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TUBERCLE.

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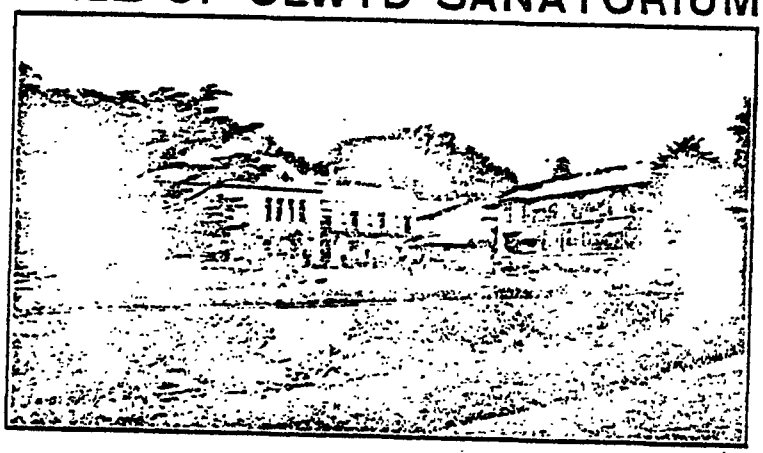
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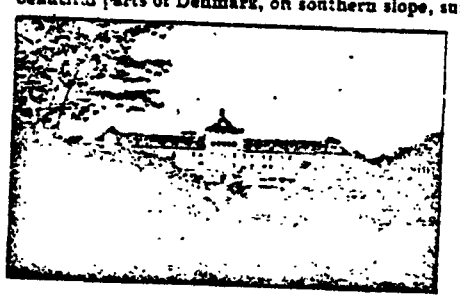
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A PLEA FOR THE RE-ORGANISATION OF THE TUBERCULOSIS SERVICE.

By W. Stobie, M.D., M.R.C.P.,

Honorary Assistant Physician, Osler Pavilion for Tuberculosis, Radcliffe Infirmary and County Hospital, Oxford.

In 1910 the Oxfordshire Association for the prevention of tuberculosis was founded under the presidency of the late Sir William Osler.

The present writer was fortunate enough to be associated with the great master of medicine as the first medical officer of the Association. The work of that voluntary body was principally concerned with the problem of tuberculosis in rural areas. Under its auspices the first country dispensary system was inaugurated. On this system the Astor report for a unified scheme throughout the country was largely based, in anticipation of the National Health Insurance Act of 1912.

The Oxfordshire scheme was itself fashioned after the Edinburgh plan of Sir Robert Philip, modified naturally to meet local circumstances in a scattered agricultural district.

Because of the above facts the writer has no qualms in making certain criticisms of a scheme at whose birth he was an interested spectator.

And firstly, in the writer's opinion, one of the great weaknesses of the present arrangement for dealing with tuberculosis is the dissociation of the general hospitals from the work. As a result of this severance the general practitioner, and still more the embryo family physician, have few or no opportunities of seeing the multifarious manifestations of what is even yet one of the principal fatal diseases. Pulmonary affections form a not inconsiderable part of the average medical practice, and, except in a few notable instances in the larger centres, there is very little chance for the medical student to study tuberculous disease in its early stage. His actual knowledge of pulmonary tuberculosis is confined, too often, to the stage of cavity formation met with at that phase of his career after which, with luck, he may find himself a fully qualified medical man. At the same time his ideas of treatment of the disease—with which many of his examiners are readily satisfied—remain as in the days immediately following Koch's discovery of the bacillus, namely, "open air, good food, rest in bed and the treatment of symptoms as they arise." And perhaps with a view of creating a really favourable impression the fine-sounding phrase "graduated exercises" might be added. It is truly surprising how few recently qualified men can carry out a simple procedure like artificial pneumothorax. Owing to the lack of opportunities for receiving tuition in the diagnosis and treatment of pulmonary tuberculosis many have not even seen it performed.

If that is the lot of the medical student, what of the disadvantages of this estrangement from the general hospital of the tuberculosis officer?

S. Roodhouse Gloyne, The Presence of the Asbestos Fibre in the Lesions of Asbestos Workers (1929)
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Left as he is to his own devices, he tends, not unnaturally, to sweep into the embrace of his special subject all manner of complaints of a totally different origin. Not only does this give an altogether wrong conception of the amount of tuberculosis in a district, but, perhaps even worse than that, affixes the label of "consumption" to patients who, quite possibly, are already severely enough handicapped in other ways.

Moreover, the treatment of the non-tuberculous condition is, as a rule, not only completely different, but infinitely less expensive. No one of us is so omniscient as not to be the better for a colleague's opinion. And all of us are the wiser, if perhaps the humbler, for the unsentimental demonstration of the pathologist.

Another grave objection to present methods is the attempted combination in one man of various functions. That any local authority can honestly believe that the responsible duties of assistant medical officer of health, assistant school medical officer and assistant tuberculosis officer, can be efficiently carried out by one individual is unlikely. The real truth is that it is an economical (*sic*) arrangement, and one convenient to the medical officer of health. Unfortunately for the patients suffering from phthisis, the line of advancement of tuberculosis officers, in the past, has been towards the position of a public health official.

This state of affairs is comparable to the evolutionary process of the medical officer in the army medical service. And, as in that case, the path of resistance is not made less difficult to the naturally keen clinician by the financial advantages on the administrative side.

It has never been clear to the writer why that class of the community grouped for convenience as insurance or panel patients should have to be content, in so many cases, with the opinion of the medical officer of health or quasi-medical officer of health on one of the most difficult problems in medicine. How many medical men are there who would send a member of their own family to consult their local medical officer of health for an opinion on a chest ailment? How can the assistant medical officer of health (plus assistant school medical officer, plus assistant tuberculosis officer) find time to keep in touch with any of the recent literature on tuberculosis? How can he have any practical knowledge of modern methods in diagnosis and treatment, as, for example, the use of lipiodol, phrenic avulsion, thoracoplasty, and so on?

The era of distribution of cod-liver oil (with or without malt extract, together with an extra pint of milk per day) is over, and has given way to an age of progressive measures requiring the knowledge and experience of skilled physicians and surgeons.

In how many tuberculosis dispensaries as at present constituted do facilities exist for ordinary routine investigations, such as examination of the fæces or guinea-pig inoculation?

And in how many sanatoriums are operative facilities available for collapse-therapy—with the possible exception of artificial pneumothorax? Some sanatoriums—so the writer has been informed—have not even an X-ray plant!

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Under the new Local Government Act there will be great opportunities for the co-ordination of forces against the scourge of tuberculosis. Foremost among these, in the writer's opinion, are:—

(1) The attachment of the tuberculosis officer (who should be a clinician and not a jack of all trades) to hospitals where he will be in touch with a group of colleagues interested in various branches of medicine and surgery.

(2) The appointment of regional consultants—medical, surgical and pathological—in tuberculosis. For these posts men and women of wide general training attached to teaching hospitals will be required.

(3) The affiliation in any scheme of special chest hospitals where the services of the physicians, surgeons and radiologists &c., of experience will be available.

By the employment of modern methods not only are the depressing end results of sanatorium treatment mitigated but the expensive stay is much curtailed.

The pivotal officer in all these services must be the medical officer of health. He will have plenty to do without attempting to engage in clinical work as well. The admirable medical service of the Ministry of Pensions would provide a good plan upon which to build. Such a service would, in the judgment of the writer, really get to grips with a problem which, despite the millions of pounds which have been spent upon it during recent years, has been brought very little nearer a solution. It would, moreover, offer much better prospects than exist at present to the younger members of the profession, and as a consequence would attract men with good medical and surgical qualifications.

The question of expense is bound to arise. In this connection the writer would plead with the administrative officer (the medical officer of health) to remember that he is not the financial adviser of his authority. His duty is to place before his council the considered opinion of modern medical science in respect of tuberculosis and other problems of a similar preventable nature. There are any number of self-styled economists on public bodies who will be ready enough to say they cannot afford this, that or the other method, especially if new in conception.

Anything more expensive than the old methods of dealing with tuberculosis can hardly be imagined. To take one instance only, the duration of stay in institutions can be cut down by at least one-half by modern methods of collapse-therapy. A comparatively easy arithmetical calculation at so much per week will in itself demonstrate considerable financial saving, to say nothing of the lessening of all the evils that a long waiting list entails.

The writer is well aware that the above criticisms could not fairly be levelled at certain areas where many of the reforms suggested are in actual being. If the forward policy of these comparatively few areas should, directly or indirectly, as a result of this short article be adopted in the vast remainder, then the purpose of the writer will have been amply fulfilled.

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THE PRESENCE OF THE ASBESTOS FIBRE IN THE LESIONS OF ASBESTOS WORKERS.

By S. Roodhouse Gloyne, M.D., D.P.H.,

Pathologist, City of London Hospital for Diseases of the Heart and Lungs, Victoria Park.

The use of asbestos as a trade product has produced a new variety of pulmonary fibrosis. The clinical and radiological features of this disease have been well described by Burton Wood [1] and the morbid anatomy by Cooke [2] and Stuart McDonald [3]. A short investigation of the condition has been made by the writer, as recorded below, with a view to detecting, if possible, the presence of asbestos in the body during life.

The Raw Asbestos Fibre.—The raw asbestos is found in various parts of the world, and differs somewhat in composition in different places. The variety chiefly used in this investigation has been blue fibrous asbestos known as Cape asbestos. This raw material when ground up in a mortar with a little water or saline solution, yields a fine bluish white, almost powdery, deposit. As the asbestos does not take up aniline dyes and does not show up well unstained, under the microscope, the dark-ground illumination technique was adopted for practically all examinations. The fibres stain faintly with both the Fontana silver and the prussian blue methods, but neither is adequate for purposes of identification. When examined by dark-ground illumination powdered asbestos is seen to consist of fibres of varying length mixed up with a varying amount of particulate matter of irregular shape and often of reddish colour, which presumably represents minute particles of ore. The asbestos fibres are straight and highly refractive, and they vary in length, according to the thoroughness with which the raw asbestos has been ground, from fibres extending across the whole field of the microscope to fibres the length of an anthrax bacillus or even shorter. In the raw asbestos the fibres are held together in thick straight bundles, but when ground up the large bundles are split into smaller and smaller bundles until eventually fine individual fibres are obtained. The broken-off ends of the bundle can also be seen like bundles of freshly cut reeds. No branching has been observed, but frequently small particles of ore will stick in the fibres, giving at first sight a very rough resemblance to a filament of mould with gonidia. When individual fibres are examined carefully they are often found to have an extremely fine saw-like edge rather suggestive of the marks on a freshly cut plank. This saw-like edge does not always extend the whole length of the fibre. Occasionally, the free edge of the fibre will vibrate like the end of a fine steel spring, whilst split ends and notches in the fibres may be found. The fibres show the ends broken off at varying angles, quite unlike the ends of bacilli or fungi generally. Indeed, the appearance of the preparation under dark-ground illumination gives the impression of sharp brittle metallic wire broken up at all sorts of angles and in all lengths. But the chief characteristic of a single asbestos fibre when viewed by this method is its marked refractivity. The whole fibre appears to be brightly illuminated like the filament in an electric bulb,

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and when very thin the fibre has a bluish luminosity, whereas bacteria, cells, crystals, &c., generally show only a refractive edge with the rest of the structure in darkness. Finally, the asbestos fibre will withstand the heat of a Bunsen flame and is not destroyed when placed in concentrated sulphuric acid.

To summarise, therefore, raw asbestos may be ground up into a fine powder, calcined in a crucible, and mounted on a slide as a wet preparation with concentrated sulphuric acid without showing any signs of destruction. When examined by means of the dark-ground illumination method with a $\frac{1}{12}$-in. objective, a film thus prepared shows bundles of fibres and individual fibres of varying lengths. The fibres are highly refractile; they are straight and rigid like steel wire and have often a very fine saw-like edge, whilst the ends are broken at different angles and occasionally split, and the finest of them have a bluish tinge.

With these appearances in mind attention was next directed to the finding of asbestos in persons exposed to the risks of the trade.

Asbestos Corns.—The first and most obvious site for the fibres is the "asbestos corns" in the skin to which the workers are liable. Fine asbestos fibres penetrate the superficial layer of the epidermis like thistle thorns and produce a minute corn of hypertrophied epidermis around them. These fine asbestos fibres may be so ubiquitous that they even penetrate the stockings of girl operatives and produce corns on the legs. The asbestos fibres can be readily removed from the corns with a fine needle and when examined by dark-ground illumination they show the characteristic appearances of the fibres noted above.

Asbestos Fibres in Upper Respiratory Tract and in Sputum.—The next step is the demonstration of the asbestos fibres in the upper respiratory tract and in the sputum. Here the task is not so simple, because it is essential to eliminate any foreign bodies which may resemble an asbestos fibre. Accordingly, nose swabs, mouth washes and sputa from asbestos workers were dealt with by the following technique:—

(1) Digestion with 10 per cent. antiformin solution (in normal saline) which was filtered through an L3 Chamberland candle before use in order to remove any minute foreign bodies.

(2) Centrifugation of the digested material.

(3) Calcining of centrifuged deposit in crucible to destroy all cellular debris, &c.

(4) Preparing a wet preparation of the calcined deposit in concentrated sulphuric acid (a control film of which should be examined to eliminate the possibility of foreign bodies in the acid) and examining with $\frac{1}{12}$-in. objective by dark-ground illumination.

By this means asbestos fibres were demonstrated in the mouth and nose of two operatives who had been at work in the factory within a few days of examination. The finding of the fibres in the sputum was more difficult. These two operatives had no sputum, and it was necessary to fall back on cases which for medical reasons had been out of the factory for some time. Sputum from six cases in all were examined. In three

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asbestos fibres were identified, in three none was found. It is advisable always to put up a control slide of raw asbestos for comparison.

Asbestos Fibres in the Lung.—Paraffin sections of the lung of a case of pulmonary asbestosis were cut and mounted on a slide in the usual way. The lung tissue was then deliberately destroyed by the addition of concentrated sulphuric acid and the acid in turn replaced by water. The section was then covered with a thin cover glass and examined by dark-ground illumination. Typical asbestos fibres were found. They were also present in the expressed juice of the lung in this case.

Asbestos Fibres in Animal Tissues.—Finally, as a confirmation of the method, a suspension of ground up asbestos fibres in sterile saline made from freshly re-distilled water was inoculated intraperitoneally into the guinea-pig. A lymphocytic response was produced in half an hour, and the asbestos fibres were readily demonstrated in wet films by means of dark-ground illumination.

A guinea-pig was also inoculated, subcutaneously, with a suspension of asbestos in saline solution. When killed twelve days later it was found that the asbestos was collected together in bluish-grey masses lying in the fascial planes between the muscles of the groin at the site of inoculation. These masses were surrounded with a small quantity of soft gelatinous adhesions but there was no sign of suppuration. Sections of these adhesions showed them to consist of granulation tissue densely impregnated with asbestos fibres. The fibres were lying in all directions, many of them being in small bundles like minute wisps of hay. No "curious bodies" were seen, but a few of the fibres appeared to be slightly bulbous at their extremities. The granulation tissue also contained large numbers of foreign body giant cells, many of which appeared to have phagocyted small asbestos fibres. These foreign body giant cells were a very prominent feature of all the sections.

