which have been proved by investigation, and if found to bear such crucial test are assimilated with medical science, but, if weighed and found wanting, are rejected. I take it that in no country are the names and doctrines of great names so worshipped as in Germany; but I must maintain that no country is so practical as England.

I will venture briefly to recapitulate a summary of the views of the prominent speakers on that occasion, in so far as they were not contradicted. It was recognised that under whatever name, tubercle or otherwise, it might pass, that a product of known anatomical appearances was found in the lung in phthisis; that caseation is not invariably tubercle, but often results from inflammatory products; that a new growth takes place in the walls of the alveoli in all cases of phthisis, leading to destruction of their nutrient vessels; that this does not take place in ordinary acute pneumonia; that caseation was not due solely to inspissation of inflammatory products, but to a new growth in the walls of the alveoli, with which destructive changes in the lung are associated almost uniformly; that if this growth dies quickly, a rapid caseous change takes place, but if slowly, a fibroid change, leading to the well-known alterations in the lung structure that bear that name; that all these changes are manifestations of the disease, and that all occur in the most typical form of acute tuberculosis; that tubercle may occur without inflammation of surrounding tissues, or that it may be secondary to inflammation.

Dr. Wilson Fox states that implication of the alveolar wall is the most constant and typical appearance in the process of tuberculation. As regards the dispute about inflammation and tubercle, he considers that both may arise simultaneously, and that inflammation may give rise to secondary growth in the alveolar walls, which is a main cause of phthisis, and that the causes of tubercle may be the causes of inflammation. As regards the nature of the so-called tubercle, he is borne out by Dr. Burdon Sanderson, in considering it to be a lymphatic overgrowth, produced by irritation under special circumstances, anatomical or constitutional, and he believes that adenoid or lymphatic tissue is found in the walls of the air-vesicles, and that as lymphatic irritation may prevail in any part of that system, so it may show itself here. The boundary line between tubercle and inflammation cannot be accurately defined, but there is no doubt that the tubercular growth precedes the caseation. We come now to another element in the case, and that is that certain constitutional conditions are essential to the production of tubercle. Any irritation of the tissue may, in the presence of local or constitutional predisposition, give rise to secondary growths, diffused or circumscribed, which constitute tubercle, and which may be the source of further infection, and that with or without antecedent caseation, although this stage, and that of softening, appear to be most favourable to the change. The real agent in infection is the small-celled growth in the walls of the alveoli.

Again, attention was called to the fact that irritants introduced under the skin of the rodentia give rise to lymphatic deposits in the lungs, liver, and spleen; even the irritation of a seton has done so. It was considered that overcrowded corpusculum in any organ may give rise to similar processes elsewhere, but Dr. Moxon thought that there was no phthisis without caseation, and that tubercle was another phase of inflammation, which latter can cause caseation, and so enter the system. I believe it may be stated with truth that no speaker upheld that common catarrh can originate caseation, and in this view it was pointed out that the initial stage of phthisis is local and not diffused, as catarrh. Finally, it may be said Dr. Wilson Fox spoke the sentiments of the majority of those present when he said, "Tubercle tends to multiply, but can it be produced in the human subject by indifferent caseous products or by any inflammatory change not associated with a peculiar liability of constitution? I strongly doubt both."

It will be seen, Sir, that the tone of this debate was broad as regards the foreign schools, but accepted the data of neither. You will have noticed also that the whole debate turned both on the nature of the product in the lung and the changes which it undergoes, but also very much on the part of the lung which was so injured. If the doctrine is no caseation or no tubercle, then no phthisis, it is also no less strongly that if there be no new cell-formation crushing and destroying the walls of the alveoli, then also there is no phthisis. The recoverable cases are those which after inflammatory or tubercular or catarrhal attacks remain with the alveolar walls intact. Destruction of alveoli is never recovered from,

there are collapse and thickening of that part of the lung, but there is no restitution of structure and no vesicular breathing in that part again. Localisation of disease rises into great importance. We used to think that the consumptive died of a constitutional irritative fever, but, tracing their symptoms alongside of the physical signs, we now know that their fever and their waste are coincident, and that it is just then that the tissues around the alveoli and in the peribronchial spaces are filled with a new cell-growth, which rapidly proceeds to caseation and softening. We also know that at this very time a mass of detritus of inflammatory or tubercular products is being carried into the blood and lymphatics, and deposited in other parts of the system, and that high fever wastes the patient while other organs or the opposite lung is being infected. Whatever initiates the disease this secondary result seems as manifest as the spreading of a fire in a dwelling, while, like the latter, the mystery remains of the originating causes of the combustion.

