A. 103°. Treated with P.T.O. sensitive; 103°, 102°, 101°, but nevertheless *gained 24lbs.*; 8st. 12lb., and sputum greatly diminished; then treated with P.T. up to 1 gr.; phlegm now about ℥ss.
Seen late in 1907. Has been at work for last 2 years; seems very well. Is a very poor girl.

15

JANUARY.

—— 34. 7st. 13lbs.

Came to me, because Dr. M. said that surgery could do no more.

1 Genito-urinary tuberculosis, one testis removed and other resected. Urine contained pus, and frequent attacks of obstruction in the ureters—two or three in a fortnight. One attack lasted 10 hours. Since treatment attacks became fewer, and at the end of 10 months he had not had an attack for 7 months.
2 →
3 Many years.
4 X.
5 Y.
6 N.
7 N,
8 No Tb.
9
10
11
12 P.T. up to ·075; several reactions of 101°, 100°, 102°, and one 103°, yet he gained weight and improved; then P.T. up to ·15 gr.; pause, then P.T.O. up to ·8 gr., T.E. up to ·15 gr.; then P.T. up to ·6 gr.; then T.E. again up to 1·6 gr. (12.xii.07). Very great improvement, as his own letter tells, and wt. 8st. 5½lbs.
If one can do this in a hopeless case of genito-urinary tuberculosis, what may one do in a relatively early case?

16

NOVEMBER.

—— 21. 11st., fat and breathless.

1 Stage III.
2 →
3 3 years.
4 X. Fat and breathless. Wt. 11st.
5 B.
6 96—100.
7 N.
8 Tb. G. 5. Abundant sputum.
9
10
11 *c.*
12 Treated with P.T.O. up to 1.1 gr.; then T.O.A.; and then P.T. up to ·65; *lost nearly 2 stone*, to her own delight and my satisfaction, and at the same time improved beyond recognition.

She lived in Tasmania, 30 miles from a doctor. So I taught her and her mother the use of a syringe, and they together gave her a second course of treatment, chiefly with P.T. up to ·75 gr. for 8 months without any trouble. Tested in January, 1907, negative. Tested again *by herself* in September, 1907, also negative. December, 1907, no cough, no phlegm; wt. 9st. 13½lbs. Almost a miracle.

17

Let me add one more case, to which three doctors can also bear witness, one of them, I fear, reluctantly.

—— 6st. 12lbs.

1 R. II; L. III. Upper lobe absolutely solid.
2 →
3 2 years.
4 X.
5 Y.
6 110—130. Very nervous.
7 100°—102°.
8 Tb. G. 6.
9 Pleurisy.
10
11
12 See below for previous history; tuberculin treatment: P.T.O. up to ·95 gr., occupying 6 months, marked improvement, *7st. 10lbs.*; then P.T. up to ·015 gr., two doses of ·015 gr., 102·8°; pause for six weeks; then P.T. up to ·375 gr.; *wt. 8st.*; then severe intercurrent mixed infection, pneumococcal; temperature, 104·4° for some days; pause, then P.T. again to ·25 gr. (103°); wt. 8st. 5lbs.; in all, *a gain of 29lbs. in such a case.*

I have published the details of these hopeless cases treated successfully with tuberculin in large doses in order to show that in these late stages tuberculin succeeds when all other methods inevitably fail. Now if tuberculin may be used in these "awful" cases—even in large doses—with favourable effects, it would seem obvious that there can be no risk in using large doses in early cases. The one great bogey that was portrayed for us in such vivid and realistic colouring by Virchow was mobilisation of the tubercle bacilli. Virchow started the idea, unfortunately gaining a currency that it little deserved, that tuberculin had the effect of mobilising a sleeping enemy. Virchow's great name, and not the reality of the idea, did more than anything else to damn Koch's magnificent work. Even in the latest stages of the disease I have seen no evidence in favour of this mobilisation of

bacilli. It is all moonshine to talk of mobilising the bacilli in the early stages or latent forms of pulmonary tuberculosis. This mobilisation is a common event in pulmonary tuberculosis sooner or later, and the tendency which tuberculin has to cause fibroid changes in the tuberculous formation prevents rather than favours mobilisation. All my experience has convinced me that this idea of Virchow is a mere bogey that need not be considered. Let us give it a decent burial and say no more about it.

It is to me quite a mystery how, after rejecting the method of Koch, the medical profession in England developed a sudden enthusiam for Wright's method with his opsonic index and his homœopathic doses. As soon as Wright's work was discussed, I at once challenged the opsonic index as a guide in the graduation of doses. The truth was I had dozens of cases which proved this idea of Wright's to be utterly untenable. Moreover I instructed two men, independently of each other, and both of them well versed in Wright's very clever technique, to test the blood of some of my cases which have been treated by means of large doses and with a total disregard of Wright's method. One of these observers, being very friendly to Wright, excused himself several times from giving me his results. He found the opsonic index *so high* that he thought he must have made a mistake. The other man supplied me with these startling opsonic indices, 2·3, 1·78, 1·57, 1·6, 1·3, 1·2, and in no single instance was the opsonic index under 1. Accordingly by disregarding the negative phase and proceeding on my own plan in contravention of all Wright's ideas and methods I arrived at results which were proved by Wright's own methods to be infinitely better than his own. In fact, I hoisted my old friend Sir A. E. Wright with his own petard. Would that the wave of enthusiam that ushered in Wright's work in 1904 had been reserved for a better cause. Sir A. E. Wright cannot be ignorant of the work that I was toiling over long before he thought of entering the field ; and although I know that my method is infinitely better than his, I had to labour conscientiously and patiently for nearly seven years before I published anything, and when I did publish

there was nobody to take any notice of my work. From my standpoint at any rate I am bold enough to assert, that even in very late stages large doses of tuberculin may do a marvellous amount of good, while nothing else does any good at all; why then should large doses do harm in the earlier stages? *A priori* there is no reason to expect harm, and *a posteriori* I have proved in hundreds of cases that large doses do an extraordinary amount of good, far more good than any other known method.

Surely, too, my experience is only in harmony with the results of experiments. The aim of tuberculin treatment is to stimulate and increase the vigour and amount of the antibodies or weapons of defence evolved by the activity of living tissue, and the more perfect the response to this antigen, tuberculin, the better the mechanism of immunity. Surely the obvious inference is that progressive and relatively large doses will develop a more perfect and more responsive mechanism of immunity than homœopathic doses of the same strength repeated at long intervals of time. Again let experience teach. The almost invariable effect of increasing doses of tuberculin has been general improvement and abatement of the symptoms. I do not deny that much skill and experience may be necessary to graduate the doses, but if we are aiming at a distant goal, we cannot expect to reach it by marking time or standing still. If anyone, who has tried the method of small doses for a number of years, has been able after a long interval of time—certainly not less than three or four years—to look back upon the results with exhilarating satisfaction, then he is justified in using and advocating small doses. I can look back over a number of years with great satisfaction upon results that have been achieved with large doses, and shall continue to use large doses.