The cellular reaction to asbestos in living tissues will be dealt with in a later communication.

The So-called "Curious Bodies."—The golden bodies occurring in the lungs of cases of pulmonary asbestosis have been described by Cooke, McDonald, Simson [4] and Stewart [5]. No "curious bodies" were found in raw asbestos or in asbestos corns, nose swabs, mouth washes or sputum of workers. They were found, however, post mortem, in great profusion in the lungs of the case referred to above. It occurred to the writer that they might be derived from the kieselguhr with which the asbestos is mixed at a certain stage of manufacture in some factories. Kieselguhr is a whitish or greyish powder which consists of fossil diatoms. These diatoms occur in many shapes—star-like, slipper-shaped, elongated, segmented forms, and so on, and a comparison between these and the "curious bodies" suggested that the latter might possibly be fossil diatoms from kieselguhr which had become shrunken and distorted by pressure and by the action of tissue juices in the lungs. This view proved on further investigation to be erroneous.

The point was settled by the following experiment. A wet preparation of "curious bodies" in expressed lung juice was placed on the stage of the

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dark-ground illumination microscope and the position of a readily recognisable group of these bodies noted. Concentrated sulphuric acid was then very gently run under the cover slip, the "curious bodies" being kept under observation all the time. Slowly these bodies began to dissolve and in the middle of their structure was a number of typical asbestos fibres. At the end of half-an-hour practically all the material of which the "curious bodies" were composed had disappeared, leaving only a faint ghost-like outline of the bodies with highly refractive asbestos fibres within them. There were several of these bodies in this particular group and the asbestos fibres lay at different angles within them. Dr. D. S. Page, who confirmed this observation with me, made the suggestion that along the length of the fibres and in the angles formed between them there is laid down the material—possibly blood-pigment—which ultimately gives to the "curious bodies" their characteristic shape. At all events, it seems clear that whatever other material enters into their composition their basic scaffolding is the asbestos fibre. The writer would therefore suggest that "asbestos bodies" would be a more accurate description of the appearances seen than "curious bodies."

Conclusions.

(1) Asbestos fibres can be demonstrated in the corns from which the workers suffer, in the upper respiratory tract, in sputum in the lung, post mortem, and in animal tissues.

(2) The so-called "curious bodies" are derived from asbestos fibres. It is suggested that asbestos bodies would be a better name for them than "curious bodies."

(3) In view of the marked prominence of foreign body giant cells in the sections from the guinea pig, it is important that tubercle bacilli should be demonstrated in sections of the human lung in cases of pulmonary asbestosis before assuming that tuberculosis is present as a complicating factor.

I am greatly indebted to Dr. W. Burton Wood, Dr. C. Leonard Williams, and Dr. W. L. Yell for kindly sending me cases and material for this study, and to Mr. A. W. Smart, Chief Laboratory Assistant, for practical help.

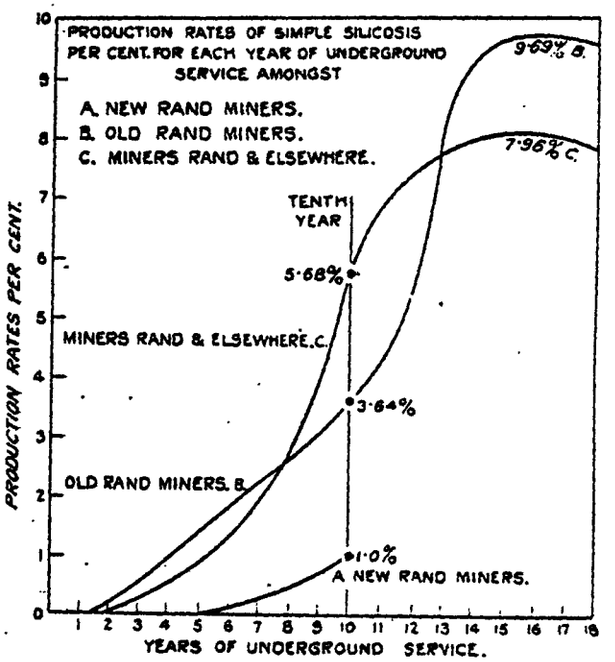
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TUBERCULOSIS STATISTICS.

MINERS' PHTHISIS ON THE RAND, SOUTH AFRICA.



Explanation.—A. New Rand Miners are men who have commenced underground work in the South African gold mines since 1916, and have all undergone a severe medical examination on engagement.

B. Old Rand Miners are men who were at work before 1916 and did not pass a medical examination on engagement.

C. Miners, Rand and Elsewhere, are men employed before 1916 who have also worked as miners elsewhere.

The cases recorded are those of simple silicosis detected at periodical medical examinations. The rates based on these cases have been smoothed out by the customary statistical method.

NOTE.—(1) The curves of incidence for both Old Rand Miners and the Miners Rand and Elsewhere begin early and rise steeply, but reach their maximum at about eighteen years of underground service.

(2) The curve for New Rand Miners begins later and rises much less steeply. It presents a striking and favourable difference. The length of service at which the maximum will be attained cannot yet be surmised.

Reproduced from—

"The Present Position of Miners' Phthisis on the Rand." L. G. Irvine, 1928.

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AMERICAN SECTION.

Under the Editorship of Dr. A. K. Krause.

ALLERGY IN CLINICAL TUBERCULOSIS.

By F. M. Pottenger, M.D.
Monrovia, California.

Introductory.

We have now reached a place in our understanding of tuberculosis in which we are able to interpret the disease and its manifestations more clearly than at any previous time. This position has been attained through diligent study of every phase of the subject. It has included careful clinical observation and much painstaking animal experimentation. The correlation of the data obtained in these fields has furnished a basis for writing the story of clinical tuberculosis anew and recognising the immunity reaction as a factor of prime importance in determining the pathology of the disease, its symptomatology, its diagnosis, its clinical course, and its healing or failure to heal. Without the consideration of allergy we can no more understand the pathologic changes and varied clinical manifestations in tuberculosis than we can understand its ætiology without considering the tubercle bacillus.

In the early years of extensive study of tuberculosis our opinions were so largely dominated by deadhouse pathology that we could not understand living tuberculosis. We failed to grasp what it is that is occurring, changing and repeating itself, which makes up the ensemble of chronic tuberculosis as we meet it day by day in the clinic.

However, through an ever-increasing accuracy in clinical observation, and a more perfect technique in physical examination, with examinations repeated at frequent intervals on the same patient, and these supplemented by X-ray films taken by the most approved technique and by laboratory studies, we have learned that clinical findings are to be interpreted, if they are to be understood, in terms of immunological response in which the allergic inflammatory reaction is to be kept to the fore. By correlating the results of these methods with the data obtained from immunological investigations we have learned that tuberculosis is a very different entity from that which was formerly believed. We can now visualise and record the ever-changing interplay between the invading bacilli and the host in a much more satisfactory manner than was possible before the nature of the body's specific resistance to reinoculations of bacilli was known. In fact, we are now realising that it is the immunity reactions which make up the disease picture.

The Koch Phenomenon.

The basis for this better understanding of tuberculosis has been within our grasp for nearly forty years. It was contained in one of Koch's early papers in which he announced the discovery of tuberculin: but, like most great basic truths, it has required a prodigious amount of thought and labour to clarify it and make it understandable to the great mass of workers.

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His observation was the first recognition of the fact that an animal, immune to tuberculosis, that is, one whose specific immunity mechanism has been aroused by a previous inoculation with tubercle bacilli, reacts differently toward living or dead bacilli from one infected for the first time. This reaction to reinoculation has come to be known as Koch's phenomenon. It was the first immunological approach to the study of tuberculosis, and has been made the basis not only of the modern immunological study of tuberculosis, but of the immunity reaction in other diseases as well.

Koch's description of the immune reaction is as follows [1] :—

"When one inoculates healthy guinea-pigs with a pure culture of tubercle bacilli the inoculation wound closes up and apparently heals within a few days. In the course of ten to fourteen days, however, a hard nodule appears, which breaks down and forms an ulcer. This remains until the death of the animal. It is quite different, however, when a guinea-pig that is already tuberculous is inoculated. Animals that have been inoculated from four to six weeks previously are best suited to this purpose. In one such animal, however, the inoculation did not heal in the beginning, but small nodules appeared, and within one or two days showed characteristic changes about the point of inoculation. Around the point of inoculation it became hard and took upon itself a darker colour, which did not confine itself to the immediate point of inoculation but spread to the surrounding tissues for a distance of from ½ to 1 cm. When the succeeding dose is administered it shows very distinctly that the skin showing the alteration is necrotic; and it is eventually cast off, a small superficial ulceration remaining, which usually heals quickly and permanently without the regional lymph-glands becoming involved. The inoculated tubercle bacilli also act very differently upon the skin of a healthy guinea-pig from what they do upon one infected with tuberculosis. This characteristic action is not entirely confined to living tubercle, but also occurs when dead bacilli are used, whether the bacilli be destroyed, as I attempted in the beginning, through low temperature of long duration, or by boiling, or through the action of chemicals.

"After these characteristic effects had been discovered, I followed them in every direction and I found that pure cultures of tubercle bacilli that had been killed, ground up, and mixed with water could be injected under the skin of healthy guinea-pigs in large amounts without producing anything beyond a local suppuration. Tuberculous guinea-pigs, on the contrary, were killed by the injection of very small amounts of such cultures of dead bacilli within from six to forty-eight hours after the dose had been administered. A dose that is not sufficiently large to kill an animal often produces an extensive necrosis of the skin in the neighbourhood of the site of injection. If the solution of dead cultures of bacilli is diluted still further so that it appears only slightly opalescent, then the animal remains alive; and if the injections are carried on with intervals of one or two days, a remarkable improvement in the condition of the animal takes place.

"The ulcerating inoculation wound becomes smaller and eventually forms a scar such as never occurs without such treatment. The swollen lymphatic glands become smaller, the nutrition of the animal improves

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and the diseased process becomes quiescent if it is not too far advanced and the animal does not go rapidly down to death as is usual.

"These observations furnished me a basis for producing a remedy against tuberculosis."

We owe much to von Pirquet's [2] explanation for the changed reaction of body cells toward bacilli and bacillary protein as we find it in the immune animal as compared with the primarily infected. He called the phenomenon met in the reinfected, "allergy," and carefully studied many phases of the reaction. In tuberculosis we speak of it simply as "allergy" or as "tuberculo-allergy."

What is Allergy?

If we undertake to define allergy we are at a loss to know how much of the body's reaction toward bacilli and bacillary protein to include in the term, and how much to exclude. Allergy is a result of a hypersensitiveness to bacillary protein and manifests itself in the cells throughout the body of the host whether they have come in immediate contact with the invading bacilli or not. Therefore it must depend, for its existence, upon bacillary products which are elaborated within the disease focus, and which wander out from it and are disseminated through the blood-stream to all body cells. We cannot separate allergy from hypersensitiveness nor can we divorce it from immunity. After the first implantation of tubercle bacilli has gotten well under way, these three reactions, as far as is now known, represent the most important factors in tissue defence.

Krause [3], whose studies on allergy in experimental tuberculosis are among the most important so far made, in discussing the subject of pathogenesis in a recent paper suggests that allergy is the body's reaction against tuberculo-protein while immunity is the reaction against bacilli, thus: "For our purpose immunity is regarded as that condition of increased specific resistance to implantation and extension of tubercle bacilli, and allergy as that state of tissue hypersensitiveness to tuberculo-protein that comes into existence with the earliest formation of specific anatomical tubercle. Both immunity and allergy continue to work within the body as long as tubercle, with its tubercle bacilli, persists."

Allergy is established by the primary inoculation. Its rapidity of appearance in experimental tuberculosis varies with the dosage of bacilli, coming on after a few days when relatively large quantities of bacilli cause the infection and after two or three weeks when few are responsible for it. We assume that the same holds for man.

Allergy is a varying force. It is inflammatory in character. The power to react allergically may be studied in animal experimentation. It may be increased many fold by repeated reinoculations. Roughly speaking, the larger the inoculation that the animal is able to overcome the higher will be his ability to react allergically. This subject has been studied and developed by many workers in many countries. Among the early workers in this field we would especially point out Römer, [4], [5], [6], [7], [8], [9], and Hamburger, [10], [11], [12], [13], [14], while among recent workers, no one has done more than Krause [15], [16], [17], [18], [19], [20], [21], [22], [23], [24], [25], [26], [27], [28], to give us an adequate understanding of this important subject.

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We are able to observe the building up of allergy following successfully withstood reinoculations in the course of clinical tuberculosis, either following many small reinfections unattended by recognisable symptoms, or one or more severe ones. From our knowledge of the incompetent wall which surrounds the tuberculous focus and its bacillary content during the time when the disease is active we are forced to assume that small reinoculations, unrecognisable though they may be, take place at frequent intervals; it may be minutes part; it may be hours. But that they take place without making their presence known by symptoms is not only a possibility but a probability. That these are followed by small, probably often microscopic, allergic reaction, and result in an ever-increasing efficiency in the workings of the immunity mechanism, and further that they stimulate the reticular elements to form new fibrous tissue and thus become potent forces in the healing of the disease is also probably true. Allergy is favoured not only by a suitable reinoculation but by a satisfactory structural and functional state of the cells on the part of the host.

Should an excessive dose of bacilli cause reinoculation, the body may not be able to respond with a competent immunity reaction, and so allergy may be depressed at least for the time, or it may even be caused to vanish. So may it disappear under stresses to which the body is subjected, such as those during pregnancy and in the presence of such disease as influenza, measles and scarlet fever. It may be depressed when the host is undergoing severe stresses of whatever kind.

The severity of the reaction of the body to reinoculations of tubercle bacilli depends to a large extent upon the degree of allergy present. The more marked the allergy the more severe the reaction. Allergy can be taken as a measure of immunity; for the resistance to bacillary invasion and the reaction of the body cells to circulating bacillary protein seem to go hand in hand. In the clinical course of tuberculosis I have learned that as a rule those patients who possess the greatest resistance to the disease show most marked local reactions to tuberculin.

The presence of allergy, as was pointed out by von Pirquet [29], hastens the body's reactions and also makes them more violent. It is a determining factor in the further course of the disease in every reinoculation that takes place in tuberculosis; in fact, it is such an intimate part of clinical tuberculosis that we cannot think of active tuberculosis apart from the allergic response. While allergy is primarily defensive it may at times become injurious by causing so severe a reaction that the host is either harmed or destroyed. This has been shown in animals where a fairly large dose of bacilli or of tuberculin is withstood by the healthy pig without any immediate reaction taking place, while the same dose of bacilli or tuberculin, injected into a tuberculous animal, may cause death within a few hours or days.

Koch [30] found that 20 to 30 mg. of tuberculin would kill most guinea-pigs suffering from a tuberculosis of four to five weeks duration, and that 50 mg. would prove fatal without exception in from six to thirty hours.

It seems that tuberculo-protein in lethal doses produces its most acute effects on the blood-vessels, death being due to a paresis or paralysis of

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their walls. The opinion that this is a form of allergic reaction is favoured by the fact that the same dose given to a non-allergic pig fails to produce any serious inflammatory response. This reaction in vessels must be borne in mind in accounting for certain hæmoptyses which occur in the course of tuberculosis.