ABSTRACT OF THE REPORT ON THE RELATION OF MICRO- ORGANISMS TO TUBERCULOSIS.

*Presented to the Association for the Advancement of
Medicine by Research on Feb. 1st, 1883.*

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A VISIT was paid to Professor Toussaint of Toulouse, and to Dr. Koch at Berlin, with a view of seeing their methods of experimentation, and the results which they had obtained. Various experiments were seen, and a quantity of material was brought back to England for more detailed examination. The result of the visits, and a full account of the observations made, will be found in the complete Report, which will be published in the April number of the *Practitioner*.

It was thought advisable in the first instance to repeat some of the experiments which have led observers, more especially in this country, to object to the view of the specific origin of tuberculosis, and to hold that in rodents, at least, any irritation might produce that disease. The present series of experiments were performed under the best hygienic conditions, with complete isolation of the animals from each other, and with thorough disinfection of the instruments employed. In six cases setons of various kinds were introduced both subcutaneously and into the anterior chamber of the eye; in ten, vaccine lymph both from the calf and from man was employed; in three pyæmic pus was injected (1) into the eye, (2) subcutaneously, and (3) into the abdominal cavity; and in six various materials (cork, tubercle hardened in alcohol, and worsted thread) were introduced into the abdominal cavity. None of these twenty-five animals became tuberculous. Some experiments are also cited in the report in which wounds in rodents have been stitched up with cotton thread, and others in which abscesses have been produced in various ways, but in none of these cases did tuberculosis ensue. In explanation of the former results, it is pointed out that at the time the early experiments on this subject were made the communicability of tubercle by mediate contagion was not recognised, and, as the precautions necessary for thorough disinfection of instruments &c. had at that time not been made out, the channels for the introduction of specific micro-organisms were left unguarded.

Two tubes of serum containing micrococci were obtained from M. Toussaint, who holds that micrococci are the cause of the disease. Toussaint obtains the organisms by inoculation of flasks containing serum, or infusion of rabbit with the blood of tuberculous animals; and he has in some cases succeeded in producing tuberculosis by the injection of these cultivations into other animals. The material obtained from M. Toussaint was injected into three rabbits, two guinea-pigs, one cat, and one mouse, and of these seven animals six were under observation for a sufficient length of time for the development at least of local tuberculosis. In no instance did tuberculosis ensue. (In all the experiments detailed in this report inoculation was made into the anterior chamber of the eye, whenever this was practicable; syringes purified by heat were employed for the purpose.) Cultivations of these micrococci were also made, and injected into

nine rabbits and three guinea-pigs. Of these four rabbits and three guinea-pigs were under observation for a considerable time without the development of tuberculosis in any case. The total result is that thirteen animals were inoculated with the micrococci with which Toussaint works, and obtained from Toussaint himself, and in no case did tuberculosis occur.

A number of tuberculous organs from animals experimented on by M. Toussaint were also obtained, some of the animals having become tuberculous after the injection of the micrococcal fluid. Careful examination of these organs has shown the presence, often in large numbers, of the tubercle bacillus described by Koch, but no micrococci have been found. The conclusion arrived at is that the micrococci described by Professor Toussaint are not the cause of tuberculosis. One of the possible explanations of the results which should not be left out of account is the following:—Professor Toussaint trusts greatly to carbolic acid as a disinfecting agent for the purification of the instruments employed in inoculation. This antiseptic, though effectual for the destruction of the ordinary forms of micro-organisms, as evidenced by the satisfactory results obtained from its use in aseptic surgery, has been shown to be ineffectual against the spores of bacilli, unless it acts for a long time. The bacillus of tubercle apparently produces spores, and there is no reason to suppose that these are less resistant than those of bacillus anthracis, and other bacilli. An experiment is given which shows that a saturated watery solution of carbolic acid, even though it acts as long as fifteen minutes, is not sufficient to arrest the development of the tubercle bacilli. Therefore, to wash a syringe with carbolic acid is not such a certain means of disinfection in this particular instance as was formerly supposed. Experiments were also made with cultivations of tubercle bacilli obtained from Dr. Koch. Twelve animals were inoculated with these organisms, chiefly into the anterior chamber of the eye, and all of them became tuberculous, and that more rapidly than after inoculation of tuberculous material. The tubercles produced in these cases were infective, and caused tuberculosis in other animals. On examination of tuberculous material, Koch's tubercle bacilli are always found, though in varying numbers. They are most numerous in bovine tuberculosis, and least numerous in human tuberculosis. About eighty organs of tuberculous animals and thirty-six cases of human tuberculosis were examined, and in all of these, without exception, tubercle bacilli were found. The inoculation of these bacilli is more certain and more rapid in its effect than the inoculation of tuberculous material from any source; and this seems only explicable on the supposition that in the cultivation of these bacilli the virus of the disease is present in a more or less pure state, and in large amount. Various facts are pointed out leading to the conclusion that in these bacilli we have the virus of the acute tuberculosis caused in the lower animals by the inoculation of tuberculous material.