PERCENTAGES IN FULLY TREATED CASES.

1902

Results in 1908 :—

Stage I. 7 out of 8 treated alive and well ; the other one went to New Guinea and died of severe fever (malaria) ; he had a double tertian infection while I was treating him.

Stage II. 4 cases : all alive ; 2 of these cases quite well for five years, but now having further treatment.

Stage II–III. 1 case quite well ; 1 case no news lately ; 1 case died in 1907.

Stage III. 6 cases : 3 alive and wonderfully well ; 3 died within a year of treatment ; all hopeless from the outset.

Percentage results after 6 years :—

Stage I. 100 per cent.

Stage II. 50 per cent. well.

Stage II–III. 50 per cent. well.

Stage III. 50 per cent. well.

1903

Results in 1908 :—

Stage I. 14 cases : 1 died of abdominal operation ; 2 were well in 1906 ; 11 remaining alive and well.

Stage II. 3 cases : 1 fully treated, quite well ; 1 partially treated, lost sight of ; 1 who gave up treatment after a few doses, died in 1904.

Stage II–III. 4 cases : 2 quite well ; 1 gave way to drink and relapsed.

Stage III. 2 cases not treated by tuberculin died—one refused treatment when in Stage I ; other was sister of Case 2, Stage III : he had tuberculin and is wonderfully well ; she had no tuberculin and died two years ago ; 1 case died of hæmorrhage.

Percentage results after 5 years :—

Stage I. 100 per cent.

Stage II. 100 per cent. of cases fully treated.

Stage II–III. 66 per cent. (the other case should be well but for potus).

Stage III. 0 per cent.

1904

Results in 1908 :—

Stage I. 13 cases : all alive so far as is known ; 1 case lost sight of since 1906 ; other 12 alive and well.

Stage II. 8 had full treatment and all well ; 2 had partial treatment, improved and left off ; 1 refused tuberculin treatment and went to sanatorium—he is dead.

Stage II–III. 5 cases : 3 fully treated, very well ; 1 partially treated, alive ; 1 case left and not heard of.

Stage III. 7 hopeless cases, died within 6 months ; 1 case lost sight of ; 4 cases fully treated, marvellously better.

Percentage of *fully* treated cases :—

Stage I. 100 per cent.

Stage II. 100 per cent.

Stage II–III. 100 per cent.

Stage III. All cases fully treated, marvellously better.

1905

Results in 1908 :—

Stage I. 27 cases : 4 lost sight of ; other 23 cases well.

Stage II. 13 cases : 1 case refused treatment and died within a year ; 8 fully treated, known to be well ; 4 gave up treatment after a few doses.

Stage II–III. 10 cases : 1 had 2 months' treatment only and died in a year ; 1 had two doses, not heard of ; 8 other cases marvellously well.

Stage III. 20 cases : 7 cases quite hopeless, some improved but history unknown ; 7 were too bad for treatment ; 6 fully-treated cases are extraordinarily well.

Percentages after 3 years :—

Stage I. 100 per cent., so far as is known.

Stage II. 100 per cent., fully treated ; the only case known to be dead refused tuberculin.

Stage II–III. 8 out of 10 were fully treated ; 100 per cent.

Stage III. 6 out of 14 cases very well ; 45 per cent.

1906

Results in 1908 :—

Stage I. 24 cases : all well.

Stage II. 24 cases : not a single death ; 22 known to be well.

Stage II–III. 15 cases : 8 known to be well ; 4 left off treatment ; 1 not at all well ; 2 partially treated, died.

Stage III. 25 cases : 11 died within 6 months, having had little or no treatment ; 7 cases more or less treated, improved ; others lost sight of.

Percentage results in 1908 :—

Stage I. 100 per cent.

Stage II. 90 per cent.

Stage II–III. 50 per cent.

Stage III. 30 per cent. improved.

AFTER RECORDS.

Summary of Results observed in 1907—1908.

Cases treated in 1902 :—

Stage I. 7 cases : 1 man went to New Guinea and died of malaria ; no deaths known ; 5 cases known to be well.

Stage II. 4 cases : all alive ; 2 had relapse after 5 years, but are improving again.

Stage II–III. 3 cases : 1 quite well, at school ; 1 lost sight of ; 1 very late, died in 1907.

Stage III. 5 cases : 3 marvellously well ; 2 died within a year, hopeless from the start.

Treated in 1903 :—

Stage I. 14 cases : 1 died of abdominal operation 1907 ; 1 partially treated, lost sight of ; 11 known to be well in 1907 ; 1 well in 1906, not heard of since.

Stage II. 3 cases : 1 case gave up treatment after few doses, he died within a year : this is "control" case ; another went away after 6 weeks, lost sight of ; 1 known to be quite well in 1908.

Stage II–III. 3 cases : 2 remarkably well in 1908 ; 1 improved immensely and then gave way to drink, yet alive in 1907.

Stage III. 1 case : improved immensely under treatment, but through foolish act brought on hæmorrhage and died.

Control cases.—In 1903 had two "control" cases. One was tested by me in 1901, was then in Stage I. She refused tuberculin treatment, and in 1903 was in Stage III., hopeless ; another had sanatorium treatment for a year and was dying.

Treated in 1904 :—

Stage I. 13 cases : all alive and well.

Stage II. 10 cases : 2 left off treatment, could not afford time ; 1 partially treated, great improvement, left off coming. Remaining 7 well.

Control case.—1 "control" case. This patient decided at first to have treatment. Then persuaded to go to sanatorium. *Was exhibitea in* 1905 *as successful case of arrestea disease*, and was dead in two years and a half.

Stage II–III. 5 cases : 2 left off treatment ; other 3 surprisingly well in 1908.

Stage III. 14 cases : 9 hopeless cases, died within a year ; 2 cases improved extraordinarily ; 2 other cases were alive in 1908 ; 1 case improved greatly, but lost sight of since 1906.

Treated in 1905 :—

Stage I. 27 cases : 22 had full treatment, and all are known to be well in 1908 except one, who died of splenic anæmia in 1906 ; 5 had partial treatment.
Control case.—M. 30, Stage I., refused treatment by me, "because she saw another doctor, who did not find tubercle in sputum." In 1907 Stage III.

Stage II. 12 cases : 3 had only few doses ; 8 had full treatment, and were well in 1907.
Control case.—1 "control" case : put off treatment because he was too busy ; he died within a year.