The Allergic Reaction Differs in Infected and Non-infected Tissues of the Body.

The body cells which possess the power of allergic reaction to the greatest degree are those in active and unhealed tuberculous foci, although all body cells in the infected organism are rendered allergic. The fact that the skin; the conjunctival, nasal, vaginal and rectal mucous membranes, the subcutaneous tissues, the muscles, the testicles, in fact, all tissues will react to the application of tuberculin when it is held in prolonged and intimate contact with them, but do not react to the tuberculo-protein which is set free into the circulation from a tuberculous focus within the body, as the cells about the tuberculous process do, indicates that the perifocal cells are the more sensitive to bacillary protein.

The allergic (auto-tuberculin) reactions met in the course of active tuberculosis are confined to those tissues which harbour living tubercle bacilli, particularly unhealed lesions. There is a continuous kaleidoscopic change going on in such tissues as a result of tuberculo-protein coming in contact with the cells in varying amounts at varying intervals of time. Sometimes the reaction is microscopic; at other times it may be severely exudative in character.

It is necessary to understand the relationship of the focal tissues to the allergic reaction, in order to comprehend the clinical aspects of the disease. The focus in which the newly inoculated bacilli settle is naturally most affected because here the chief contest between the invading bacilli and the cells endowed with specifically developed defence takes place. Here tuberculo-protein is set free as a result of the destruction of bacilli and is found in greatest concentration, whether the amount be small as in minimal reinoculations, or large, as in massive ones. Therefore, it is evident that the conditions favourable for severest reactions, viz., largest quantities of tuberculo-protein and most highly allergic cells, are present at the site of reinoculation, and in the tissues immediately surrounding it. It is also evident that small reinoculations will be accompanied by comparatively slight reactions in the focus of implantation, while massive ones will be accompanied by violent exudative inflammatory change.

While the conditions in the tissues at the point of new bacillary implantation are favourable for the production of the severest reactions, it is clear that all unhealed foci in the body contain highly sensitised cells which are capable of reacting with the circulating tuberculo-protein which contacts them. In comparison with that in the original focus, however, this reaction is of milder degree, because it is caused by relatively minute quantities of tuberculo-protein playing upon the sensitised cells of unhealed tubercle wherever they may be found in the body and are to be considered as important factors in the healing of the disease. Even reactions followed by extensive exudation and necrosis carry with them the power to stimulate

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the reticulum in the tubercle to the formation of new fibrous tissue. Necrosis in the centre of tubercle and fibrosis at the periphery are phenomena not uncommonly found simultaneously in tubercle; in fact, they go hand in hand, as a rule.

The Importance of the Allergic Reaction in Diagnosis.

Tubercle bacilli may enter the body of a non-infected individual in fairly large numbers, and not make their presence known for several days, because they have no way of showing their effects clinically until the specific immunising mechanism, with its cell sensitisation, and the ability of the tissues to react allergically, has been established. When this has occurred, the entire picture changes. When tuberculo-protein is again brought into intimate contact with the body cells they show a condition of increased sensitisation toward it. This is shown at the site of inoculation as an inflammatory reaction when the tuberculin is applied locally, or as a general reaction with clinical symptoms appearing from within a few hours to one or two days, when the dose is administered subcutaneously.

Koch, in 1890, recognised this allergic reaction, although not by the name, as the basis of tuberculin diagnosis. He found that a tuberculous animal or man reacted to the subcutaneous injection of tuberculin with the same symptom complex which follows reinoculation of bacilli, while a non-tuberculous individual did not. He therefore suggested its use as a diagnostic test for tuberculosis. After considerable experience in administering the subcutaneous tuberculin test, he came to the conclusion that a reaction or failure of a reaction to 10 mg. of tuberculin could be taken as deciding the presence or absence of tuberculosis. Koch's method of administering the tuberculin test was first to give 1 mg. If no reaction occurred, he gave 5 mg.; and then if no reaction occurred, 10 mg. The reaction of the test at the site of injection was not heeded. It was the general reaction, accompanied by symptoms such as chill, rise of temperature, increased pulse-rate, increased cough and expectoration, and general malaise, that was taken to mean a positive reaction.

Focal reactions were also taken into consideration in all areas of visible tuberculous involvement. Later, the presence of râles in the lung, following the injection, when they had not been present before, or an increase in those that had been present, was taken to mean a focal reaction. When X-ray examinations began to take part in diagnosis it was found that focal reactions appear as increased exudative (parenchymatous) phenomena. Later, it was shown that, in the tuberculous, the cells of the skin, subcutaneous tissue, conjunctival, nasal, vaginal and rectal mucous membranes all possessed increased sensitiveness to tuberculin. This gave a new meaning to the Koch phenomenon, and several new methods of applying the tuberculin test were suggested, such as the cutaneous of von Pirquet [31], the intradermal of Mandel [32] and Mantoux [33], the percutaneous of Moro [34], and the conjunctival of Woll-Eisner [35] and Calmette [36].

Some of these tests were so simple, and the effects on the patient were so slight, when compared with the subcutaneous method, particularly in that they were not accompanied by the discomfort of a general reaction. that they came into general use and were employed in testing very large

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groups of individuals of all ages. This applied particularly to the cutaneous and intracutaneous methods.

It was a great surprise to find that nearly all children reacted to tuberculin, while comparatively few of them were suffering from active tuberculosis. From this it was learned that there is a difference between being infected with tubercle bacilli and suffering from tuberculosis. The important fact was also established that being infected is sufficient to cause positive reactions. Therefore, it became evident that the tuberculin reaction must be interpreted as cell hypersensitiveness to tuberculo-protein, due to prior infection with bacilli and not be taken as evidence of tuberculous disease.

In making a diagnosis in borderline cases, the fact of the absence of Koch's infection can be utilised to great advantage [37]. This is particularly true when examining nervous and psychical individuals, and those individuals with endocrine imbalance, who have symptoms simulating tuberculosis, also those suffering from post-influenzal, and subacute and chronic bronchitis, in whom it is necessary to consider the possibility or probability of a tuberculous disease. In such cases a tuberculin test should always be given as one of the first procedures in diagnosis, because of the value of a negative reaction. A negative test in a healthy individual is presumptive evidence that no tuberculosis is present, or at least no tuberculosis of an active form.

There are certain conditions, however, under which immunity may be markedly depressed and the reaction fail to appear, or appear but feebly, even though active disease be present. Such are pregnancy, the acute infections, particularly influenza, measles, whooping-cough, scarlet fever, cachexias, and extremely low states of general strength, also active advancing tuberculosis with relatively massive reinoculations.

Skin sensitisation, as found in the application of the tuberculin tests, is not the only use of allergy in diagnosis. The acute symptoms in tuberculosis, those of the toxic as well as those of the reflex group, and some of those due to the local action of the disease, are dependent upon the inflammatory allergic reaction for their existence. The disease may come on slowly after weeks or months of increased fatigability and loss of vigour; or, on the other hand, a patient may feel perfectly normal, with tuberculous infection slumbering within his body to-day. A reinoculation may take place, and to-morrow, as a result of his allergy, he may be suffering from the symptoms of acute disease. He may have temperature, malaise, increased pulse-rate, and the other symptoms belonging to the toxic group. He may further have a cough with expectoration. Or, if the disease should be in the pleura, he may have an acute pleurisy. The rapidity of the reaction is due to the shortening of the incubation period as a result of previous inoculation. Without allergy, as in the non-immune, the individual would not develop symptoms until he had gone through the incubation period and the bacilli had had time to fix themselves in the tissues and start in motion the immunity mechanism. Therefore, these important diagnostic symptoms are but an expression of disturbed function on the part of the host, brought about by the sensitised cells reacting to the presence of tuberculo-protein set free from active foci, particularly those at the seat of reinoculation. Bacilli destroyed by the host probably furnish a second source of supply.

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The diagnostic importance of the X-ray in early tuberculosis is also based partly on the allergic reaction—not only the allergic reaction but a fairly marked allergic reaction in which considerable exudation takes place in the tissues. An allergic reaction may be of any degree from that of the mildest proliferation of cells, or the slightest exudation of serum into the tissues, to the most severe exudative process in which serum, cells and fibrin pass into the tissues and, in case of the lung, into the air passages; or even necrosis may take place. The reactions are all the same qualitatively, that is, they are all allergic, but they differ quantitatively. In order to get the increased flaky effects about a focus in an X-ray plate, as a result of a tuberculin injection, one must have exudative phenomena of a fairly marked degree.

The so-called parenchymatous lesion, as shown in the X-ray plate in early tuberculosis, is nothing more than an allergic reaction, sufficiently marked to produce more inflammatory changes and a greater amount of exudation into the tissues than that which usually accompanies the slightly exudative or the predominantly proliferative process.

Pathologic Change Produced by Tuberculo-Allergy.

The allergic reaction has a varied and varying pathology of injury, hyperæmia, proliferation, exudation and necrosis. While tubercle is the normal response of the tissues to primary infections, it is also present, but as a secondary process, in reinfections.

Reaction to reinoculation always means some degree of inflammatory response. It may be only a hyperæmia or a mildly proliferative reaction, or, on the other hand, it may be a severe predominantly exudative or necrotic one.

At the present time there is an unfortunate tendency on the part of students of tuberculosis to accept the classification of tuberculosis into the proliferative and exudative, suggested by Aschoff [38], as a basis not only of pathologic study but also as a basis for clinical consideration. This classification is a purely anatomical one and leads to faulty clinical conception. (Pottenger [39].)

The proliferative is suggested as the benign type, favourable for healing; and the exudative type as the severe type, unfavourable for healing. These are not different types of tuberculosis, nor even different types of reaction. They are both reactions of the tissues towards tubercle bacilli and tuberculo-protein in infected individuals. The reaction is the inflammatory allergic response of the tissues. One is a mild response, the other a severe response, but neither in itself can be made a basis for prognosis.

Neither proliferative nor exudative reactions exist alone as distinct pathologic entities. They are both found in the same pathologic picture. In fact, one cannot conceive of inflammatory reaction without hyperæmia and some degree of exudation of serum or cells; and, furthermore, one finds in the severest reactions to bacillary disease a proliferation going hand in hand with exudation and destruction [40]. Even necrosis is not separated from and unattended by proliferative reaction.

Proliferative tuberculosis is not an exclusively proliferative form,

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pathologically, but only one in which the formation of new tissue, only predominates. Cell proliferation is present to some degree in all lesions, for it is the normal response of the cells to injury. It goes hand in hand with exudation as the two regular responses of the body tissue to bacillary reinoculation or to tuberculo-protein.

At times in the course of clinical tuberculosis the allergic reactions are so mild that the response on the part of the tissues seems to be almost wholly proliferative. There are reticular cells in every tissue in which tubercle formation takes place. These are a very important part of the cellular defence in tuberculosis. They take part in the allergic reaction, are irritated as a result of it, and as a result of the irritation proliferate and form new tissue. These cells are present in greatest numbers in the periphery of tubercle, but are found in less numbers in the centre of the focus itself. As a result of the irritation produced by the allergic reaction new cells are formed which push inward and often obliterate the entire tubercle, converting it into a fibrous mass. Even caseous tubercle may be replaced by fibrosis as a result of this proliferating process.

All tubercles containing viable bacilli are subject to allergic or auto-tuberculin reactions, as long as reinoculations occur, or as long as tuberculo-protein escapes from unhealed foci. The effect of such reactions, except at the site of reinoculation, unless they be severe, is most evident in the perifocal cells, where they cause increased fibrosis, and may produce encapsulation and healing. The larger reactions produce a more profound effect. They are followed by exudation, sometimes severe, affecting any unhealed tubercles that may exist in the body at the time. But even in the severest exudative reactions, stimulation of the local reticular cells takes place producing an increase in fibrous tissue. Both proliferation and exudation are a part of all allergic reactions.

Exudation may be so slight as to be microscopical, in which event its main effect is to favour proliferation of fixed cells. It may be more severe and result in the watery elements of the blood passing into the tissues, or the exudate may contain cells and fibrin. The tissues may be literally engorged with exudate and, in case of the lung, the alveoli and bronchi may be filled with it.

In many such instances necrosis becomes an important part of the process.

It readily can be seen that the exudative process, when it results from a severe reaction, is pregnant with most serious damage to the tissues, and, if it were not changeable, it might result disastrously. But it is subject to change. In case reinoculations cease, the exudate may resolve and after a time the entire process take upon itself a mild character in which proliferation becomes the most important part of the picture.

The exudative process *per se* is not serious. The fact of its existence in the course of clinical tuberculosis in a severe form, however, indicates that bacilli are escaping in fairly large or very large numbers from existing foci to form new implantations, and that much tuberculo-protein is gaining access to the blood-stream. This may be taken as presumptive evidence that large quantities of bacilli may continue to escape: and, if they do, the resulting allergic reaction will cause an increase and a prolonged continu-

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ance of the exudative process which may cause not only serious damage to the tissues, but may even prove to be inimical to life.

It is of the utmost importance that clinicians should hold the classification of tuberculosis into proliferative and exudative types as being without definite prognostic importance. An exudative process does not mean a bad prognosis, nor does a proliferative process mean a favourable prognosis. A predominantly proliferative process may be transformed into a predominantly exudative one inside of twenty-four hours through a fresh invasion of tubercle bacilli. A predominantly exudative process may, on the other hand, in the absence of a repetition of too many and too severe reinoculations, be transformed, in the course of a few weeks or months, into a process which is predominantly proliferative. We often see these changing effects during the course of clinical tuberculosis, quickly in the one case, even over night, slowly in the other, requiring weeks and months. Where the exudative process disappears particularly slowly we are justified in assuming that repeated reinoculations are taking place, which keep up the allergic reaction, even if they are not sufficiently severe to produce an elevation of the temperature curve with other accompanying toxic symptoms.

It required the labour of two geniuses, that of Laennec in pathology, supported by that of Koch in bacteriology, to establish for all time the ætiologic unity of all tuberculous processes. Now we have reached the stage in our study of tuberculosis when our conceptions of the various pathologic reactions and clinical responses to reinoculation should be unified; for they are all, in truth, only various degrees of response of cells, which have been rendered allergic by previous infection, to bacilli and bacillary protein, the degree of response being particularly determined by the quantity and quality of previous infections, the character of the tissues involved, and the reacting stability of the patient at the time of the infection and thereafter. Let us hope that it will not take so long to establish this unity.

One form of tissue response, or rather lack of response, is now recognised, in which tubercle bacilli may lie in the tissues without calling forth any recognisable response from the body cells (Bartel [41] and Opie [42]). This fact is difficult to explain on the basis of any of our notions of immunological or pathologic response, and requires future study for its elucidation. One may well speculate on the possibility of such bacilli taking upon themselves pathogenic qualities at times and being the sources of re-infections.

Necrosis, with its death of tissue, fits into the picture as a phase of the response of hypersensitive cells.