In applying the facts obtained from experiments on animals to the pathology of tuberculous diseases in man, it is pointed out that all that has as yet been absolutely proved is that a variety of materials in man, which we class together as tuberculous, produce acute tuberculosis when inoculated into rabbits, guinea-pigs, and other animals, and that this result is due only to the tubercle bacilli present in the materials inoculated. It therefore remains for inquiry, what relation these bacilli bear to the morbid processes in man in which they are found.

Acute miliary tuberculosis in man resembles in every respect, in histological structure, in tendencies, and in the presence of bacilli, the disease produced in the lower animals by the inoculation of tuberculous material; and there can be little doubt that the cause of both diseases is the same—viz., the tubercle bacillus. It is, however, much more difficult to understand the relation of these organisms to localised tuberculous processes in man (phthisis, scrofulous diseases of glands, joints, &c.). Phthisis is alone considered in the present report, and, with a view of making clear the conception which the author has formed as to the relation of bacilli to this disease, the following facts are brought forward, which he has observed as to the mode of distribution of the organisms in the tissue, and their relation to its histological elements.

Two distinct structures have been described as tubercles in lungs of rodents—viz., nodules of lymphatic tissue in close proximity to the vessels and bronchi, and nodules which are largely made up of epithelioid cells. If a case of com-

mencing artificial tuberculosis be examined, it will be found that bacilli are only present in the latter nodules; indeed, it is rare even in the later stages to find them in the former, and in that case epithelioid cells will be found as well. The bacillus being the cause of this disease, the nodules containing epithelioid cells are alone tubercles. Further, on careful investigation of these nodules, it will be found that bacilli are only present in the epithelioid cells themselves. In making this statement only young tubercles and those in which the bacilli are present in moderate numbers are referred to. When there are enormous masses of bacilli, or where there has been confluence of tubercles forming a largish tuberculous deposit, some bacilli may be found in the outer part of the tubercle, but the great majority of them occupy the epithelioid tissue. When the bacilli are few in number, one need only look for them in the epithelioid cells. Around the epithelioid cells the tissue becomes inflamed, and converted more or less completely into granulation tissue. As the tubercle becomes older the epithelioid cells at the centre undergo caseous degeneration, and in this case the bacilli are present in the caseous mass; but are often best seen at its margin, where epithelioid cells still exist, and they may also be found penetrating into the inflammatory tissue. The giant cells of tubercle can be distinctly traced as originating from epithelioid cells, especially from epithelioid cells containing bacilli. As to the origin of these epithelioid cells in the lung, the great majority are derived from the alveolar epithelium. The bacilli escape from the bloodvessels or lymphatics and pass into the alveolar epithelium, where they grow and cause multiplication of the epithelial cells until the alveolus becomes completely filled with them. In some instances, however, those cells are probably derived from the endothelium of blood and lymphatic vessels. In the case of the liver, the author thinks they are frequently developed from liver cells, for bacilli have been found in liver cells at the margin of commencing tubercles, and gradations in size and form can be traced between those liver cells and the epithelioid cells in the centre of the tubercle. The accumulation of the epithelium in the centre of the nodules leads to obliteration of the vessels around and to fusion of neighbouring nodules.