Stage II–III. 9 cases : 8 improved immensely, and were well in 1908.
Control case.—1 gave up treatment because he had not time ; he died within 18 months—a further "control" case.

Stage III. 20 cases : 9 hopeless cases, could not take treatment, died within 18 months ; 7 cases are alive, marvellously better ; 4 cases temporarily improved.

Treated in 1906 :—

Stage I. 28 cases : 24 fully treated, all well ; 4 partially treated.

Stage II. 26 cases ; 22 cases well ; 4 cases partially treated.

Stage II–III. 14 cases : 2 deaths before completion of course of treatment ; 3 improved but left off ; 1 not benefited ; 8 cases very well.

Stage III. 20 cases : 13 cases hopeless, and died within 6 months ; 7 improved very much, two of them to an extraordinary degree.

Treated in 1907 :—

Stage I. 34 cases : 25 cases fully treated, all better or well ; 7 partially treated ; 2 to have treatment.

Stage II. 21 cases : 10 had treatment, and are better or well ; 9 still under treatment ; 2 gave up after a few weeks' treatment.

Stage II–III. 14 cases : all improved, 4 fully treated are surprisingly well ; others under treatment.

Stage III. 12 cases : 9 hopeless ; 3 improved.

These records bear out the conclusions I had formed in 1902, that in Stage I. tuberculin secures a successful result in almost 100 per cent. of the cases, certainly in 90 per cent. of the cases, *controlled by after examinations even five years after treatment.* Even in Stage II. my results with tuberculin show successful treatment in fully 60 per cent. to 70 per cent. of the cases. According to my experience it is quite exceptional to meet with a case in Stage II. which cannot be arrested for many years, and it is my opinion, based upon experience, that if these cases are subsequently examined once a year by means of tuberculin, and, if reacting, treated further, the disease may be held in check, and perhaps even

permanently arrested. With regard to the later stages, I have been surprised beyond measure at the many successes I have obtained in Stages II.–III., and even in Stage III.

Indeed, to any one who has had my experience, it appears clear that the results claimed by Moeller and Weicker, Bandelier and others, for the combined methods of the sanatorium and tuberculin treatment, are in effect the *results of tuberculin treatment.* Accordingly, if a scheme for the exploitation of tuberculin treatment among the poor be substituted for sanatorium treatment, we shall be acting in obedience to the direct teaching of science and experience. As I have already indicated, *the ideal method of exploiting tuberculin treatment among the poor is by means of city dispensaries for consumption instead of sanatoria* (see Appendix).

APPENDIX

APPENDIX

THE *RÔLE* OF THE CITY DISPENSARY IN THE CRUSADE AGAINST CONSUMPTION

Address by W. CAMAC WILKINSON, *B.A., M.D.Lond., F.R.C.P., Alderman of the City of Sydney, Lecturer in Medicine, Sydney University, and Hon. Physician, Prince Alfred Hospital*, 1904.

(FROM SHORTHAND NOTES.)

LADIES AND GENTLEMEN,—

I wish to speak to-day in a dual capacity—as a physician who has for many years studied the question of the prevention of consumption, and as an alderman of the City of Sydney who considers that, as tuberculosis is the special curse of cities, it is the primary duty of the City Council to do something to arrest the spread of this disease. It is, I think, an open secret that my entry into the municipal life of Sydney was due to an earnest, and I hope a righteous, desire to help forward the crusade against consumption. I consider, and I am speaking from a considerable experience and a more or less intimate knowledge, that there is no greater problem awaiting solution at the behest of medical science, in the name of humanity, than the prevention of consumption. The experience which we have recently gained owing to the visitation of plague has demonstrated two very important facts. Firstly, it has been shown that it may be necessary in the interests of public health even to interfere with the personal liberty of the subject, in order to protect the community against the fault or the folly or the misfortune of the individual. Secondly, this visitation in Australia and elsewhere has demonstrated the enormous value of scientific discoveries in dealing with the prevention of this disease. The success which attended the efforts of the Board of Health and the City Council in dealing with the outbreak of plague was essentially due to a practical and intelligent exploitation of the discoveries of science.

We know that, of late, all over the world a great wave of municipal activity has invaded local governing bodies, and the very state of our streets alone shows that this epidemic of municipal activity has also visited our own shores. Within the last two or three years the City

Council has launched a very costly and a very elaborate scheme for the exploitation of electricity as a source of motive power, of heat, and of light. Of course, it is for scientific authorities to probe the hidden secrets of Nature and to explain their discoveries ; it is for practical men and governing bodies as far as possible to utilise these discoveries in the interests of the general community. In introducing electricity into our city, the City Council hopes to save money, and perhaps to reduce taxation. I come to plead for the exploitation of medical science, not, indeed, to save money or reduce taxation, but, in the interests of humanity, to save lives. Hundreds of lives can be saved every year in our metropolis by the prevention of consumption. May I state in a few words what I mean by consumption? In simple language, with a practical, though not perhaps strictly scientific, meaning, consumption may be, for our purposes, described as the infectious form of Tuberculosis. There are many forms of tuberculosis. When I speak of consumption this afternoon, I am referring to the infectious form of tuberculosis ; but there are many forms of tuberculosis that are non-infectious. It is a mistake to suppose that even plague is always infectious—or, in other words, that it is easily conveyed from man to man. In bubonic plague, the infectious germs are so firmly imprisoned in the tissues that they cannot escape to infect. There are, however, very infectious forms of plague. In like manner, tuberculosis may for years, and even for a lifetime, remain non-infectious. In children, tuberculosis meningitis is a very common form, and is absolutely non-infectious. Even tuberculosis of the lungs may remain for years, even for a lifetime, non-infectious ; but the most infectious form of tuberculosis in man is known as pulmonary consumption. Nevertheless, it is equally true that the prevention of the infectious form of tuberculosis is tantamount to the prevention of all forms of tuberculosis, because the infectious form is the source of all forms of tuberculosis. That is a very important fact to recognise. Tuberculosis is a world-wide disease, which has been more closely and more accurately studied than any other disease. We know its origin, we know its nature, yet in spite of this knowledge, in spite of very obvious reasons for adopting some system of rational prophylaxis based upon scientific discoveries, tuberculosis is allowed to extend and multiply its ravages in this and many other cities, almost without let or hindrance. Tuberculosis is the special curse of cities, being favoured, alas ! by conditions that are more or less inseparable from city life. Overcrowding, want of space, dampness, want of air, want of sunlight—these are all conditions that favour tuberculosis. These, too, are the usual accompaniments of poverty, and poverty too often calls into its aid the dread spectres of the tenement—intemperance and ignorance. Intemperance, Ignorance, and Tuberculosis ! These are the devouring vultures that prey upon the life and health and happiness of those who live in tenement houses in all cities. Too often, alas ! these instruments of destruction increase, intensify, and perpetuate the sorrows and afflictions of the poor ; in their cruel and pitiless career, demoralising, ruining, and even sometimes annihilating whole families.