In attempting to unify the body's reactions on the basis of immunological response, we meet many factors which are followed fairly consistently by more or less definite response; but there are so many of these factors, such as numbers and virulence of bacilli, the inherited structural peculiarities of tissue, the nerve, hormone and psychical balance of the host, factors of environment, both physical and emotional, which enter into the body's response, that there is no way of determining in any given individual what his particular reaction to a particular reinoculation will be. We do assume, however, that the more serious the infection, and the lower the

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physical state of the host, the more severe the disease, and the less the infection and the more perfect the physical state of the host the less severe the disease. The term "more serious" as used here means more widespread, as a result of the immunological mechanism of the host being unable to function adequately. It must not, however, be confused with a more violent body response, which is found in case the host's specific immunologic mechanism is highly developed. The most serious thing that can happen to a tuberculous patient is to have a given reinoculation fail to be met by an adequate immunological response.

Caseation and necrosis are the common destructive changes which take place in tissues which are subjected to allergic inflammatory reaction. Confusion in assigning to them a satisfactory explanation arises from the fact that contradictory conditions seem to be followed by necrosis. Primary tubercle caseates, even though the infection be relatively mild; and the regional lymphatic glands which receive drainage from the primary nodule usually show marked caseation. Again marked caseation with loss of much tissue rarely takes place in chronic tuberculosis except as the result of severe reinoculation, and then the necrosis is most evident at the point where the reinoculation has taken place. Frequently the first severe reinoculation that takes place when the disease becomes active is followed by a fairly large cavity, after which the disease may spread and subsequent reinoculations occur without other large cavities appearing.

From the standpoint of allergic reaction the conditions seem to be markedly different in these three instances. In the primary nodule caseation seems often to occur quickly before marked allergy has developed; the glands caseate under much the same condition. Ranke [43] has described this period of developing sensitization as the period of the primary complex. Caseation with marked loss of tissue and cavity formation occurring as a result of the implantation of large numbers of bacilli early in the course of the disease, or following large reinoculations later in the course of the disease, seem to be favoured by a relatively low specific defence; or the correct explanation may be that the tissues are highly sensitised and the antigen is set free in large quantities. While the specific defence of the host as a whole may be lowered, it may actually be very high at the point of inoculation.

The facts do not seem quite clear enough to warrant a dogmatic statement. But it is generally accepted among students of immunology (Long [44], Krause [3]), that caseation is due to an excessive reaction in highly sensitised tissues. Caseation with cavity formation in the lung performs a protective service to the host in that large numbers of bacilli are cast out by the body with the expulsion of the cavity contents. These otherwise might spread and overcome all natural and acquired resistance and produce a rapidly fatal disease.

Several factors may contribute to caseation, some of which may be only incidentally a part of the immunological ensemble. Thus it has long been suggested that necrosis may be due to a failure of the cells to respond with a granulation tissue carrying the necessary new blood-vessels (Huebschmann [45]), or to a poisoning and destruction of local cells by toxins which are set free by the infection while the specific protective factors are held more or less in abeyance (Pagel [46]).

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Clinical Tuberculosis and Allergy.

An acute infectious disease, such as measles, whooping-cough, diphtheria, scarlet fever or small-pox, represents one inoculation and one immunity response on the part of the host. The symptom complex which ushers in the disease with temperature, rapid pulse, and so forth, is the immunity reaction. This does not appear until the infecting micro-organisms have been in the tissues sufficiently long to set in operation the specific immunity mechanism of the individual. The result of the disease is either a sufficient and usually permanent immunity, and freedom from attacks by that particular micro-organism thereafter, or a failure of immunity and death. There is only one episode of infection and immunity response.

Tuberculosis, on the other hand, is different. The first infection with bacilli, after a period of incubation, calls forth an immunity response, and the disease may heal if the response is competent; it may be held in check so that further extension does not occur, in cases where healing is not accomplished; or, in case the immunity is inefficient the disease may spread, producing an immediate widespread disease. Where healing fails to occur, each new spread calls forth an immunological tissue response, and if the immunity is finally raised to a sufficient competency, healing may be brought about, even in relatively severe primary infections. Failing this, however, the disease progresses with a gradual lowering of specific resistance until death occurs [47].

Chronic tuberculosis is tuberculosis which develops in an immune individual, that being one whose immunity mechanism has been excited to action by a previous infection; and, usually, one in whom the immunity was sufficient, at least for the time, to cause the early infection to become quiescent.

Active tuberculosis is not brought to a close by the destruction and eradication of bacilli from the body, as is the case in the acute infections mentioned above, but bacilli remain in the tissues ever ready under favourable conditions to spread, multiply, and cause a reinoculation. The inability of the host readily to destroy the bacilli is due to their peculiar waxy content which makes them particularly resistant.

The course of chronic tuberculosis is, as a rule, punctuated by several or many episodes of reinoculation and immunity reaction [48]. These continue to take place in cases which heal until all avenues of escape from existing foci are closed and the bacilli are walled in. Except in instances in which all of the bacilli forming the infection are destroyed, a competent encapsulating fibrous wall is the determining factor in healing, and also in maintaining healing when attained. An incompetent wall is always a danger because it furnishes the point from which reinoculating bacilli may escape to set up new activity. The competency of this wall depends upon the production of fibrous tissue by the perifocal reticular cells, in response to stimulation by tuberculo-protein (tuberculin) which escapes from certain foci surrounded by incompetent investing walls. Thus, there comes a time in the healing of tuberculosis when the tuberculo-protein necessary to stimulate the perifocal cells to completely envelop the bacillus-containing tubercles may be lacking in amount, and so healing may be incomplete.

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This is probably a very common cause of incomplete healing and relapse in clinical tuberculosis.

The allergic inflammatory reaction which is called forth by the spread of bacilli is probably the cause, directly and indirectly, of nearly all of the symptoms attendant upon active tuberculosis, as may be judged from the following classification, which I first offered in 1913 [49], but which has been slightly modified as follows [50] :—

ETIOLOGICAL CLASSIFICATION OF SYMPTOMS OF PULMONARY TUBERCULOSIS.

Group I Symptoms due to toxæmia and other causes acting generally	Group II Symptoms due to reflex cause	Group III Symptoms due to the tuberculous process *per se*
Malaise	Hoarseness	Frequent and protracted colds (tuberculous bronchitis
Lack of endurance	Tickling in larynx	Spitting of blood
Loss of strength	Cough	Pleurisy (tuberculosis of pleura)
Nerve instability	Digestive disturbances (hypermotility and hypersecretion), which may result in loss of weight	Sputum
Digestive disturbances (hypomotility and hyposecretion)	Circulatory disturbances	
Metabolic disturbances resulting in loss of weight	Chest and shoulder pains	
Increased pulse-rate	Flushing of face	
Night sweats	Spasm of muscles of shoulder girdle	
Temperature	Diminished motion of affected side	
Blood changes		

There are probably three sources of toxins which are responsible for the general or toxic symptoms of tuberculosis: (1) Broken-down focal tissues; (2) the serous, cellular and fibrous exudate; and (3) broken-down tubercle bacilli. We must assume that any and all of the products of a tuberculous focus, or of a tuberculous exudate, as a result of the changes which they undergo as they are subjected to the action of the defensive forces of the body, may produce toxins which are capable of causing toxic symptoms.

The reflex symptoms can readily be seen to be caused by the inflammatory process in the lung, which irritates the nerve-endings of the vagus and sympathetic afferent neurons which carry the stimuli to the centres in the medulla and cord and transfer them to other efferent neurons to complete the reflexes.

The local symptoms, or those due to the tuberculous process *per se*, originate in the tissues which are the seat of the disease; thus the hæmorrhage from the injured vessels, the expectoration from the diseased focus or from the exudate in the air passages, the pleurisy from the tuberculous involvement of the pleural leaves, and the "cold," tuberculous bronchitis, from the inflammatory exudate or ulceration into the bronchi, may all be expressions of allergic reaction.

Not only the individual symptoms, but the course which the disease takes is determined by the degree of competency of the immunity mechanism which we may measure fairly well by the allergic reaction.

As mentioned above, proliferation and exudation go hand in hand as parts of the same process; necrosis, too, often accompanies. A predominance of the proliferative phenomena denotes mild reaction and

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indicates at that particular time a disease process of relatively slight degree; but through a severe reinoculation an allergic (auto-tuberculin) reaction might be precipitated, which would within a few hours cause exudative phenomena to predominate. A preponderance of exudation, on the other hand, denotes at the time a more severe process. But this, too, may change, and after a time assume a predominantly proliferative form.

There are also varying degrees of reaction which occur with relatively the same infection, the difference being on the part of the host and the degree of allergy with which his tissues are endowed.

We can best illustrate the pathologic changes as well as the clinical manifestations in their relationship to allergy by a clinical illustration. A patient with an advanced tuberculosis involving the upper half of both lungs has been relatively free from symptoms for two months. He has gained in weight and feels relatively well. He has no temperature, normal pulse, good appetite and digestion, and a satisfactory degree of physical strength. He coughs and raises 30 c.c. of bacillus-bearing sputum per twenty-four hours. Suddenly he feels toxic and aches, his appetite fails, his temperature starts to rise, and on the third day reaches a maximum of 102° F. His pulse increases in rate. Cough increases. Expectoration reaches 60 c.c. per day and the numbers of bacilli per microscopic field treble. He feels prostrated, is irritable, and fails to sleep. In one week temperature has again attained normal. He is regaining his nerve balance; appetite returns, and he rapidly regains his usual well-being. Cough and expectoration, however, continue more severe than before for some days.

The cause of these clinical phenomena was that the patient gave himself a reinoculation of quite a large number of bacilli. Some exit in the defensive wall which had been thrown around his tuberculous foci was unguarded; or, it might be that some new break occurred, allowing the walled-in bacilli to escape and attempt a new infection.

The cells which contacted the migrating bacilli, being allergic, reacted quite violently. The bacilli were in part destroyed and tubercle protein was set free which, circulating in the blood-stream, caused further inflammatory allergic reaction in other unhealed tubercles in the body. So there was widespread allergic reaction of varying degree called forth throughout the infected lungs.

Pathologically, changes from a proliferative and a mildly exudative process to a markedly exudative process took place. The pulmonary tissues and air spaces were filled with exudate. The exudation which took place in the air-passages caused an increase in expectoration and also an increase in the cellular content of the sputum. Ulcerated surfaces were stimulated and cast off an increased number of bacilli in the sputum.

Physical examination showed an increase in moist râles throughout the areas of active disease, being much greater in some areas than in others, the difference being in part at least caused by the state of healing of the affected tissues. The X-ray showed an increase in exudative reaction, being most marked in the areas in which the disease was most active.

The effect of the immunity reaction in this instance was to exert protective influence against the invasion of new tissues by the bacilli and also to exert a beneficial influence by promoting healing in former foci.

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The first purpose was carried out by an attempt at destruction of the bacilli which had escaped from their former foci, and failing this an immobilisation of them to prevent their spread. The second purpose was effected through stimulating unhealed foci containing living bacilli, thus increasing the fibrous tissue about them and causing them to assume more and more of a proliferative character. Incidentally, the patient's immunity was raised by the process, and he became better able to oppose future attempts at reinoculation.

A few weeks later the acute symptoms had disappeared. The cough had lessened and the expectoration had dropped to an amount slightly below its previous level. Examination revealed a lessening of râles and the X-ray showed a diminution of the exudative inflammation which had characterised the picture taken at the time of the reaction, and an increase in the proliferative phenomena, showing that healing had been promoted by the reaction.

The Protective and Healing Effects of Allergy.

From the fact that allergy is a manifestation of immunity, it is necessary for us to assume that it is a necessary part of the body's defence-reaction. It is profitable to inquire what its defensive properties are and the manner in which they may be exerted to the greatest advantage of the host. Particularly is this true since we can follow it through the course of the disease from the time that the sensitisation of cells takes place, a few days or a few weeks after the primary infection, until healing has been accomplished, or death has occurred.

Every case of chronic tuberculosis shows throughout the stage of activity a continuous play of allergic manifestation. When in a strong and healthy individual all traces of allergic inflammation are gone and the body cells no longer react to tuberculo-protein, then the individual is no longer tuberculous. Krause [51] reports such a case and I have seen numerous such who have been free from disease for years.

It is very important to understand the relationship of proliferative and exudative processes to allergy, for otherwise the changes which take place during the clinical course of tuberculosis and the process through which healing is effected must remain unintelligible. The basis of this understanding lies in the fact that the allergic reaction is inflammatory. A part of every tuberculous inflammation, whether it results in tissue formation or tissue destruction, is exudation. The exudation differs in character according to the strength and character of the exciting cause.

The allergic reaction may be so slight as to cause only minimal exudation. There may be only a slight amount of serum and only a few cells poured into the tissues and this effect may be transient. On the other hand, it may be so severe that the tissue spaces and natural channels, such as bronchi, may be filled, literally gorged, with serum, cells and fibrin. In the case of the lungs there is a true pneumonic process.

Allergy, other factors being equal, increases with the repetition of successfully combated reinoculations. The summation of effects from repeated minimal reinoculations, such as we conceive of as taking place regularly and frequently—it may be hourly, it may be daily—in chronic tuberculosis in which the bacilli are not walled in, is a well established

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allergy. The same may be brought about much more quickly and probably more surely by the host overcoming a severe reinoculation, or several severe ones.

From the time of the primary inoculation and the early reinoculations up to the time the disease has become well established as a chronic process, allergy is built up to such a height and with it the immunity of the host is raised to such a point, that millions of bacilli may pass over the mucous membranes of the bronchi daily without producing infection.

The allergic reaction renders certain very important services to the host, thus: (1) Allergy enables the individual who receives a reinoculation to withstand much greater numbers of bacilli without becoming infected, than are required to produce a primary infection, and furthermore it is further increased by each successive reinoculation which is successfully combated; (2) it aids the host in destroying many bacilli, both before and after implantation has occurred, causing many infections to be abortive in character; (3) it detains bacilli at the point of inoculation and prevents their spread throughout the body; (4) it heals lesions when once formed by causing a proliferative process in the reticulum in the periphery of tuberculous tissue by which a fibrous network gradually displaces inflammatory tubercle and necrotic tissue.

Before entering upon a discussion of these protective characteristics of the allergic reaction it is necessary to understand that the body's immunity mechanism can also be set in motion, though less efficiently, by the injection of dead bacilli (Koch [1], Petroff [52], Lowenstein [53], and Baldwin, Petroff and Gardner [54]), and probably to a minimum extent by tuberculo-protein (tuberculin) (Stewart and Rhoads [55]).

I have discussed this phase of the subject quite fully in recent papers [56], [57], [58].

It further must be understood that while the greatest degree of immunity is established by living bacilli, the stimulation of immunity reactions and the increase of the immunity itself after once established can be brought about by either living or dead bacilli or any bacillary substance containing specific tuberculo-protein. This is the basis of the natural healing process in tuberculous lesions and for the curative effects produced by tuberculin when administered therapeutically (Koch [1], Pottenger [56], [58]). "Every tuberculous patient treats himself with tuberculin whether he wishes it or not" (Long [59]), and, without so doing, his lesion would doubtless fail to heal.

In order to make clearer the protective and healing qualities of allergy we shall discuss these protective properties in greater detail, realising that they cannot be wholly separated but that they will dovetail into each other and overlap, because they are all parts of the same reaction.