With regard to phthisis, the two extremes, the rapid phthisis or caseous pneumonia, and the chronic or fibroid phthisis, are considered. In the rapid phthisis the alveoli are distended with caseous material, or in parts where the process is less advanced with epithelioid cells. Surrounding these, the trabeculae are thickened, and converted into granulation tissue. Here the bacilli are found in moderate or considerable numbers in the caseous material and epithelioid cells which fill the alveoli. By-and-bye the walls of adjacent alveoli disappear, and thus irregular cavities are formed containing caseous material surrounded by epithelioid cells and inflammatory tissue. In this case the bacilli are most numerous, and sometimes in enormous masses at the free margin of the cheesy material, and they are also present, though not as a rule so numerous, in the epithelioid cells at the line of junction of the caseous mass with the surrounding tissue. In fibroid phthisis the bacilli are, as a rule, extremely few; but here and there if a cavity exists, or in the centre of a caseous mass, one may find them in considerable numbers. They may, though very rarely, be also found in the giant cells, which are generally pretty numerous among the fibrous tissue. As a rule, however, the bacilli are extremely few, but nevertheless, if a sufficient number of sections be carefully examined, a few will be found here and there at the margin of or in the caseous masses.

The foregoing facts seem to indicate that when the tubercle bacilli reach the alveolus of a lung which is in a suitable condition for their growth, they develop in the epithelial cells lining the alveolus. This alveolus becomes filled with cells, neighbouring alveoli become affected, and the same process goes on in them. The further result will depend on the number and rapidity of growth of the bacilli, and on whether the patient is a good soil for their development. If they develop well we have caseous pneumonia; if they grow slowly and with difficulty we have fibroid phthisis. In the former case the alveoli become early distended with epithelioid cells, inflammation of the walls of the alveoli ensues, the epithelioid cells soon undergo caseous degeneration, and the presence of the masses leads to atrophy or sloughing of the walls of the alveoli. Infection of neighbouring parts of the lungs occurs by continuity, and also by partial coughing up and re-inhalation of the bacilli into other parts of the lung. In this rapid phthisis fibrous

formation around the alveoli only takes place imperfectly, and the lung rapidly breaks down. In the case of fibroid phthisis the bacilli are few, and grow only with difficulty. Thus fibrous formation occurs extensively, and giant cells are entangled in the fibrous tissue. In parts, however, the process may be more rapid, and there cheesy masses are formed, which may lead to breaking down of the lungs and the formation of cavities.

In the report it is pointed out that on this view we have one explanation of the rarity of acute tuberculosis in connexion with phthisis, and of the presence of bacilli in sputum, even before physical signs are marked, while it is shown that this view is directly corroborated by the results obtained by Tappeiner in his inhalation experiments. Against the statement that phthisis is due to the tubercle bacilli might be urged the fact that the bacilli found in the lung after death are often very few in number. Among other facts brought forward with regard to this question, it is stated that extensive tuberculous processes may be found in animals, containing only few bacilli, and that in cases where bacilli alone were inoculated, and where it is certain that the bacillus was the only agent at work. With regard to the production of phthisis by the inhalation of dust of various kinds, it is pointed out that the foreign particles inhaled probably only prepare the lung for the reception of the bacilli; for in these cases also bacilli are found. It has often been urged that the milk of tuberculous cows is infective. This may be the case when the mammary glands become tuberculous, and the mode in which the bacilli might get into the milk was well illustrated by the appearances found in a tuberculous kidney. There not only were bacilli present in the tuberculous mass, but they were also found in large numbers in the epithelium of the kidney tubules and in the interior of these tubules, both in the vicinity of the mass and at some distance from it. The author has not yet investigated the subject of tuberculosis of the kidney, but from what he has seen he thinks it probable that the epithelium of the tubules is the favourite seat of the bacilli in the kidney, just as the alveolar epithelium is in the lung. In that case bacilli would be present in the urine, not merely when there were marked tuberculous masses in the kidney, but also where the disease was but slightly advanced. From analogy it is probable that the same is the case in the mammary glands, and bacilli might be present in the milk, even though the disease of the gland was not sufficiently advanced to be noticeable.