Inasmuch as tuberculosis is essentially a disease of cities, I hold it to be one of the primary duties of the City Council, not merely to join, but to lead and direct this crusade against consumption. Let us consider the prevalance of this disease in various cities of the world. Of course, in newer countries, this infectious disease is not so prevalent as in the older countries of the world. We have here a diagram which shows the incidence of this disease in the chief cities of Austria and Germany. I have added also the cities of Melbourne, Sydney, and Brisbane. I may say that in Vienna, in Prague, and in Moscow the mortality used to be something like 10,000 per million living souls. Since the discovery by Professor Koch of its infectious nature in 1881, and probably as a direct result of the knowledge gained by his researches, the mortality has been reduced in these cities by one-half. The statistics of ten years ago show that in the chief cities of the world—of more than half a million inhabitants and upwards—to begin with the highest, the mortality in Moscow and St. Petersburg was about 4,500 per million of living souls ; in Vienna it was about 4,200, in Paris about 3,800, in Berlin about 2,200. In London it is much lower—about 1,600. In Hamburg and Naples it is relatively low. In Melbourne it is about 2,200, and in Sydney 1,200. But, in Sydney, the most recent records show (I have the statistics in my pocket, published within the last fortnight) an evident increase of tuberculosis in the City of Sydney and in New South Wales, even relatively to population. In Brisbane the record is the lowest in the world ; but it is hardly reliable, because probably the basis of the statistics is not the same as those in other cities. In Sydney one may say that there are about 650 deaths from consumption every year. (I do not, of course, speak of the city under the immediate control of the City Council, but of the whole metropolitan area.) But you have to add to this a very large proportion of the deaths from pleurisy, which is essentially a form of tuberculosis. If one adds the different forms of tuberculosis together, I am pretty certain there is a death-rate of something like 800 or 900 every year from tuberculosis.

We have to remember also that tuberculosis is a disease that generally maims and disables a man for many years before it kills him. From an economic point of view, this is very important. It is also a disease which strikes its victims in the very prime of life, and in this respect it essentially contrasts with the incidence of cancer in most cases. Now surely there is an economic interest in checking a disease which causes something like 800 deaths a year, and probably maims and disables, at any rate, four or five times that number. If we consider the human unit merely as a consumer, as a producer, and as the head or part of a family, even a low estimate makes his value to the State, at any rate, equal to £50 a year. Now, supposing we even keep alive every such individual for six years, it would mean that a total loss would be saved to the State of £300. Multiply £300 by 800, and you see that the loss to the community, to the State, represented purely in £. *s. d.*, is enormous.

The prevalence of this disease is appalling. Tuberculosis kills, at any

rate, five times as many people as typhoid fever; ten times, perhaps fifteen times, as many people as diphtheria; twenty or thirty times as many people as hydatids; thirty to fifty times as many people as measles or scarlet fever; and one hundred to a thousand times as many people as small-pox, snake-bite, or even as plague. I have selected these diseases for comparison because the Government of the State spends tens of thousands of pounds every year in order to prevent them. In contrast with this more or less energetic attack on these diseases, it is strange—indeed, it is inexplicable—that little or nothing should be done to check the ravages of a disease *which claims more victims and causes more deaths* than all the diseases which I have mentioned put together. What is the cause of this extraordinary apathy in the face of such an enemy of mankind? There are no doubt many contributing factors. In the first place, for a very long time the disease deceived the very elect. The medical profession has only recently recognised its infectious nature. For many generations this disease has deceived and thus disarmed mankind. Literature and art have helped in the delusion. If we examine some of the finest works of art in Italy, we find the tubercular type represented as models, and as a type of beauty. Botticelli, and still more recently Rossetti, have drawn and painted from consumptive models. In Tennyson's "May Queen" we have the poetical embodiment of that peculiar phthisical habit of mind that hopes against utter hopelessness. In one of the best novels of recent times, "Robert Elsmere," the hero, is struck down in the very prime of life by consumption. Recently in Sydney we have had the opportunity of witnessing Puccini's entrancing opera, "La Bohême," in which Mimi meets her tragic fate from consumption before the very footlights. I tell you hundreds and thousands of our fellow-citizens are playing the same pathetic *rôle* as Mimi—not, indeed, before the footlights, but upon the world's stage of stern reality. Thus Sentiment, in all the superb settings of fiction, poetry, and art, has cajoled and narcotised us with these romantic and fanciful pictures of death from consumption. But at last Science, through the transcendental genius of Professor Koch, to whom the world owes a debt of gratitude which it hardly realises, and can never repay, has torn away the mask from the face of this treacherous enemy of mankind, and revealed him to us in all his hideous nakedness.

Tuberculosis is no open enemy. It works its way by stealth and ambush. It may lie in ambush for many years, and is always invisible in its beginnings. It begins so quietly that it never excites alarm or dismay. It progresses so slowly that it gives one plenty of time to get accustomed to its presence. Sudden deaths, sudden catastrophes, attract our attention and appeal to us most powerfully by their very suddenness. Cholera strikes down its victims without a moment's warning; small-pox has caused a panic in our midst. These sudden catastrophes alarm, appal, horrify us; but this disease, that is ever with us, that spares few families and may wreck whole families, that causes more trouble and sorrow than perhaps any other disease, that counts its victims not by

hundreds but by thousands, that is no respecter of persons, invading equally the palace and the cottage—this disease, forsooth, because it is so common and so unpitying, in spite of our very accurate knowledge with regard to its nature and origin, in spite of obvious reasons for adopting some system of prophylaxis based upon science—this disease, forsooth, is allowed to extend and multiply its ravages in our city almost without let or hindrance. What is the cause of this apathy? In my judgment, this apathy is only another name for fatalism—a fatalism born of the popular, erroneous, and very mischievous doctrine that consumption is a constitutional and hereditary disease. If consumption were of this nature, if tuberculosis were due to some inherent inborn condition of tissue, 'twere well to bear this sad degree of fate with the heroic calmness of the Stoic. But it is not true ; it is absolutely false, and until public opinion is fully seized of the true nature of this disease, we cannot expect to move those mighty engines of progress and enlightenment, the Press and Parliament, to recognise their duty, and perform a great public service. I say with all the energy of my nature that consumption is not a constitutional or an hereditary disease. Constitutional tendency is powerless, hereditary tendency is equally powerless, chills and colds are powerless, to cause consumption, unless the tissues are invaded by the living germ which Professor Koch discovered in the year 1881.