(1) *Allergy enables the individual who receives a reinoculation to withstand much greater numbers of bacilli without becoming infected than are required to produce a primary infection: and furthermore, allergy is further increased by each successive reinoculation which is successfully combated.* This has been shown experimentally by many observers. The reports of Römer, Hamburger and Krause previously mentioned are conclusive. It is also evident to those who observe their clinical cases closely. Oftentimes a reinoculation is attempted with bacilli sufficient to produce a

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severe reaction with fever, aching, increased cough and expectoration. An X-ray plate taken at the time will show a general exudative process throughout the areas of active tuberculosis, due to the auto-tuberculin reaction. Sometimes it is impossible on account of the generally increased density of the shadows to tell where the inoculation occurred; but at other times it can be determined quite readily. Plates taken at comparatively frequent intervals thereafter may show in the course of a few weeks the entire reaction to have faded away and even the field of reinoculation to have cleared, indicating that permanent implantation had either failed to take place or that it had been limited to a small focus.

It is generally recognised that the infection which takes place by way of the air-passages, when a bronchus becomes plugged with infected cavity contents or material from a caseating bronchial gland, results in one of the most serious forms of tuberculosis; yet bacilli in large numbers may pass over the same bronchus day after day, provided the lumen is free, without producing infection. This is definite proof of the specific resistance of the immune tissue to the tubercle bacillus.

Many abortive infections occur in the course of chronic tuberculosis when bacilli escape from previously infected areas into the immune tissues of the host. While no one can tell how many such occur in the lung, because of our inability to determine accurately, they are frequently observed in the eye, larynx, lymphatic glands, and testicle during the course of chronic tuberculosis. Inoculation occurs; allergic reaction follows; and after an inflammatory course of a varying period of time the seat of infection clears. The body has been able to withstand the bacillary invasion with no resulting harm because of its well developed specific defence.

(2) *Allergy aids the host in destroying many bacilli both before and after implantation has occurred.* It is generally accepted that the bactericidal effects of the tissues of the immune is much greater than those of the non-immune. Wolff-Eisner [60] maintains that lysis of bacilli is an important factor in the immune. Metschnikoff [61] and Wright [62] based important immunologic studies upon the increased phagocytic power of body cells in the presence of immunity.

Bacilli injected into the blood-stream are quickly destroyed often without being responsible for new implantation. Krause and Hofer [63] noted the destruction of bacilli within twenty to thirty minutes when injected into the peritoneal cavity of pigs infected with tuberculosis. The fact that infection does not always follow moderately large doses of bacilli in the immune is evidence that lysis takes place.

(3) *Allergy detains bacilli at the point of inoculation and prevents their spread throughout the body.* This fact was first observed by Koch and described as a part of the immunity response. He noted that in the immune animals the bacilli were detained at the point of injection by an inflammatory process; that ulceration took place in an attempt to eliminate the bacilli from the body, if the inoculation were in the superficial tissues, and that the neighbouring lymph glands took little part in the picture, showing that passage through the tissues was interfered with, as compared with the rapid involvement of lymphatic glands which takes place in primary infection.

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The same thing has been shown clinically. In chronic disease of the lung, or larynx, or intestines, the lymphatic glands into which the affected parts discharge their lymph take little part in the process. Quite different is it in the primary lesion. A small nodule in the lung is often followed by enormously enlarged and necrotic mediastinal glands. Ghon [64] learned that by examining the mediastinal glands and determining what ones, if any, were involved, he could follow back into the areas in the lung which drained into the particular gland and find the primary focus.

Krause and Willis [20] have performed a most important service by comparing experimentally the different rates with which bacilli make their way through the tissues of the immune and non-immune. Their results show that:—

"(1) In normal non-immune guinea-pigs tubercle bacilli, inoculated intracutaneously or subcutaneously, are carried almost immediately (within an hour) from the portal of entry by the lymphatics.

"(2) Within three or four days they have made the circuit of the body.

"(3) In immune (allergic) animals their transmission is greatly retarded.

"(4) They remain fixed at or near the portal of entry for about seven days.

"(5) They do not reach the regional lymph nodes (superficial, inguinal and axillary) until two weeks after infection.

"(6) They do not become generalised to the body until three or four weeks after infection."

These experiments can only be interpreted as meaning that the tissues of the infected animal develop a defensive property that renders passage of bacilli through them very difficult. If we inquire into the nature of this defence we find that it consists of an inflammatory response. When bacilli enter immune tissues, an irritation, followed by inflammation with accompanying exudation, the degree depending on the severity of the response, takes place very quickly. The bacilli are hindered in passing through the tissues by this protective process which manifests itself in each new group of cells into which the bacilli find themselves carried by the phagocytes.

There is no doubt that this localising power of the allergic reaction is one of the greatest defensive forces of the immune host. When this power has once been established in an animal or man small reinoculating doses of bacilli are probably held regularly at the point of inoculation and destroyed without permanent harm being done to the host. We assume that this must occur during the course of clinical tuberculosis, and if we are right in our interpretation of our physical findings and X-ray plates, we see definite evidence of it. Larger reinoculations are not regularly restrained so easily, but even they frequently are cared for by the specific defensive mechanism without permanent harm resulting to the host.

(4) *Allergy heals lesions when once established, by causing a proliferative process to take place in the reticular cells in the periphery of tuberculous masses by which a fibrous network gradually displaces inflammatory tubercle and necrotic tissue.* This effect of the allergic reaction is the most important one in the healing of tuberculosis. The first three

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aids rendered by the allergic reaction are directed particularly toward protecting the organism from new invasion of bacilli, but the fourth is particularly directed toward healing the disease when established.

The fact that the infected animal is able to withstand larger doses of bacilli without serious harm than the healthy one, the fact that he is able to destroy more bacilli than the healthy one, causing implantations to fail or infections, if they occur, to heal, and the fact that bacilli pass through the tissues of the immune more slowly are [redacted] evidence that resistance to tubercle bacilli is conferred upon the host by previous infection.

A specific inflammatory reaction occurs at the site of invasion as a result of each reinoculation. Furthermore, a reaction occurs in other unhealed foci in the body if the numbers of bacilli engaged in the infection are sufficient to cause more than a minimal amount of tuberculo-protein to gain access to the blood-stream. These two reactions are of the greatest importance in healing tuberculous lesions.

The allergic reaction which is called out by the tuberculo-protein which escapes from foci and from the bodies of bacilli which go into solution as a result of the bactericidal properties of the immune tissues, is responsible for the inflammatory reaction which has as its sequel the formation of fibrous tissue, in and about tubercle, changing the tubercle into fibrous scar and encapsulating bacilli where they cannot be destroyed.

Without this effect the probabilities are that tuberculosis would not heal save in exceptional instances; but, with it, nearly all tuberculous foci in the body of the immune host show some tendency to heal, and even those that at times seem most serious show healing along with their destructive processes.

In order to understand the allergic, inflammatory, reaction, it is necessary to recall the fact previously stated, that the action of tuberculo-protein is, relatively speaking, a quantitative one, the more concentrated the product the more severe the reaction. And, further, it is just as important to know that the cells of the immune host are endowed with different degrees of ability to react allergically, and that cells in and adjacent to foci which contain living bacilli possess this power to the highest degree.

So it is evident that there are three different reactions to tuberculo-protein possible in the tuberculous individual: (1) The severest one at the site of reinoculation because the bacillary products are most concentrated and the cells are most sensitive at that point; (2) a less severe one in other tuberculous tissue containing living bacilli; and (3) a possibility in all the tissues of the host, in case the protein is introduced artificially in the tissues. This latter has diagnostic import, aids one in forming an opinion of the degree of allergy present, and aids healing should infection occur.

Taking into consideration the effect of the allergic reaction, one can visualize what this inflammatory exudative and proliferative process means in the course and cure of tuberculosis. It is primarily protective, protecting against spread of infection and healing existing lesions.

The slighter stimulations cause a minimum of inflammatory signs and favour healing, the larger ones produce more marked evidence of inflammation and may be harmful. But no matter how severe the reaction, and no matter how marked the accompanying exudative phenomena, a very

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important part of every reaction is the stimulation of local reticular cells to the formation of fibrous tissue and healing of lesions by encapsulation.

What the further course of an inflammatory exudative reaction about unhealed tubercles will be, depends on future reinoculations. If they are of only mild degree, the exudate will resolve, leaving the area with an increase of fibrous tissue which was formed as a result of the stimulation. If, on the other hand, severe reinoculations continue, the exudative phenomena do not disappear, in fact, may increase in severity and be accompanied by destruction in the most densely infected areas.

Thus the combat goes on in every individual suffering from active clinical tuberculosis. If immunity can be kept at a high level, very large quantities of bacilli may escape from previous foci, provided they do not escape too frequently, and still serious new infections may be prevented; and, as a result of the stimulation of fibrous tissue, after the acuteness of the episode has passed, the field of infection may clear, leaving the host in a better condition than he was in before; and after a number of such episodes a satisfactory healing may be brought about.

While tuberculin has not been used generally as a therapeutic measure, yet there are certain clinicians who have never ceased its employment after once beginning. They have always felt that it had a definite effect in healing. Now we are beginning to understand the nature of tuberculosis better, and to realise that both the pathologic and clinical variations of the disease consist of a series of reinoculations and immunity reactions, and that these reactions are produced by tuberculo-protein acting upon the specifically sensitised body-cells, and are an intimate and necessary part of the protective mechanism developed to guard the host from the dangers of the tubercle bacillus. We are further, as a result of this, forced to recognise that tuberculo-protein (tuberculin) is Nature's own protective and curative substance, because it raises immunity already established, and calls forth allergic reaction without which the body would not be protected against the spread of bacilli, and without which healing could not occur. When we realise further that small doses are wholly curative and only large doses are harmful, we have at our command the principle of its administration. Long [59], who, with his associates, has done so much to clarify our opinions of the nature of tuberculin, accepts its value with the following statement regarding its influence on the disease focus: "The focal reaction occurring at the site of disease, on the other hand, is of the utmost importance. Mild focal reactions are beneficial, and severe ones detrimental." Again, he states [65]: "All patients are under tuberculin treatment spontaneously, if not by actual therapeutic use. Sometimes this spontaneous tuberculin treatment is harmful, much of the time it is certainly beneficial. It is inevitable in any event." Tuberculin, too, has the advantage over the tuberculo-protein produced by reinoculation of favouring healing without the danger of a reinfection.

If one understands tuberculosis and the part tuberculo-protein plays in in its cure, he can administer tuberculin therapeutically to the great advantage of the patient. Severe and dangerous reactions may be avoided if only the physician will study sufficiently carefully the sensitiveness of each patient to the remedy.

MacCullum [66] recently reviewed the status of our present knowledge

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of tuberculin and ventured the following prediction: "From all this it is evident that very great advances have been made in our knowledge of the chemistry of tuberculin and of the tubercle bacillus, and it is not too much to hope that in the next few years we shall be in a position to combat tuberculosis fundamentally and effectively."

With the present increased knowledge of tuberculin and its importance in the production of the allergic reaction, the most promising field for addition to our present-day therapy of tuberculosis seems to be the production of mild allergic reactions about tubercle by supplementing tuberculin in measured doses at appropriate intervals and suited to the patient's tuberculo-protein sensitiveness for the haphazard doses and haphazard intervals which characterise spontaneous therapy.

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TUBERCLE.

JUNE, 1929.

Pulmonary Tuberculosis.

THE cynic is occasionally heard to whisper that the early case of pulmonary tuberculosis does not exist. He has our sympathies. All too often he is confronted with the tragedies, the well-established and the advanced cases, and all too rarely he finds the hopeful cases seeking advice early. But apart from the question of the stage of the disease as it affects treatment, a careful investigation of the stage of each case may throw considerable light on the mode of onset and be of value in illustrating the problem of infectivity. Here the advanced case, viewed strictly from the scientific point of view, has a lesson and a moral as important as the early case.

Since 1926 Dr. Th. Rehberg and Dr. W. Zürcher [1] have at their dispensary in Tilsit attempted to trace the source of infection in all their new cases, grouping the cases according to Redecker classification. In their paper they report on 200 cases which were diagnosed during the period March to September, 1927. Each group is dealt with separately.

In the primary stage there was only one case. A child of 8 years, whose skiagram showed an infiltration, which in five weeks absorbed, leaving only a small focus. Exogenous superinfection was traced as the ætiological factor.

The secondary stage is characterised by the escape of the bacilli or their toxins through the bronchial glands into the pulmonary or general circulation, and also by the allergic condition. The authors consider the bronchial glands as the starting point of this stage, but in the series found only five cases in which the diagnosis of uncomplicated bronchial gland tuberculosis could be maintained. In 12 cases of secondary infiltration (a series of skiagrams of one case are shown), i.e., three infants, four children and five young adults, these findings were in agreement with Lydtin, who, however, maintains that this type is not confined to children. The majority of these cases did very well when removed from the source of infection.

In hilum tuberculosis, if the infiltration takes place near the glands or around a metastasis near to the glands, a picture of hilum tuberculosis is produced which often progresses to cavitation. Three such cases are reported.

Disseminated secondary tuberculosis takes place mostly in the lungs, but may extend throughout the body. In the lungs the foci are to be differentiated from the primary foci in that they are usually multiple; if single they are not connected to the glands by lymphatics. Fourteen cases of this nature are given in the series. One case of miliary tuberculosis occurred.

Of the above cases, eight were infected by an "acute mass infection," two "intrafamily," six "extrafamily." In 17 cases the source of infection was more gradual, 11 intrafamily, 6 extrafamily. In two cases no new infectious source could be found, but a debilitating ætiology was present. In two other cases an intrafamily source had been present two to seven years before; an endogenous infection was therefore likely. In six cases no ætiological factors could be traced.

These cases of bronchial gland tuberculosis could give no date of the onset of the disease. In five cases the duration was some years. In 22 cases the length of illness was given as eight days to six months. The authors consider that the history of an acute illness with an open source of infection, made the possibility of an exogenous "re"-infection almost certain, particularly so when these cases improved rapidly on removal from the open source.

The authors agree that in this "generalisation period" the protective forces of the body are at their highest, and it only requires some factor to lessen the attack in order that the body may defeat the infection; indeed in this stage cavitation may heal, for this tendency to quick healing is a characteristic of the secondary disease, and in this lies

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the importance of its recognition and differentiation from the tertiary stage.

In the tertiary stage a certain degree of immunity is present, the bacilli are no longer able to escape into the circulation, but do their damage slowly in the lungs. The reaction of the body to the invader is one of fibrosis. The commencement of this stage is extremely difficult to ascertain, but is definite. The allergic condition ceases and metastases no longer take place. The most common form is the productive fibrosive phthisis, clinically recognised by the usual signs found at the apex in "early cases," radiologically as small soft clover-leaf infiltration, later becoming more fibrotic and spreading over a greater area of lung. The starting place may be a primary focus or a metastatic foci of the secondary stage. In the authors' series 75 cases were in this class. In 24 no source of infection could be traced. In 36 it was "intrafamily." On the average this source had been closed (by death) nine to seven years before. Six cases were contacts at the time of investigation. The authors argue that for the production of phthisis a temporary source of infection is unimportant, except inasmuch as the secondary metastatic stage may be caused by an exogenous infection and that one of these metastases may be the focus around which the tertiary lesion starts.