The staining solution employed was the Weigert-Ehrlich solution. The formula is: of a filtered saturated watery solution of anilin one hundred parts, of a saturated alcoholic solution of the basic anilin dye (methyl violet, gentian violet, fuchsin, &c.) eleven parts. Mix and filter. Rapid staining is obtained by warming the solution. The specimens are then decolourised by immersion in nitric acid (one part to two of water), and stained in a suitable contrast colour. Very delicate sections are apt to be injured by immersion in the nitric acid. In this case, after staining them in the Weigert-Ehrlich fuchsin solution, they may be washed in distilled water, immersed in alcohol for a moment, and then placed in the following contrast stain for one to two hours: distilled water 100 ccm., saturated alcoholic solution of methylene blue 20 ccm., and formic acid 10 min. Whenever it is possible, however, Ehrlich's original method is recommended, as being most rapid, most simple, and most satisfactory. By this method of staining, tubercle bacilli and leprosy bacilli remain red. Psorospermiae and the outer coat of some parasites also retain the red colour. Lichtheim has further stated that a micrococcus is frequently found in the fæces, which reacts in a similar manner to the tubercle bacillus.

ROYAL LONDON OPHTHALMIC HOSPITAL. — The annual meeting of the governors of this hospital was held on the 9th inst. From the report of the Committee of Management it appears that the total number of patients treated during the past year was 24,018. Operations for cataract had been performed in 303 cases, and with more than the usual success. The chairman, in moving the adoption of the report, paid a tribute to the memories of the late Mr. George Critchett, who was for thirty-four years a member of the surgical staff, and of Mr. R. W. Lyell, a junior member of the staff, both of whom had died during the previous twelve months. A vote of thanks to the medical staff brought the meeting to a close.

THE ETIOLOGY OF DIPHTHERIA, THE NATURE OF ITS CONTAGIUM, AND THE PHENOMENON OF SUDDEN DEATH OCCURRING IN IT, RECONSIDERED.

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THE views put forward in the following communication not being in accord with those generally received on the subjects of which they treat, may I venture to solicit for them the considerate attention of the reader on the following grounds? First, because, though briefly summarised, they represent the outcome of many years' careful observance of the disease, under circumstances not unfavourable to its study. Secondly, because I have brought to bear upon the solution of the problems at issue a principle new in its application to such questions. This principle I had the honour of submitting in detail to the medical section of the International Medical Congress held recently in London in a paper discussing the evidences which support it, as well as some of its bearings on the determination of the phenomena of disease. The principles upon which the chief points attempted to be established in this communication rest may be briefly stated as follows:—1. The afferent fasciculi of a given sympathetic ganglion (which are usually found accompanying the sensori motor nerves, being contained within their sheaths) are in reflex relationship with the efferent vaso-motor nerves furnished to the arteries from the same ganglion. 2. That the medium of this reflex relationship is the particular ganglionic centre with which the afferent and efferent fibrillæ alike communicate. 3. That afferent impulses transmitted through a sympathetic ganglion take effect upon the walls of the bloodvessels supplied by it with vaso-motor nerves, and by enlarging or contracting the calibre of these vessels, produce alterations of nutrition, or of function, in the areas to which the vessels so innervated are distributed. 4. That in this way the sympathetic ganglia constitute centres for correlating widely separate tracts of tissue, bringing them into mutual interdependence in regard of such operations as come within the sphere of their functions. 5. By means of this correlating faculty the sympathetic ganglia are found to be largely instrumental in determining the localisation of morbid symptoms. It is to this coördinating function of the sympathetic centres that I appeal in attempting to elucidate those aspects of diphtheria discussed in the present paper. One of my earliest experiences of diphtheria occurred about the year 1860, just after the appearance of Bretonneau's second memoir. The patient, a middle-aged female, was treated with calomel, as recommended by this author, though to a much less extent than practised by him, and recovered. The case, though well characterised, was not, comparatively speaking, a severe one. During the subsequent fifteen years over which my experience of the locality where this case occurred extended, it was repeatedly visited by the disease. During the latter part of this period I was afforded, in the capacity of medical officer of health for the district, every opportunity of investigating the conditions under which it made its appearance. Two conclusions respecting the causation of diphtheria gradually took shape in my mind, as year after year it presented itself under very varying circumstances.

The first was, that while I was never able to connect an outbreak of the disease, either in its single or multiple forms, with anything of a distinctly zymotic nature—understanding by this term some subtle poison introduced from without,—yet I was nearly always able to connect the cases with some precedent climatic condition or state of the weather. These antecedent conditions were frequently quite dissimilar, comprising periods of intense cold, prevalence of very high summer temperature, long prevailing east wind, or a continuance of cold and wet. It was after such states of atmospherical environment, and not during them, that the disease made its appearance.

Secondly, I observed a very decided proclivity on the part of certain families to take the disease. By this I mean, there were some families in which, when the disease occurred, it usually ended fatally as regards the first subject