A quarter of a century ago Professor Koch established beyond all dispute that consumption was an infectious disease, and I think we should have reached, nowadays, that stage when the paramount idea upon which we should deliberate and act is not merely that tuberculosis is an infectious disease, but rather that *tuberculosis can be and should be prevented, because we know its nature and origin, and its source and distribution are proved to be limited.* In order that you may rightly understand and appreciate the scope and purpose of my proposal, it will be necessary for me to discuss very briefly the nature of the infection, and especially its source, for unquestionably the surest way of preventing infection is to deal with the infectious material at its source—it may be by destroying it ; it may be by placing it beyond the range of risk, or in other ways by rendering it harmless. Science has placed beyond all doubt the true nature of consumption. Without the germ—the tubercle bacillus, as it is called—there can be no tuberculosis. This germ, or bacillus, is a true parasite. It is not ubiquitous. It grows under natural conditions exclusively in the living tissues of man and animals. How far tuberculosis in man may be traced to tuberculosis in cattle is one of the great questions of the day. But I believe that if tuberculosis were eradicated from cattle, the effect in reducing the mortality amongst human beings would probably be very small. The source of infection is the source of this bacillus, and I believe the main, if not the exclusive, source of this bacillus is man suffering from the infectious form of the disease—pulmonary consumption. Nevertheless, infection requires certain conditions. The infection may be potential or actual. Actual

infection requires that the infectious material should be carried from the sick to the healthy. Ample evidence shows that the chief sources of infection are the overcrowded homes of the poor ; and the prevalence of tuberculosis in other classes of society is largely due to its prevalence in the homes of the poor.

There are at least three different ways of dealing with this infectious disease in the cause of public health. For half a century and more the philanthropic spirit of London has built and maintained Hospitals for the reception of advanced cases of consumption. High authorities hold that this plan devised for the treatment and relief of the disease has had a further beneficent effect in preventing consumption by bringing about the segregation of highly infectious cases. This result was quite accidental and unintentional. Nowadays, Hospitals for advanced cases of consumption are regarded as an indispensable element in any system of rational prophylaxis. Half a century ago, Brehmer initiated in Germany the open-air treatment in special institutions (Sanatoria) for the cure of consumption. The value of Sanatorium treatment on the rational dietetic system has been clearly demonstrated by Brehmer and his followers ; but it has its conditions and its limits. The Hospital for advanced consumption aims specially at prevention, and is in the interests of the State. The Sanatorium aims specially at the treatment of the few, and is mainly in the interests of the few. At any rate, in the cause of public health, the Hospital for advanced consumption is of far greater public utility than the Sanatorium. As an economic measure, the Sanatorium must be reserved for cases of tuberculosis in the early stage. The system is so troublesome and costly, and involves such loss of time and wages, that it cannot be advocated on economic principles in the interests of the poorer classes, unless there is a good prospect of such a return to health that the patient can again become an efficient bread-winner. To compass this object, the Sanatorium must be reserved exclusively for early stages of the disease. Germany has worked out the problem for us, chiefly by means of social legislation, which compels the workman to insure against accident, disease, and old age. It is waste of time and money—and manifestly unfair to those in an early stage of disease—to send cases in the third stage to a People's Sanatorium. As the result of such a policy, patients in the first stage have to be rejected, and perhaps, when their time comes, some of them will be in the second or third stage of the disease. At the best—even with the most careful selection of cases—permanent arrest of the disease may need a six-months' stay at the Sanatorium, and fails in 50 per cent. of the cases. Sanatorium treatment has reached its highest development in Germany, where the success of the People's Sanatorium is largely due to wise social legislation. Whatever may be thought of Monarchical or Imperial systems of government, the social laws of Germany might well be imitated in communities that boast of Democracy. The health of the German artisan has been the subject of the Emperor's special attention and solicitude—partly, perhaps, because every healthy man is an

efficient fighting unit. The huge army of Germany aims at the maintenance of a high standard of efficiency, which depends absolutely on the well-being and health of the human units. Health is also the capital of the labourer, and if through failure of health the labourer becomes a charge upon the State, it is to the State's interest that his health should be protected, and, if his health fails, that he should get well as soon as possible. By compelling the artisan to insure against sickness, the social law of Germany is making a colossal effort to protect the State by taking care of the human units. The object of this law is to maintain the health of the unit and to hasten recovery from sickness. A huge fund is provided by the masters and men, which is spent entirely in the interests of the artisans themselves and their families when they are stricken with sickness. These grand State Insurance Societies have a reserve fund of £37,000,000, and spend over £20,000,000 a year in relieving the lot of the artisan class. Pulmonary tuberculosis in Germany, as elsewhere, is far the most frequent cause of sickness, with consequent loss of wage and work, and, among other ways of dealing with pulmonary tuberculosis, a large part of this fund is devoted to treatment in Sanatoria. But it must be remembered that those who benefit thus by Sanatoria have themselves contributed to the general fund. Thus, in Germany, Sanatorium treatment is no charity, but part of an elaborate policy of State-enforced, not State-supported, insurance. These two distinct plans—Hospitals for the segregation and treatment of advanced cases of consumption, and Sanatoria for the treatment of the disease in its early stages—are well known, and have existed for many years.

The City Dispensary as a weapon of attack in the crusade against consumption is a relatively new idea, which may be recommended unreservedly because it is founded on the sound principles of economy and efficiency. In different countries the idea of establishing City Dispensaries has appealed to expert authorities on various grounds. Professor Calmette, of Lille, the great pioneer of this new development in France, advocated Dispensaries as a means of educating the masses. If his method has not met with the success it undoubtedly deserves, the fault lies rather with the masses than with the system. Calmette appealed to their intelligence in the hope of stimulating a desire for self-help and self-improvement ; he hardly appealed to their self-interest, because treatment was not a primary object of his system. Others have modified his system so far as to introduce self-interest as a main motive force in the Dispensary system. For many years I have advocated the Dispensary system, combining therapeutic, prophylactic, and social functions, as a highly convenient, economical, and effective way of securing *voluntary notification*, which is an indispensable factor in any system of rational prophylaxis. Notification is a primary condition of success in any scheme for the prevention of consumption. In the present state of public opinion, it is idle to propose compulsory notification. This may come in time, but not until public opinion has