The other form of tertiary phthisis is the "early infiltration," which is a peripheral exudative focus, usually under the clavicle, which shows itself in skiagrams as an indefinite infiltration. Physical signs are absent. The clinical manifestations are those of a febrile attack.

The infiltration may be accompanied by daughter infiltration in the other lung. When the infiltration is treated by collapse therapy often the daughter infiltration recedes. The early infiltration may proceed to cavitation unknown to the patient. The prognosis is dependent on the extent to which it has progressed before treatment is commenced. Fifty-four cases fell into this group. In all of them the onset was acute and the duration short (average sixteen weeks). Twenty-five were in contact with an open source of infection, 10 had had their resistance recently lowered by illness, 12 had been in contact with an intrafamily infection two to eight years before; 17 gave no source of infection.

In 18 cases the lung condition was overshadowed by complications, i.e., 4 meningitis, 1 peritonitis, 13 pleurisy, and 4 in which the process had advanced so that no classification could be made.

From the standpoint of preventive medicine the authors sum up as follows: The primary infection in childhood is clinically exceedingly difficult to diagnose. Any child suffering from an indefinite illness should be X-rayed. Exogenous infection plays the important part and healing is rapid. The adults are divided between fibrous productive phthisis and the "early infiltration" in this series (75 : 62). The phthisical patients had been under observation five years, while the other group only eighteen weeks. It is suggestive that the phthisical cases had gone through the early infiltration stage. In phthisis the diagnosis is comparatively easy. The first infection is exogenous, and the progress to phthisis is through the secondary stage. Exogenous reinfection may make the phthisis worse. Debilitating causes also play their part, and healing is uncertain. In the early infiltration group the symptomatology is indefinite. The patient is often traced because of the cases he has infected. A fresh reinfection was present in the majority of cases. Here debilitating causes play a part. The prognosis is good if the case is seen early. In the later stages it is unfavourable. Finally the early infiltration must be "hunted for."

This report is a detailed piece of work which needs to be studied with care. On the whole it gives support to the theory of exogenous superinfection.

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BOOK NOTICES AND ABSTRACTS.

Book Notices.

Elementary Hæmatology. A Handbook for Students and Practitioners. By W. E. Cooke. London: Charles Griffin and Co. 1928. Pp. 96. Price 7s. 6d. net.

This excellent volume should prove distinctly useful to students and practitioners. It presents, in an easily accessible form, our knowledge of blood diseases, including the most recent work on the subject. Each disease has a section, on Ætiology, Pathology, Symptoms, Diagnosis and Treatment. There is a welcome chapter on Embryology, and the various cells met with, normal and pathological, are described and figured. Designed as it is largely for the use of the student, one could have wished that the chapter on Instruments and Methods had been amplified somewhat. This section is dimissed in 13 pages. For example, no mention is made of the nature of the correction used in estimating the hæmoglobin with the Sahli hæmoglobinometer, nor the fact that a tinted glass standard is now on the market, obviating a possible source of error due to fading of the standard colour tube used for comparison. It may be, however, that the latter refinement was introduced since the book was in the press. But any small defects of this kind are put in the shade by the usefulness of the work, which can be confidently recommended as an admirable vade mecum for students and others for whom it is intended. The book contains no coloured plates.

I. Report of the Miners' Phthisis Medical Bureau for the Year ended July 31, 1927. Union of South Africa. Pretoria, 1928. II. The Present Position of Miners' Phthisis on the Rand. L. G. Irvine (Private circulation).

These two documents have reached this country together. They are both written by the Chairman of the Miners' Phthisis Medical Bureau. The Annual Report presents the work of the Bureau directly from the compensation viewpoint. In the separate address Dr. Irvine envisages the position more directly from a medical standpoint. At last the annual incidence in cases of silicosis has, as predicted, been converted into a decline. At the same time a change to a milder type in the cases is reported; it is apparent in radiographs. Even more definite than heretofore the position is presented that silicosis and tuberculous infection work hand in hand, and that the fibrotic condition usually ascribed to the influence of silica particles alone only develops in the presence of the tubercle bacillus. Nevertheless, a great difference exists clinically between cases with overt tuberculosis, and those wherein no direct evidence of the presence of tuberculosis can be detected. Silicosis with tuberculosis is a particularly grave condition; next comes tuberculosis with no signs of silicosis; and, lastly, simple silicosis, which, however, tends, at the end of about ten years, to present a similar survival rate to simple tuberculosis. Candidates for work underground have been subjected to a severe medical examination since 1916; since that date only 19 out of some 18,564 new miners have become the subjects of simple silicosis. The great majority of new cases are found among old miners who started work under conditions prevailing before the war; but the production-rate for simple silicosis for the year was over 30 per cent. lower than that of the previous year. The figures for 1925-26 were 490 cases as compared with 364 for 1926-27. The reason is discussed at length why the reduction may be considered statistically satisfactory. Miners' phthisis depends for its occurrence upon the amount of silica dust present, the length of the period of exposure, and the amount of exposure to tuberculous infection. The dust risk has been greatly reduced, and, since 1916, so has the amount of infection present. The total incidence of the disease is contributed to by three groups of miners: (*a*) New miners who have passed the severe medical examination, (*b*) miners at work before this examination was started, and (*c*) other old miners who have worked in mines on the Rand and elsewhere.

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The incidence among new Rand miners rises slightly with years of exposure, but is far lower than that for either of the other two groups (see p. 408). The relative importance of the tuberculous factor in the cases at present arising is held to be steadily increasing as compared with the dust factor; indeed, Dr. Irvine holds that in a majority of cases of apparently "simple" silicosis, an element of tuberculous infection is present from the beginning. Still, however, the incidence of silicosis falls more heavily upon machine miners with special exposure to dust. Once the disease has started continuance at underground work makes little difference in the progress. Compensation is awarded directly the disease is detected and the men may retire or remain at work as they like; but the mortality from silicosis in subsequent years is nearly the same whichever course is chosen. The lesson to be drawn is the importance of the infective factor, whether considered from the standpoint of the further course of cases or of prevention. Whereas dust prevention was a matter for engineers, the prevention of infection is a medical problem. It is upon this problem that efforts must be and are being concentrated. The most recent efforts in this direction are to control and eliminate the cases of tuberculosis occurring among the natives employed, who are ten times more numerous than the Europeans. Initial examination and periodic supervision of these natives is being steadily improved, so that by a sustained and intensive campaign carriers of tuberculosis among mine natives may be brought under control.

Abstracts.

ABSCESS OF THE LUNG.

BRYAN, J. H. **The Laryngological Aspect of Suppurative Inflammation of the Lungs.** *Trans. Amer. Laryngol. Assoc.*, 1928, **49**. 162.

Under the term suppurative inflammation of the lungs are two conditions: bronchiectasis, or suppurative bronchitis, and abscess of the lungs. These conditions, which the writer believes to be definitely on the increase, cannot always be dissociated. At a symposium on streptococcus carriers, which was held in America in 1919, it was clearly established that the tonsils were the carriers of many forms of infection, especially the various types of streptococcus. Careful assembly of reports from the military camps and from the various laboratories, showed that infected tonsils were a source of infections and inflammations in the lower respiratory tract, the infection being carried downwards and producing laryngitis, bronchitis, pneumonia and empyema. The infections may also be carried upwards, producing sinusitis, varying in intensity from a mild catarrhal inflammation to suppuration in one or more of the accessory cavities. Yet another source of suppurative bronchitis and pulmonary abscess is pyorrhœa and abscess at the roots of the teeth. Suppurative inflammation of the lung may also follow influenza, as occurred in a recent case of the writer's.

The operation of tonsillectomy is not infrequently followed by abscess of the lung, mainly, in the writer's opinion, because of undue haste and operating in a bloody field, not sufficient care being exercised in the removal of infected blood and secretions expressed from the tonsil in the process of enucleation. Infected infarctions may be carried into the lungs from the seat of operation, usually owing to undue traumatism.

Diagnosis of suppurative inflammation of the lungs is readily made, in both chronic and acute types, by the foul odour of the breath and the excessive, foul, purulent secretions, frequently hæmorrhagic in character.

Bronchoscopy has shown many brilliant results and should always be tried before resorting to surgery. Artificial pneumothorax in acute conditions is also a valuable method of treatment; where the case responds to neither of these, general surgery must be resorted to.

MANGES, W. F. **Suppuration of the Lung: Roentgenological Aspect.** *Trans. Amer. Laryngol. Assoc.*, 1928, **49**. 167.

Lung abscess should not reach a chronic stage without diagnosis, and

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therefore, early and repeated examinations by X-rays are necessary. By this means it is possible to determine the manner of development of the disease, its rate of increase or decrease, and, more important still, to determine whether it has a tendency to advance towards the periphery or towards the root of the lung. Early X-ray diagnosis is not always easy from a single examination. In the early stage the shadows of exudate are, as a rule, ill-defined, and, if the area involved is small and near the root of the lung, may be entirely overlooked. In other cases it may be mistaken for a pneumonia, or, in the apical region, it may be considered evidence of active tuberculosis. With a series of X-ray studies it may be possible, especially with the aid of other data, to make a diagnosis of lung abscess before a cavity actually develops, but the diagnosis is positive by the X-ray picture alone when there is free air and free exudate in the cavity, so that a fluid level can be demonstrated.

The rate of destruction of lung tissue varies considerably in different cases. In some the infected area becomes more or less walled off; in others the lesion extends progressively into the more distant portions of the lobe or lobes involved; in yet others metastatic abscesses break out successively in different parts of the lung. In all these types repeated X-ray examinations are valuable. Another type of lung abscess is that developing in connection with a foreign body. When this lesion follows the aspiration of non-opaque substances, the X-ray diagnosis is not easy and must depend upon the character of the shadow, its location and distribution. This lesion in appearance and location is not unlike many of the so-called post-tonsillectomic abscesses. The abscess that follows the aspiration route of infection is always in relation to some fairly large bronchial branch, and as a rule is easily accessible to bronchoscopic drainage. The abscess that follows pneumonia is usually centrally located in an area that has recently been the site of a pneumonia. The abscess found near the surface of the lung, isolated from the larger bronchial tubes and not in an area of recent pneumonia, has probably arrived by the lymphatic route; while those that develop in the interior and are in contact neither with pleura nor with root area are probably by way of the blood-stream.

Localisation as a function of the radiologist should be considered: (*a*) with relation to bronchial distribution, and (*b*) with relation to the chest wall, the former being particularly important when treatment is to be by means of bronchoscopic drainage, and the latter when the treatment is to be by way of thoracotomy. Pneumonography is the most dependable method for giving accurate and complete information, the insufflation of dry bismuth being best for localising any lesion or object in relation to a bronchus. Iodised oil is best for the examination of cavities, or for studying the terminal branches of the bronchi and the distal portions of the lungs.

Apart from the functions of diagnosis and of localisation, radiology offers the best means of observing the progress of a case, as well as reliable information for intelligent selection of the best method of treatment—bronchoscopy or surgery. Repeated examinations should always be made in the case of doubtful lesions, to note the changes that take place from day to day, or week to week.

McCRAE, T. Lung Abscess. *Trans. Amer. Laryngol. Assoc.*, 1928, **49**. 174.

The ætiology and treatment of lung abscess are the two aspects of the question of most interest to laryngologists.

Ætiology.—When the abscess is due to a foreign body in a bronchus or is secondary to a bronchial neoplasm the ætiology is obvious, as also when the abscess follows pneumonia, accompanies pulmonary tuberculosis, or is the consequence of an infarction. In many other cases no cause can be established; and frequently when lung abscess has followed operative procedure on the upper air passages—usually tonsillectomy—the true cause of the condition is not recognised. The writer emphasises the importance of a careful examination of the patient before tonsillectomy, so as absolutely to exclude acute respiratory-tract infection or pulmonary tuberculosis. Another question

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for the answering of which extensive study is required is as to the advisability or otherwise of abolishing the cough reflex, administering morphine before operation or sedatives after operation. The presence of spirochætes in the pus is another problem. Are they merely accidental invaders after the abscess has ruptured into a bronchus, or are they of ætiological importance? Careful study of the first pus which is expelled and study of the contents of the abscess obtained through the bronchoscope as soon as possible after rupture should be useful. The value or otherwise of arsenical treatment should also be determined.

Prognosis is always serious, except in cases due to a foreign body. Early skilled bronchoscopic drainage is, in the writer's opinion, one of the most important single points.

Treatment.—Before rupture occurs into a bronchus the problem of external drainage must be decided. Much here depends on early diagnosis. After rupture has occurred bronchoscopic drainage should be tried first, both in acute and chronic conditions, and the earlier it is begun the better it is for the patient. Postural drainage should always be carried out as thoroughly as possible. Creosote, by mouth or by inhalation, is usually worth a trial in chronic abscess. Artificial pneumothorax sometimes does good, but sometimes does definite harm by stopping drainage owing to complete closure of the bronchus with collapse of the lung. In any case, pneumothorax should be used only after the greatest consideration and after bronchoscopic drainage has been given a thorough trial.

TUCKER, G. **The Bronchoscopic Aspect of Suppuration of the Lung.** *Trans. Amer. Laryngol. Assoc.*, 1928, **49**, 179.

Bronchoscopy is helpful both in diagnosis and in treatment of suppurative pulmonary disease. In diagnosis it is valuable in several ways: (1) by giving definite information as to the location and extent of the lesion from direct endoscopic study; (2) by the removal of secretions from the bronchi, uncontaminated by mouth secretions, that will show on culture the infecting organism; (3) by excluding foreign body by direct examination of the larger bronchi (X-ray study and physical examination will be necessary for exclusion of foreign body in the smaller bronchi); (4) by the bronchoscopic introduction of bismuth subcarbonate and lipiodol where the radiologist thinks it necessary. Obstructive secretions and granulations can be removed and the opaque substance introduced into the desired portion of the lung; (5) by removal of tissue from the lesion for histological examination.

With regard to treatment, bronchoscopy will help: (1) by improving bronchial drainage through the aspiration of secretion, and local treatment of the lesion; (2) by securing an uncontaminated specimen of the infecting bacteria for an autogenous vaccine; (3) by dilating strictures and removing obstructing granulations: (4) by removal of foreign body. In suppurative disease of the lung due to foreign body, 93 per cent. of the cases will be cured by the bronchoscopic removal of the foreign body. The bronchial drainage is improved to such an extent in practically every case that no further bronchoscopic treatment is required.

The paper concludes with several case reports, illustrating some of the phases of the value of bronchoscopy in diagnosis and treatment of suppurative pulmonary lesions.

KLINE, B. S. **Pulmonary Abscess and Pulmonary Gangrene.** *Journ. Amer. Med. Assoc.*, 1928, **90**, 2008.

These two conditions are quite distinct, though they are frequently considered and treated as one disease. Pulmonary abscess, which is found more frequently in children than in adults, is caused by pyogenic organisms reaching the lung, either by aspiration, or via the blood-stream from a distant focus of suppuration. Cases of pulmonary abscess at Mount Sinai Hospital, Cleveland, were usually of the aspiratory type, generally with multiple lesions, but with one or more principal areas of suppuration. The lower lobes of the lung were most frequently involved. In

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the embolic type of case the abscesses were small, multiple, involving several lobes, and were associated with abscesses elsewhere, notably in the myocardium, kidneys or brain.

Pulmonary gangrene is a more severe disease, generally affecting adults rather than children, and is caused by a group of organisms—mainly spirochætes, fusiform bacilli, and vibrios—aspirated from the mouth. The differentiation between these two diseases is imperative, since the treatment is essentially different, pulmonary gangrene being amenable to arsphenamine therapy rather than to treatment for abscess. The sputum in the case of pulmonary abscess is mucopurulent or purulent, has no appreciable odour, and cultures and smears show the pyogenic organisms. The sputum from gangrene is foul smelling, and grayish-brown or grayish-green, and shows the characteristic spirochætes, fusiform bacilli and vibrios in smears stained by strong carbolfuchsin solution or by the Fontana method. Both aspiratory abscess and gangrene may follow operations under general anæsthesia.

The writer quotes the clinical and experimental evidence for the embolic origin of pulmonary lesions recently presented by Schlueter and Weidlein. He himself believes, however, that the experiments referred to throw little light on the pathogenesis of the so-called typical lung abscess of man, and he adduces a good deal of evidence suggesting that the organisms reach the lungs by aspiration.

More important, however, than the pathogenesis of the "typical lung abscess" is the actual character of the lesion present and the organisms producing it. In sections of early lesions stained by the Gram method, the abscess of the aspiratory type is readily differentiated from that of the embolic type. In the former, numerous clusters of staphylococci are found in the bronchial branches and in the alveoli, the blood-vessels being uninvolved. In the embolic abscesses, on the other hand, early lesions show clusters of staphylococci in the lumen and walls of the blood-vessels, and none in the regional bronchial branches. The lesion of pulmonary gangrene, when fully developed, is a ragged, foul-smelling, brownish or greenish ulcerated area.

Pulmonary gangrene of spirochætal origin may be prevented by proper oral hygiene and therapeutic measures.

CUTANEOUS TUBERCULOSIS.

DERBYSHIRE, R. **Sur un cas de tuberculose verruqueuse chez l'enfant.** *Thèse de Paris*, 1928, No. 143.

The author, who records an illustrative case in a girl aged 12 years, remarks that verrucose tuberculosis is one of the best known forms of cutaneous tuberculosis and is not very infrequent in childhood. In the primary form it is in most cases the result of exogenous inoculation, while in its secondary form it may be due to auto-inoculation or to dissemination of tubercle bacilli by the blood and lymph. Clinically it is characterised by the development of single or multiple foci of verrucose tuberculosis which are often associated with open or closed scrofulo-tuberculides.

As a rule the general health is not affected, as the condition is merely a form of local tuberculosis which is readily cured by cauterisation or complete extirpation.

BRION, A. **Du traitement chirurgical de certains lupus tuberculeux de la peau.** *Thèse de Paris*, 1928, No. 340.

Brion, who records 30 illustrative cases in patients aged from 4 to 54 years, maintains that cutaneous lupus should be treated by excision pure and simple, followed by immediate suture of the edges of the skin whenever this method is possible, namely under the following conditions: (1) when the lupus is of small or moderate size, (2) when it is situated on a covered part of the body, and the size of the strip of skin removed allows the edges to be sutured, and when it is not confined to special situations such as the nostrils and external ear. The method has the advantage of being rapid, painless, and simple, of not leaving unsightly scars and of rarely being followed by recurrences. When union by first intention appears to be impossible, excision should be followed by the application of a graft

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which should be carried out at once during the same operation as the removal of the lupus.

In Brion's experience it is never beneficial to leave an interval of twenty days between the excision of the lupus and the application of the graft. Whatever the local method employed, general antituberculosis treatment should be instituted. As early a diagnosis of lupus as possible is important, as there is a more likely prospect of a radical cure when the lesion is of small size.

PAVIA, J. L., and DUSSELDORP, M. Un caso de tuberculosis verrugosa del parpado. *Arch. de Tisiol.*, 1928, 3, 41.

The authors, who record an illustrative case, state that tuberculosis of the skin of the eyelids is rarely seen by ophthalmologists, as it may assume any of the forms of cutaneous tuberculosis. It is generally caused by spread of the tuberculous process from neighbouring foci in the nasal fossæ or sinuses, lachrymal tract, conjunctiva, or bones of the orbit. In a few cases the lesion appears to be primary without any connection with a tuberculous periocular affection. Children are more frequently affected than adults. The present case is that of a boy, aged 10, who developed a tender nodule first on the lower and then on the upper lid of the right eye. Apart from enlarged cervical glands no other lesions were found. Histological examination of the nodules showed the characteristic appearance of verrucose tuberculosis. The condition must be distinguished from simple warts which are non-inflammatory, epithelioma which is progressive and shows no tendency to spontaneous cicatrisation, and papular syphilides which have no tendency to suppuration.

TANGUY, R. Essai sur le rôle des infections associées dans la détermination de certaines formes cliniques de la tuberculose cutanée. (Parasitisme spirillaire et tuberculose végétante péri-anale). *Thèses de Lyon*, 1927-8, No. 11.

This thesis is devoted to the description of a special form of cutaneous tuberculosis which is found in the perineo-anal region and is known by the name of vegetative or framboesiform peri-anal tuberculosis. In the case which Tanguy records in a man aged 51, the tubercle bacillus was associated with a rich growth of spirilla, which presented all the characteristics usually seen in ordinary genital vegetations. A spirillar inflammation of this character cannot be regarded as being of a purely saprophytic nature without any action on the tissues. It is now a well-established fact that it acts on the connective tissue and Malpighian layer, causing them to assume a vegetative growth. Vegetative tuberculosis whose site of election is the perineo-anal region appears to be due to the combined action of the tubercle bacillus and spirilla. While the tubercle bacillus by itself is capable of giving rise to a verrucose character of the lesions, verrucose tuberculosis is very different from the ulcero-vegetative lesions of which Tanguy's case is an example.

NICOLAS, J., and LEBEUF, F. Psoriasis et tuberculose. *La Médecine*, 1928, 9, 1062.

The connection, or possible connection, between these two diseases has long been studied, many observers having suspected the tuberculous origin of psoriasis. For several years the two present writers have systematically studied from the point of view of tuberculosis every case of psoriasis which turned up at their dermatological clinic. Each such patient was questioned with regard to his personal and family history, and clinical, radiological, serological (Arloing and Courmont), complement deviation with Besredka's antigen (Calmette and Massol) and Wassermann tests were carried out in each case. The material comprised 60 patients with psoriasis, of whom 20 had a personal or family history of tuberculosis, or were suffering from tuberculous lesions, mostly of a mild type (pleurisy, emphysema, cervical adenitis, white swelling cured by ankylosis, etc.). Two patients, however, had active pulmonary tuberculosis with tubercle bacilli in the sputum. The serological tuberculosis test was positive in all but one of the psoriasis patients, contrasting with the

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50 per cent. of positive tests in other dermatoses. Complement deviation was positive in 35 out of the 60 cases. The Wassermann reaction was positive in three cases only, two of these patients having contracted syphilis long after the psoriasis had appeared.

The writers conclude that clinical evidence showed the frequency of tuberculous antecedents or of tuberculous manifestations in patients with psoriasis; and that laboratory evidence also gave positive results which, in spite of their equivocal interpretation were, nevertheless, very much greater in psoriasis than in other dermatoses. Although at the present time there is no absolute proof of the tuberculous ætiology of psoriasis, the writers' investigation appears to confirm the existence of some definite relationship between the two diseases.

CENTRAL AND LOCAL AUTHORITIES.

MINISTRY OF HEALTH.

TREATMENT OF TUBERCULOSIS. CIRCULAR 969.

Facilities have been provided, with the approval of the Minister of Health, by the National Association for the Prevention of Tuberculosis at their institution at Burrow Hill, Frimley, Surrey, for the treatment and technical education of tuberculous youths between the ages of 14 and 19 years.

The Minister is advised that, in certain cases where tuberculosis is contracted by youths within this age period, the condition responds well to treatment and the prospects of complete arrest of the disease are frequently more favourable than in adults. But as the age period from 14 to 19 is normally that in which remunerative occupation is begun and any necessary training undertaken, the prolonged treatment necessary to secure the arrest of tuberculosis is, if the disease is contracted during that period, a serious handicap which prevents the patient from competing for work on equal terms with other youths of the same age. Even when treatment is brought to a successful conclusion, it has necessarily occupied time which the patient's competitors have employed in securing practical experience and increasing their value in the labour market.

In this class of case, therefore, the onset of tuberculosis imposes a special economic disadvantage which is not adequately met by successful treatment of the disease: and the arrangements made by the National Association for the Prevention of Tuberculosis accordingly include provision for technical education to be given during the course of treatment, so that, on the patient's discharge, this special disadvantage may be as far as practicable reduced and possibly in some cases eliminated. It is to be noted that a large proportion of tuberculous youths of this age will not have learned any trade or occupation prior to admission to Burrow Hill, and the problem of providing technical education is therefore simplified. Since it has not been necessary to take into account the variations of practical experience which would be found among patients of more advanced age, it has been possible to concentrate upon two main types of technical education (gardening and clerical work), and to make provision for a more practical and thorough teaching of these two subjects than could be given if it were necessary to provide a more varied range of instruction.

Further particulars of the arrangements made, and a fuller indication of the type of patient for whom they are designed, are given in a brochure which, it is understood, has been circulated by the National Association for the Prevention of Tuberculosis to Local Authorities responsible for tuberculosis schemes.

It is appreciated that, in the area of any one Tuberculosis Authority, the number of cases of the special class under consideration will be comparatively small, and that it would therefore be difficult for any one authority to make adequate arrangements to deal with such cases specially in one of its own institutions. It seems, therefore, preferable that these special cases should be dealt with in an institution organised for this purpose, and the Minister hopes that Tuberculosis Authorities will appreciate the advantages of the arrangements which have been made at Burrow Hill, and that, by making use of these facilities for any cases of this class which may occur in their areas, they will co-operate with the National Association for the Prevention of Tuberculosis in dealing with this difficult problem.

It should be noted that these arrangements are designed for a special type of case and will be more effective if confined to the treatment of that type, and that a patient must ordinarily be prepared to stay in the institution for a period of twelve months or longer if a satisfactory result is to be secured. The selection of patients for this institution must therefore be made with particular care.

Finally, it is of great importance, if the maximum of benefit is to be obtained from the scheme, that the Tuberculosis Care Committee, or other suitable local organisation, should make every effort, when a youth has been admitted to Burrow Hill, to secure that suitable employment is available for him on his discharge from the institution.

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SOCIETIES.

THE TUBERCULOSIS ASSOCIATION.

A meeting of the Association was held on Friday, March 15, at the British Medical Association House. The President, Sir StClair Thomson, was in the chair. After the transaction of the necessary business, papers were read by Dr. L. S. T. Burrell and Dr. F. R. G. Heaf on "Sanocrysin Treatment."

Dr. Burrell began by describing his technique and dosage. The starting dose was 0·1 grm.; if there was no reaction, 0·25 grm. was given three days later, and then at weekly intervals 0·5 grm., 0·75 grm., and 1 grm., this last dose being repeated three or four times at weekly intervals. In the case of children, small women and very delicate patients, the maximum dose was usually 0·5 or 0·75 grm. The question was sometimes asked whether it was the sanocrysin itself or the associated shock that did the good. The effect could not be due entirely to the latter, since neither tuberculin nor serum given in increasing doses till shock was produced brought about the good results that were seen in the case of sanocrysin. Under sanocrysin treatment tubercle bacilli disappeared from the sputum, and the sputum itself became less and less. Two illustrative cases were cited of children treated at Brompton Hospital, in whom injections of sanocrysin brought about both these results—disappearance of tubercle bacilli and greatly lessened sputum. The ideal case for treatment was the young patient (about the age of puberty) with acute disease. In cases of continued slight pyrexia sanocrysin was useful in reducing the temperature which tended to stay down: but this effect was probably not due to the sanocrysin *per se*, since other drugs had the same effect. Another type of case that sometimes did very well with sanocrysin was the patient with chronic fibroid disease with acute exacerbation.

With regard to complications Dr. Burrell said that serious gastro-enteritis and stomatis could be avoided by giving small doses, but it had to be remembered that if very small doses were given no results could be expected. Rashes were not uncommon; they occurred in 11 of the speaker's first 60 cases, and in some of these they were very severe, but the pulmonary condition of each of these patients showed definite improvement. Albuminuria was nearly always present, but this might be safely ignored unless it was very severe. On the whole, the dangers and complications of sanocrysin treatment were so slight as to be negligible, provided the doses were small enough and were well spaced.

Dr. Heaf who followed said that he had treated by this method 97 cases of pulmonary tuberculosis. These cases included 87 adults and 10 children in all stages of the disease, except the very advanced; the adults all had positive sputum, and eight of the ten children showed definite clinical signs of tuberculosis. The interval between the injections now tended to be lengthened, and the weight of the patient was used as a guide in treatment. The dosage he employed was 0·1, 0·2, 0·35, 0·5, 0·6, 0·75, 0·8, 1 grm. Any shock reaction was carefully avoided. In a good proportion of cases they were successful in decreasing the physical signs, in freeing the sputum from tubercle bacilli and in clearing the X-ray picture. A decrease in the sedimentation rate of the red corpuscles was also noted, and a general rise in the weight curve of the patient. There was a difference of opinion as to suitable cases. In his own opinion *any* case was suitable provided the renal and cardiac systems were normal, the physique and general condition good, and no abdominal symptoms were present. It was, however, waste of money to treat Stage III cases with sanocrysin as the results were not permanent. Even in Stage II cases relapses were also common, and the results were not as good as in a series of control cases treated by artificial pneumothorax. The dosage of sanocrysin in the case of children was very small and the increase very gradual, special regard being paid to weight. All the children in his series had had long periods of treatment, and the weight and appetite improved and the sputum diminished. Sanocrysin appeared to be well tolerated by children.

In the discussion which followed the Chairman said he was impressed with the trouble and care taken by the two speakers. He asked whether laryngeal complications were a contra-indication to sanocrysin treatment. Dr. Andrew Morland said that rashes could be most alarming. In one such case he had persisted with the treatment without ill effects. With regard to albuminuria he recalled that Dr. Gravesen had reported two or three cases in whom the albuminuria had never cleared up. He himself had had a case in which a small dose of sanocrysin caused marked albuminuria which had never stopped, although this patient had improved in other respects and was back at work. Another speaker said that in a first series of very small doses there had been severe stomatitis and rashes, but that in a second series with larger doses there had only been one case of vomiting and one of eczema. He now made it a routine procedure to give 50 per cent. glucose before injecting sanocrysin.

Dr. Vere Pearson said that he thought the proper spacing of doses was very important. Sodium thiosulphate he looked upon as a useful antidote for stomatitis or skin rashes. He doubted whether sanocrysin should be used in cases that were doing well without it: but he suggested that too much distinction was

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made between the exudative type and the fibroid type as far as sanocrysin was concerned. Another speaker reported that even small doses of sanocrysin had been followed in one of his cases by intractable diarrhœa; and in another case a rash and albuminuria supervened after a dose of 0·5 grm. sanocrysin. These cleared up, but after another dose of sanocrysin the albumin returned and never cleared up again.