been educated to understand the meaning of, and necessity for, notification. Meanwhile, we may be content with *voluntary notification*. It is only by some form of notification that we can discover the primary sources of infection, and, until the original sources of infection are known, our methods of dealing with the infection must be haphazard, indirect, and to a great extent ineffective. To quench infection at its very source is ever the great aim of the hygienist. We destroy rats to extinguish the infection of plague ; we aim at purifying the water supply in order to prevent or extinguish outbreaks of typhoid fever, dysentery, and cholera. In like manner we should attempt to deal with the infection that causes tuberculosis and consumption, by destroying it at its chief sources, or at least by rendering the infectious material harmless. It is a sad fact that the overcrowded homes of the poor are the chief breeding-grounds of the germs of consumption. If we wish to eradicate infection, we must attack and destroy or remove the infection in these homes, which are otherwise veritable hotbeds of infection. This is entirely in the interest of the sufferer, but especially of the sufferer's relations and friends, and even in the interest of the whole community, for the infection may spread from these centres widely in ever-increasing circles. It requires no vivid imagination to realise how, in the over-crowded homes of the poor, one person suffering from advanced consumption may infect the whole family. I have had sad experiences of instances in which whole families have been swept out of existence by this infection in the family circle. Such infection spreading in the family gave rise to the erroneous notion that the disease was hereditary. Science has taught us that hereditary infection hardly ever occurs, while *infection in the family* is occurring many times a day in every large city. In these crowded homes, the poor consumptive, ignorant and careless, may spit everywhere, and thus easily infect children crawling about the floor ; but, even if he has learned to be careful with his massive sputum, he cannot help coughing, and with each cough he may project into the air a regular fusillade of invisible infectious particles, which may reach the lungs of the healthy, and there sow the seeds of a fresh case of the disease. It is my experience that wherever an advanced case of consumption exists in a family, where no precautions are taken, another case of the disease in an early stage, or two cases or three cases in various stages, may be discovered by a careful search among other members of the family with the new methods of examination which recent science has placed at our disposal. Pathetic it is, but none the less true, that the very bonds of love and natural affection give this treacherous disease the best opportunity of showing its malignity. The husband infects the wife, the wife the husband ; the mother infects the daughter, and the daughter the mother ; and so on. The more intimate the ties of domestic life, the greater the risk, unless proper precautions are adopted from the very outset. It is true that the danger is potential rather than actual. In the crowded homes of the poor the danger is always imminent. Overcrowding through want of space, and uncleanly

habits, concentrate and intensify the risk of infection, while want of sunlight and want of fresh air keep the infectious material alive and potent for months and even years. No doubt, in the well-ordered homes of the well-to-do, with wise medical supervision, and the constant services of a trained nurse in advanced cases, the risk of infection may be reduced to a variable minimum, but exists nevertheless. The infection may have spread before the doctor has been consulted, or before the doctor has enforced all the precautions necessary to reduce the risk to a minimum. I have seen instances in which infection has spread widely in well-regulated families. The best medical supervision may fail; the trained nurse cannot follow every invisible particle of infectious material. If, then, infection often occurs in well-to-do families, we may safely argue that, in the homes of the poor, infection of the healthy is the rule. Let me repeat that, *according to my experience and observations, if there is a case of advanced tuberculosis of the lungs in a poor home, it is the rule that one or more cases of the disease in earlier stages will be discovered by a careful and systematic examination of the other members of the family.* This serious and well-considered statement, based on actual experience, is the keynote to my urgent advocacy of the City Dispensary as an indispensable and invaluable factor, both in the treatment and prevention of consumption. If we have a Dispensary for the treatment even of late cases, we shall have firm hold of the golden thread that will lead to the discovery of the early cases.

Thus the City Dispensary, if properly organised and designed to assist in every possible way the poor consumptive in all stages of the disease, not only ensures a satisfactory automatic system of notification, but also leads to the discovery of many cases in an early stage of the disease, which can be restored to more or less perfect health by proper treatment. Such a Dispensary serves two great purposes—the discovery of the primary sources of infection, and the treatment of the disease at a stage when cure is possible. Yet, although the Dispensary has this twofold purpose, its methods are various and manifold.

Before we consider the equipment necessary for a Dispensary, let us pass in review the main objects of such an institution.

1. The first object is the *treatment of the consumptive*, by which the sources of infection are automatically discovered. The sources of infection being thus discovered, the real work of the Dispensary begins in a systematic attempt to prevent further infection. This work embraces—

2. *Instruction of the patient and his immediate relatives* as to the nature and origin of consumption, and the best means of avoiding and preventing infection, by printed cards, etc., explaining and giving explicit directions. The value of these written instructions would be greatly enhanced by practical lessons given in the homes of the poor by a trained nurse, *viva voce*.

3. *The services of a trained nurse* to attend in their own homes those who cannot be taken to a Sanatorium or Hospital.

4. *Inspection of the home and disinfection of the rooms*, especially those occupied by the patient, and disinfection also of the clothes and bed-clothes of the patient. Some Dispensaries make a special feature of washing the clothes of the family regularly.

5. *Supply of medicines, disinfectants, and, not the least important, of food* (milk and butter) and other necessaries for invalids (sputum flasks, etc.).

6. *Examination of the other members of the family for evidence of fresh infection.*—Such early cases are the cases *par excellence* for treatment on modern lines with specific measures, notably Tuberculin and the rational methods of the Sanatorium. It is anything but easy to find these early cases except through the help of advanced cases ; and until some such system is introduced, suitable cases will not reach the Sanatorium. Thus the City Dispensary becomes an invaluable factor in the success of Sanatorium treatment, because, through the Dispensary, suitable cases are readily discovered.

Needless to say, the Dispensary should be situated in the densely-populated part of the city, and especially near factories. It will be well to consider the various functions of the institution in some detail, in order to understand the requirements and equipment of such an institution.

1. *Treatment.*—Treatment of the patient, which leads to the discovery of the sources of infection, must be a primary function of the Dispensary, because it is safer and better to appeal to self-interest than to intelligence. Patients in all stages of the disease will come to the Dispensary, and classification of the disease in its stages will begin at once, and treatment adopted accordingly. Some cases can be treated at the Dispensary ; others may be sent to Sanatoria ; while advanced cases of a highly infectious character should be sent to special Hospitals. It is the plain duty of the State Government to provide such Hospitals, where these advanced cases can be properly treated, fed, and nursed. Thus segregation of the infectious cases is ensured, and the public health safeguarded. Both Sir John See and our new Premier heartily approve of this proposal. The methods of treatment of the early cases should be up-to-date, and if I have any voice in the selection of methods, I shall strongly advocate that method of treatment which I have proved to my own satisfaction, after a long, laborious, and patient experience, to be of very great value in suitable cases. *If I have my way, I shall make such a Dispensary a medium for demonstrating the value of Tuberculin treatment, and a school for instruction in the details of the method.* Nor will the value of Sanatorium methods be lost sight of. Nothing should be left undone that at a reasonable cost might relieve the patient's symptoms or favourably influence the course of the disease. The Dispensary should consist at least of a waiting-room, a consulting-room, and a small laboratory. In

some Dispensaries a disinfecting chamber and a wash-house are added; in others a kitchen and store for food, especially milk. The supply of food and milk attracts, and the main object is to attract the poor people who suffer from consumption. For such work, the Dispensary staff should consist of at least three or four physicians—a senior or two, and two or three junior physicians, and our young graduates would there find a very useful field of activity. A caretaker, who might also help in the laboratory, would be necessary.

There is every reason to anticipate that many an artisan in the early stages of the disease, who has to work for his family and is still in relatively good health, might have regular treatment through the Dispensary without losing his daily wage. The Dispensary offers much to the poor man and woman who cannot afford to lose the daily wage. Such treatment for four or five months at a Dispensary need not cost more than a few pounds. In a Sanatorium, the cost would be at least £30, and the patient would also have to sacrifice his daily earnings. As I have completely arrested the disease in several cases without any loss of work or wage, and without any exile from home, it is surely worth while giving the system a trial for the sake of the poor man who must work to save his family from misery and starvation. Such treatment often needs a generous supply of milk, butter, and other foods, but even so, the total cost of treatment for five or six months need not exceed £10; and let it not be forgotten that sometimes the patient may still continue to work, if it does not involve severe strain. Thus there is a high economic advantage in this method, which fails in the Sanatorium. I believe that, after treatment with Tuberculin, even better results will be obtained by a month in a Sanatorium than by six months without Tuberculin treatment. Even in the best-regulated People's Sanatorium, though the cases are carefully selected, failures occur in 50 per cent. of the cases. In our present haphazard system, failures are far higher. It has yet to be shown to what extent Dispensary treatment (including the use of Tuberculin) can arrest the disease, and, as this method of mine is the most economical of all, the experiment should certainly be made, and should have a fair trial before it is decried or condemned. To make such a trial on economical lines, one needs at least £500 for one year's work. I have strongly advocated a trial of the system by the City Council in the cause of public health, but my plan was stale-mated by the technical objections of the City Solicitor, although the City Aldermen as a body approved of my idea, and regretted they had not legal power to make the trial. It is devoutly to be hoped that a new Municipality of Sydney Act will be passed, which will give to the Council the power to adopt rational and economical means, such as a Dispensary, for checking the spread of consumption in the City of Sydney.

Meanwhile, I am not disposed to wait till Parliament gives the Council these legitimate powers. The poor are the special victims of this cruel and devastating disease, and their sufferings have been tolerated all too long.

This address of mine is the first step in this great and urgent crusade. Who will help? Who may be expected to help? One generous Alderman, who is convinced of the practicability of my proposal at a reasonable cost, has already promised me £50. May we not appeal to the Insurance Societies to help the crusade? Insurance Societies know by the daily lesson of heavy money losses how much they suffer financially from the ravages of consumption among their own clients, and should in self-protection help forward any effective and economical method of checking the spread of this disease. Certainly Insurance Societies should contribute more liberally to the support of Dispensaries than to the support of Sanatoria, though both are worthy of support, because the grand object of the Dispensary is to prevent, of the Sanatorium to cure, consumption. If thousands of pounds can be subscribed by the public to the support of Sanatoria, at least we have a right to expect that hundreds will be forthcoming for a scheme which will not only increase and concentrate the usefulness of Sanatoria, but will also in itself, perhaps, do a much greater public service than Sanatoria can ever do, in preventing the spread of consumption by dealing directly with the sources of infection in the homes of the poor.

Treatment at the Dispensary is but a means to a great end—is but the necessary stepping-stone to the higher purpose of prevention. The establishment of the Dispensary for the purpose of treatment gives us the key to the solution of the otherwise difficult problem of notification as a means of prevention. The special machinery for prevention embraces (1) instruction of the patient and his relatives, (2) inspection and disinfection of the infected homes and their contents, and (3), last but not least, the examination of those who have long been exposed to infection, with the great object of preventing such cases passing into a hopeless stage, and becoming fresh centres of infection.

Let us now briefly consider the means to the great end of preventing consumption.

1. *Means of Instruction.*—(*a*) Printed leaflets will be given to the patient and his relatives, explaining the nature of the disease and the best means of dealing with infectious sputum and articles—advocating the use of rags or paper, which can be burnt at once, instead of handkerchiefs or nothing at all (rags and paper for this purpose can be gratuitously provided at a nominal cost), emphasising the importance of covering the mouth always during the act of coughing, etc.; explaining why the patient should always sleep by himself, and perhaps even have meals by himself. These and a hundred other practical lessons can be easily taught to the intelligent by the eye and ear—by written instructions and oral directions; but their full force and meaning can only be instilled into human nature by dint of reiteration, example, and object-lessons. For these specific objects (*b*) the trained nurse becomes an invaluable and indispensable agent in dispensing the daily lessons upon which success essentially depends. Thus the trained nurse becomes an integral part of

the complex machinery, explaining directions by practical illustrations, and enforcing them on the spot. The nurse also helps much in selecting and preparing the patient's food, securing plenty of fresh air and sunlight, thus acting as a ministering angel to the afflicted family. For many years I have been advocating the cause of the District Nursing Association, which provides trained nurses for the sick poor in their own homes, because I have long recognised that their services in teaching the poor how to avoid consumption must be of incalculable benefit. These nurses would readily co-operate with the Dispensary in this splendid mission to the poor of our own city. Instructions must be forcibly stamped into the minds of the careless and ignorant, else they make no impression, and the trained nurse is the one best fitted to do this, through the mother of the family, upon whom the whole household economy rests.

2. *Inspection of the home, and disinfection.*—Already the City Council provides the machinery for this work. To secure the good offices of the Disinfecting Department at the Town Hall, it might be well to have forms, such as—"I wish the Council to disinfect rooms in my house," to be signed by the householder. Let me add that in some Dispensaries provision is made for washing the clothes of those who are infected, and even of those exposed to infection in the family. Washerwomen are often infected through clothes, and the mother of the family, who does all the washing in the house, may become infected by washing the clothes of one suffering from consumption. Inspection by a medical officer of health or other doctor might be made at least once in three months, or more often.

3. *Supply of food, etc.*—The Dispensary should supply sputum flasks and disinfectants (Lysol) with directions for use. It adds greatly to the efficiency of the system if food is given, especially to the less advanced cases. This has the effect of encouraging the sick to come to the Dispensary, even in the early stages of the disease. Bread, milk, butter, eggs, soups, even meat, can be dispensed; clothes also—Government blankets, and the left-off clothes of the well-to-do. Everything should be done to make the Dispensary attractive, and much will depend on the tact and sympathy of those who control and work the machinery. Provision for washing the clothes acts in this way, provided that the clothes are not spoilt by washing or the use of disinfectants. Formalin as a disinfectant for this purpose has decided advantages of its own.