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Statistische beschouwingen naar aanleiding van het artikel van Oudendal over de bepaling van de prognose bij tuberculose. J. Sanders—p. 525.

March 30.

Over de polymorphie van den tuberkelbacil. A. Klein—p. 562.

New England Journal of Medicine.
March 21.

Tuberculosis of the Spine, with Particular Reference to Fusion of the Vertebral Bodies. H. Ragolsky—p. 668.

April 11.

Tuberculous Meningitis with an Unusual Cerebro-spinal Fluid. J. W. Watts and H. R. Viets—p. 757.

Norsk Magasin f. Laegevidenskapen.
February.

Parallellunder sokelser av det leukocytaere blodbilledo og senkningsreaksjonen ved barnetuberkulose. H. Bjelke—p. 81.

April.

Lokal lysbehandling av larynxtuberkulose. R. Moe—p. 346.

Om sanatoriebehandlede plevrittpatienters, skjebne. L. B. Smith—p. 414.

La Pediatria.—April 15.

Influenza dei raggi ultravioletti sulla cutireazione alla tubercolina. L. Storchi—p. 416.

Il Policlinico (Sez. Prat.).—April 8.

Contributo allo studio della tubercolosi muscolare ematogena. G. Corradino—p. 482.

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Temperatura del gas usato per lo pneumotorace artificiale. L. Vercelli—p. 596.

Post-Graduate Medical Journal.—May.

Fibrosis of Lung in Children. L. S. T. Burrell—p. 140.

La Prensa Medica.—March.

Cartillas de higiene y de profilaxis antituberculosas. L. P. Romaguera—p. 13.

Presse Médicale.—March 16.

Traitement de la tuberculose par l'allergine. A. Jousset—p. 353.

La morrhuate de cuivre colloidal dans le traitement de la tuberculose. J. Mouzon—p. 358.

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La tuberculose et les agents des postes. G. Poix—p. 411.

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Tuberculose et dengue; étude clinique. N. Chaniotis and E. Scordombekis—p. 419.

April 17.

Une méthode de contrôle anatomique des radiographies pulmonaires. P. F. Armand-Delille, C. Lestocquoy and J. Vibert—p. 504.

Los Progresos de la Clinica.—March.

La Conferencia para el estudio de la vacunación antituberculosa con el B.C.G. y nuestras observaciones y resultados durante cuatro años de aplicación al niño. L. Sayé—p. 145.

Rassegna di Clinica, Terapia e Scienze Affini.—January-February.

Emostatico ed emotonico in diverse forme di tubercolosi polmonare. F. Pistolese—p. 27.

Calcioterapia emostatica. A. Pomodoro—p. 35.

Cura di calcio nelle malattie oculari da rachitismo e scrofolosi. D. Bruno—p. 39.

Recueil de Médecine Vétérinaire.—March.

Six années de prophylaxie de la tuberculose bovine par le B.C.G. dans une exploitation infectée (1922-28). P. Brinet—p. 134.

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Revista de Higiene y de Tuberculosis.
JANUARY 31.

De la tuberculosis del peritoneo. V. JUARISTI —p. 1.

Formas anatómicas de la tuberculosis gastrica. URRUTIA—p. 5.

El régimen alimentico en las diferentes localizaciones tuberculosas. J. L. YÖGUE Y ESPINOSA—p. 7.

La origenoterapia en la peritonitis tuberculosa. J. CHABÁS—p. 20.

Revista Médica de Barcelona.—FEBRUARY.

Sobre el diagnóstico de las micosis pulmonares primitivas. C. XALABARDER and E. ALEGRET —p. 122.

Revista Médica Latino-Americana.
FEBRUARY.

Signos radiológicos que permiten el diagnóstico de la parálisis diafragmática cuando ésta existe simultaneamente con neumotórax. A. VADONE—p. 489.

La serorreacción de floculación de Vernes en las tuberculosis osteoarticulares. J. VALLS and V. C. GIRARDI—p. 506.

Revista Oto-Neuro-Oftalmológica y de Cirugia Neurológica.—FEBRUARY.

Tuberculoma de la calota peduncular. M. R. CASTEX, H. J. MOLLARD and A. F. ARNAUDO —p. 51.

Revue Médicale Française.—MARCH.

Les images radiographiques en bec-de-perroquet dans le mal de Pott. C. CLAVELIN and A. SICARD—p. 209.

Les rayons ultra-violets en bains généraux dans le traitement des affections oculaires d'origine dite "scrofuleuse." L. DELHERM and MOREL-KAHN—p. 225.

Riforma Medica.—MARCH 23.

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Rivista di Clinica Pediatrica.—MARCH.

Contributo allo studio dell'intradermoreazione con siero di cavallo (Busacca) nella tubercolosi dell'infanzia. B. BENVENUTI—p. 187.

Rivista di Patologia e Clinica della Tubercolosi.—MARCH 31.

La questione della trasmissione transplacentare dell'ultravirus tubercolare e considerazioni sui suoi presumibili rapporti colla patogenesi di alcuni quadri morbosi della tubercolosi. L. AJELLO—p. 173.

Ricerche sul valore del procedimento culturale all'acido solforico nella diagnosi dei processi tubercolari. C. VERDINA—p. 190.

Su alcuni rari effetti dannosi del pneumotorace artificiale. A. CAMPANI—p. 202.

Stato attuale della questione della vaccinazione preventiva della tubercolosi. A. OMODEI ZORINI—p. 209.

Il ricambio azotato e minerale nella tubercolosi polmonare. G. GHERARDINI—p. 219.

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La roentgentherapie de la tuberculose de la langue. A. EGHIAYAN—p. 463.

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Terapia de sales metálicas en la tuberculosis pulmonar (tratamiento estimulante, tónico. de acuerdo con los principios biológicos experimentales sustentados por el Dr. L. E. Walbum). N. LUNDE—p. 771.
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El estado actuel en los tratamientos modernos de la tuberculosis. A. LAMADRID—p. 835.

Southern Medical Journal.—APRIL.

Influence of Progenic and Tuberculous Infections upon Epiphyseal Activity and Growth of Bone. A. COTTON—p. 303.

A Report of the Study of 25,048 School Children for Tuberculosis. P. C. MCCAIN—p. 310.

Surgical Clinics of North America.—FEB.

Tumor in Right Submaxillary Region (Noncaseous Tuberculosis). F. W. RANKIN and C. L. CHUMLEY—p. 102.

Pulmonary Tuberculosis. S. W. HARRINGTON —p. 138.

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The Surgical Treatment of Pulmonary Tuberculosis. C. A. HEDBLOM—p. 307.

Tuberculosis—Cavity—Pneumolysis. R. B. BETTMAN—p. 465.

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Den moderna uppfattningen om lungtuberkulosens uppkomst och utveckling. E. FAGERSTRÖM *et al.*—p. 17.

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Neue Gesichtspunkte bei der Fachbegutachtung Lungenkranker in bezug auf Kriegsdienstbeschädigung. K. H. BLÜMEL—p. 78.

U.S. Veterans' Bureau Medical Bulletin.

MARCH.

Report of Fifty Autopsies in which the Primary Cause of Death was Pulmonary Tuberculosis. A. O. SAUNDERS—p. 178.

Urologic and Cutaneous Review.—APRIL.

Benzyl-Cinnamic Ether in the Treatment of Cutaneous Tuberculosis. J. LUCAS—p. 243.

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Sobre la vacunación contra la tuberculosis en el hombre. Reflexiones ortodoxas de un heterodoxo. PLA Y ARMENGOL—p. 212.

El pneumo-peritoneo como accidente del pneumotórax artificial. J. MARTOS—p. 242.

Wiener klinische Wochenschrift.

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Indikation und Durchführung der Tuberkulinbehandlung. H. POINDECKER—p. 450.

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Röntgendiagnostik gutartiger Formen tuberkulöser Wirbelerkrankung (Spondylitis tuberculosa benigna). M. SGALITZER—p. 562.

Wiener medizinische Wochenschrift.

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Ueber Verkalkung von Riesenzellen bei einem Fall von Tuberkulose der Hypophyse. E. LÖFFLER—p. 474.

Zur Kenntnis der Tuberkulose des Wurmfortsatzes. H. POPPER—p. 478.

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Ueber die trockene fibröse (fibrozystische) Form der Knochentuberkulose an Gelenken. R. KIENBÖCK—p. 544.

Zeitschrift f. ärztliche Fortbildung.

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Ueber die Bedeutung des "Frühinfiltrats" für die Entwicklung der Lungenphthise des Erwachsenen. A. GÉRONNE—p. 205.

Zeitschrift f. Hals-, Nasen- und Ohrenheilkunde.—Bd. xxiii, Heft 1.

Fremdkörper-Veränderungen der Kieferhöhlenschleimhaut, die eine Tuberkulose vortäuschen können. A. ESCH—p. 29.

Zeitschrift f. Immunitätsforschung.

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Zum Tuberkulinproblem. J. BAUER and S. LEVY—p. 240.

Zeitschrift f. Infektionskrankheiten, parasitäre Krankheiten und Hygiene der Haustiere.—Bd. xxxv, Heft 1.

Hauttuberkulose des Rindes. E. SICKMÜLLER—p. 70.

Zeitschrift f. Kinderheilkunde.

Bd. xlvii, Heft 2.

Zur Klinik der paratuberkulösen serösen Pleuritis. H. G. HUBER—p. 169.

Zur Bedeutung des vegitativen Nervensystems für die Klinik der Kindertuberkulose. A. KROVITZKY and D. SCHENKMANN—p. 248.

Zur Frage der Herdreaktion bei Tuberkulose. H. KOCH—p. 265.

Zeitschrift f. Kreislaufforschung.

Bd. xxi, Heft 5.

Kreislauf und Lungentuberkulose. J. PORAS—p. 121.

Zeitschrift f. orthopädische Chirurgie.

Bd. li, Heft 3.

Die reelle Verlängerung der Extremitäten bei der Knochen- und Gelenktuberkulose. S. TSKUHOW—p. 282.

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Zeitschrift f. Tuberkulose.
Bd. lili, Heft 2.

Die neuen Anschauungen über die Entwicklung der Lungenphthise und die Auswirkungen auf die praktische Tuberkulosebekämpfung. G. LIEBERMEISTER—p. 97.

Neurasthenia peripherica localis. J. PETRUSCHKY—p. 102.

Dengufieber und Lungentuberkulose. B. PAPANIKOLAU—p. 107.

Ueber prä- und intramenstruelle Temperatursenkungen, besonders bei Tuberkulose. S. KAUFMANN—p. 112.

Zur Frage des Bazillennachweises im tuberkulösen Sputum mittels der Dunkelfeld-Methode. S. v. STUBENRAUCH—p. 122.

Das aus Meerschweinchenlunge gewonnene fettspaltende Ferment und dessen Wirkung auf den Tuberkelbazillus. D. KANOCZ—p. 124.

Paul von Baumgartens Anteil an der Tuberkuloseforschung. A. DIETRICH—p. 128.

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Die intravenöse Kalkbehandlung der Lungentuberkulose. F. BECKER—p. 198.

Kalkinhalation bei Lungentuberkulose nach dem System Lax-Zeyen. H. SIEGEL—p. 206.

Thorakoplastik und Geburt. F. JESSEN—p. 214.

Ueber die biologische Reaktivität der Tuberkelbazillenfette. L. M. MODEL and E. F. SSIDELNIKOWA—p. 227.

Die Massnahmen und Einrichtungen der Landesversicherungsanstalt Sachsen im Kampfe gegen die Tuberkulose, unter besonderer Berücksichtigung der neueren Forschungsergebnisse. ZWERG—p. 234.

Tuberkulose-Krankenhäuser. K. HENIUS—p. 240.

Aerztlicher Jahresbericht der Prinzregent Luitpold-Kinderheilstätte Scheidegg für 1928, nebst diagnostischen und therapeutischen Bemerkungen. K. KLARE—p. 281.

NEWS, APPOINTMENTS AND DIARY.

News.

The National Association for the Prevention of Tuberculosis have, with the approval of the Ministry of Health, adapted their Sanatorium Colony at Burrow Hill, Frimley, so as to render it available to all local authorities in England and Wales who may desire to utilise it for the long period treatment and technical education of suitable tuberculous youths between the ages of 14 and 19. The Institution contains 80 beds, 60 of which are provided for arrested cases, and 20 in a specially designed block for patients in the earlier stages of the disease. Although primarily intended for youths suffering from pulmonary tuberculosis, cases of glandular tuberculosis without sinuses, or of tuberculous disease of bones and joints no longer requiring active orthopædic treatment, will also be eligible for admission.

Technical education in gardening and clerical work forms an essential part of the régime, and thus only those who are physically and temperamentally suitable for such education can be accepted.

Negotiations are in progress with a view to the school being recognised by the Board of Education. The duration of stay aimed at should be one year or longer. The cost of maintenance will be 50s. per week inclusive.

The scheme, which is an experimental one and one which so far has not been undertaken in this country, has been approved by an Order of the Charity Commission, provided that preference is given to the dependants of ex-service men.

Applications for admission should be sent as soon as possible to the Medical Superintendent of the Institution.

The Indian, a monthly medical journal, published in Madras, is this year celebrating its silver jubilee. The April number is very considerably enlarged to form a special number. In addition to the special articles reviewing the work of the journal through a quarter of a century, the number contains twenty-eight original articles, covering a very large field. Two of these articles deal with tuberculosis, viz., "Recent Advances in our Knowledge of the Causation and Prevention of Tuberculosis," by Rao Bahadur Dr. M. Kesava Pai, M.D., and "Abdominal Tuberculosis," by S. Subba Rau, B.A., M.B., M.R.C.S.Eng., D.P.H.Camb.

Appointments.

The undermentioned appointments have been notified to the Minister of Health during the month ended March 31, 1929:—

(1) TUBERCULOSIS OFFICERS.

Kent: Dr. T. R. Cole, T.O., vice Dr. J. M. Clements (resigned). *Nottinghamshire:* Dr. G. G. Buchanan, T.O., vice Dr. C. Kingston, (resigned). *Blackburn:* Dr. I. M. MacLachlan, T.O., vice Dr. G. C. F. Roe (resigned). *Manchester:* Dr. G. V. Ashcroft, Consultant T.O. (part-time), vice Dr. N. C. Haring (deceased). *Smethwick:* Dr. C. Cookson, Asst. T.O. and Deputy Medical Superintendent of Sanatorium, vice Dr. A. G. M. Severn (resigned).

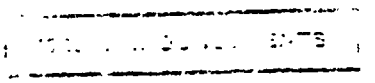
(2) MEDICAL OFFICER OF HEALTH (WHOLE-TIME).

Catherington R.: Dr. H. Stanley.

Diary.

The provincial meeting of the Tuberculosis Association will be held at Cambridge from Thursday, June 27, to Saturday, June 29. Particulars may be had from the Secretary, Dr. C. T. Hebert, St. Thomas's Hospital, S.E.

S. Roodhouse Gloyne, The Presence of the Asbestos Fibre in the Lesions of Asbestos Workers (1929)
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