3. *Examination of the other members of the family.*—Of all the functions of the Dispensary, I am inclined to give the first place to the careful inspection, and even examination by special methods, of the other members of the infected household by an experienced and well-trained physician. Tact, above everything, is necessary for this delicate and responsible work. The physician must first win the confidence of the family. His aim is to detect disease in the early stages, when there may

be no symptoms, or at most symptoms that may be overlooked or misinterpreted. Often nothing short of a scientific test will disclose an early stage of the disease, and this test is now adopted as a routine method in the diagnosis of these early cases in the chief public Sanatoria of Germany. I can assert, after many years' experience, that the test can do good, and nothing but good, in suitable cases. If there is no disease at all, there is no effect, good or bad ; if there is an early focus of disease, the test demonstrates its existence, and secures treatment for the patient at a stage when permanent recovery can be secured by suitable measures —surely a "consummation devoutly to be wished." Such cases, which can be discovered with certainty in no other way, are the cases *par excellence* for Sanatoria, and form the majority of the cases treated in the public Sanatoria of Germany, where the test is widely used. This test in itself secures a vantage-ground of pre-eminent utility. If Sanatorium treatment is too costly or too exacting for the bread-winners of families, these early cases can also be most successfully treated with Tuberculin, and that sometimes without loss of work or wage. No doubt, Tuberculin treatment, combined with Sanatorium treatment, is the ideal method at present, but, if both cannot be used, I firmly believe, as the result of a long, careful, and systematic investigation, that Tuberculin in early cases is a more efficient, less costly, and less irksome therapeutic agent than the Sanatorium.

To inaugurate the Dispensary system on a modest basis, and in the most economical way, it would be necessary to find a suitable house of a few rooms in one of the poorest districts of the city—scrupulous cleanliness is a *sine quâ non*—as an object-lesson. There should be printed directions in prominent places, and receptacles, containing disinfectants, for sputum. Such a house could be rented for £70 a year. The furniture would cost another £30 (exclusive of instruments and scientific methods of disinfecting).

Sputum flasks, etc., antiseptics ..	£30
Medicines, etc.	£70
Caretaker	£150

On this rigid estimate the minimum cost would be £350 a year ; but one must allow a fund which can be increased or diminished at will, for the supply of food, clothes, bedding, blankets—say, £150–250. Thus for £500 or £600 a year a Dispensary could be established, and surely such a magnificent experiment should be made on behalf of the poor victims of consumption. Professor Calmette, from his own experience, says that in France an average maintenance of 120 families every day, including the washing of the clothes of 53 families, should not exceed £1,025. Think what can be done for £1,000! Would that one could find in Sydney a philanthropist who would be ready to venture £1,000 on such a magnificent experiment on behalf of 120 families already in the meshes of this deadly enemy of the human race!

What has been done can be done, and I doubt not that under this

Dispensary system, combined with specific treatment, the results might approach those of the best Sanatorium—would certainly equal the results of our own Sanatorium at the present time, because suitable cases would be selected for treatment. Moreover, a Dispensary for the same outlay would be able to reach four or five times as many cases as the Sanatorium. The Dispensary puts it in our power to treat the disease under more favourable circumstances, because many cases in a very early stage would be discovered. The important *rôle* of the City Dispensary in the crusade against consumption has been recognised in many countries—in France, Belgium, Italy, Holland, Spain, Portugal, Russia, in South American Republics, Cuba, and recently in Germany, the home of Sanatorium treatment for the poor. This last fact shows clearly that the Germans find that Sanatoria by themselves are not equal to the task of grappling with this immense and many-sided problem. But, even granting that Sanatoria (leaving cost out of the question) can do much for early cases, it is utterly hopeless to expect to bring the benefits of Sanatorium treatment within the reach of all our poor brethren. Even in Germany, where millions of pounds are spent among the artisan classes to insure against sickness, not more than 30,000 can have the benefit of Sanatorium treatment. It is estimated that in Germany nearly a quarter of a million are suffering from this disease, of whom the vast majority belong to the proletariat. Thus, at a cost of millions, the benefit is for but 30,000, while hundreds of thousands are left unheeded and untouched by the Sanatorium system.

The prevalence of consumption in the poorer classes of Sydney, as of every other city, loudly calls for the establishment of a City Dispensary for consumption, in the interests of the poor themselves, but also in the interests of the whole community. I trust that I have awakened your interest in this great problem. I think our next step should be to call a public meeting to consider my proposal, and I will be bold enough to ask the Lady Mayoress to enlist the sympathy and assistance of the Lord Mayor himself, who has always been ready to identify himself with good objects. I believe that a public meeting would soon secure a sound financial basis for this undertaking, and we should be able to establish a City Dispensary in Sydney without further delay.

Let me, in conclusion, remind you of Horace's famous epigram, "Non omnis moriar," for truly it may be said of all who come forward to inaugurate and maintain the Dispensary system, that, long after we are dead and gone, there will still live to our everlasting credit a rich harvest of lives which but for our efforts would have been sacrificed to appease the insatiable appetite of this invisible, treacherous, and malignant enemy of mankind. (Loud applause.)

Mrs. A. N. JONSEN said: I have very much pleasure in proposing a vote of thanks to Dr. Wilkinson. I would like to add that I for one can testify to the great sorrow that there has been amongst the poor, and is still, owing to this treacherous disease. Only yesterday morning, I was called

out at the Glebe to a woman only forty-five years of age who has had this disease for some three years, and she has two little boys, who are unable to help her very much. She is now dying, and I can assure you it was very heartrending to me to see this case. Those ladies who work amongst the poor of the various districts of the city and suburbs will know that it would be the poorer classes who would use this Dispensary, and we should have the satisfaction of knowing that something was being done, not only to stem this disease, but to help the poorer people of the districts. (Applause.)

Miss CRISPIN seconded the vote of thanks, which was put by the Lady Mayoress, and carried unanimously.

Dr. WILKINSON, in replying, said: I hope we shall not be satisfied with merely having our ideas on this subject, but that we shall do our best to put those ideas into practice, and try to introduce this scheme into the City of Sydney, which I believe will do more good than any other philanthropic movement at present existing. (Loud applause.) I thank you heartily for your vote of thanks to me.

A vote of thanks to the Lady Mayoress for presiding was, on the proposition of Mrs. Littlejohn seconded by Mrs. Goldschmidt, carried unanimously.

Miss VALENTINE said: Dr. Wilkinson was one of the first professional men in Sydney to help the Ladies' Sanitary Association by lecturing. One of the very first course of lectures was given by him at the anniversary of the Society, and another very fine lecture was given by Dr. Wilkinson on the plague. (Applause.)

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