the claims made regarding the sterilizing powers of certain well-known mercurials are dependent upon unsound methods of bacteriological investigation. He found that in a series of 120 surgical operations the cutting instruments revealed 12 per cent contamination with spore-forming anærobes, and that eight per cent of the knives used for making the skin incisions were contaminated with *Clostridium welchii* or similar organisms.

Special Article

LEPROSY AND TUBERCULOSIS

A lecture delivered to the post-graduate course in tuberculosis organized by the King George Thanksgiving (Anti-tuberculosis) Fund, Calcutta, February 1939.

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I ATTEMPT in this lecture to discuss some of the ways in which these two diseases resemble each other or else differ from each other.

Ætiology

Both these diseases are caused by organisms which are classified under the group 'mycobacterium'. It might be interesting for a moment to go back to the early days of bacteriology and remind ourselves how these two bacilli were discovered. In the seventeenth century, Kircher had stated in a primitive way the theory of contagion and infectious disease. A little later van Leeuwenhoek had seen bacteria, bacilli and spirilla in smears taken from the teeth, but he did not connect such bodies with disease. It was nearly a century later that von Plenciz advanced the idea that minute bodies might convey diseases from person to person and that each infectious disease had its own causative agent, but he did not state what these agents could be. Nearly three-quarters of a century later still, these early theories began to bear fruit. In 1836 Schwann showed that putrefaction was caused by a living body which could be destroyed by heat. About the same time the nature of yeasts was discovered. In 1840 Henle surmised that disease was spread by living infective bodies. In 1846 Klenche showed that tuberculosis could be transmitted in some unknown way by cows' milk, and twenty years later Villeman showed that tuberculosis was caused by an invisible living agent which was transmissible. In the same year, 1865, Davaine discovered the bacillus of anthrax in sheep. In 1870, bacteriology was in its early infancy. Few people believed that bacteria had anything to do with disease. Diseases were attributed to all kinds of other causes and no bacteria pathogenic to man had been seen or described.

In 1870, a young man, Armaur Hansen, was studying leprosy in Norway. He concluded that leprosy was infectious and was probably caused by a micro-organism, and he was searching in microscope preparations made from leprous lesions for the causative organism of the disease. He had poor microscopes compared with our modern instruments, and he had no aniline dyes with which to stain the bacilli. He stained the specimens with osmic acid and he saw bodies which he thought must be the organism of leprosy. This was the first organism to be reported as occurring in the tissues of man. This was one of the great landmarks of medicine and an achievement for which full credit has not been given to Hansen. Hansen published his results in 1873 and practically no one believed him. His preparations were not good enough to convince the sceptics.

In the same year, 1873, a young doctor, Robert Koch, was working in a country practice in Germany after serving in the Franco-Prussian war. In his spare time he studied the bacillus of anthrax which had been discovered in sheep 8 years before. He observed and described the different forms of this organism and its development. A few years later, while still doing country practice, he studied the staining of bacilli by aniline dyes which had recently been used in histology, and found that bacteria stained very well with these dyes.

Later, Hansen adopted Koch's method of staining bacilli and was able to make preparations of the leprosy bacillus so good that the sceptics were convinced and Hansen's bacillus assumed its proper place as the cause of leprosy.

A few years later Robert Koch, now an officer of the Imperial Health Department of Germany with a laboratory and staff at his disposal, turned his attention to tuberculosis and was able to demonstrate a bacillus practically identical with Hansen's bacillus.

Thus, in the early days of bacteriology leprosy research and tuberculosis research helped each other and so we hope will continue to help each other. Koch cultured the organism of tuberculosis and infected experimental animals and thus opened a vast field for research. The leprosy bacillus which was discovered first was soon left far behind and the bacillus even to-day has probably not been cultivated and no experimental animal has been infected. All attempts to apply to the organism of leprosy the refinements of cultural technique which have been worked out so thoroughly in tuberculosis have so far failed.

Immunology

As the result of developments in bacteriology there have been developments in knowledge of immunology of these two diseases. In tuberculosis we have various forms of the tuberculin test the most useful of which is the Mantoux test. It is interesting to note that in leprosy we have rather a similar test, the leprolin test. Leprolin is prepared not from cultures, because we have none, but from leprous lesions themselves. There are certain important differences between the leprolin test and the tuberculin test, but I cannot discuss these now.

In tuberculosis much work has been done in recent years on complement fixation and similar work has also been done in leprosy. For complement fixation in leprosy we find that the best available antigen at the present moment is what is known as the WKK antigen which is prepared from tubercle bacilli, and there is considerable evidence to show that the immunological reactions of these two diseases have very much in common and that the two bacilli are antigenically related.

Transmission

In transmission, however, we find that there are considerable differences between the two diseases as well as certain similarities. Let us deal with the differences first.

Tuberculosis is transmitted either by inhalation or by ingestion of tubercle bacilli. There is however no proof that leprosy can be transmitted by either of these methods, and it seems most likely that it is transmitted by inoculation of bacilli into broken skin or mucous membrane.

Let us consider resemblances in transmission. Leprosy and tuberculosis are both very often diseases of families, and in both diseases, in the past the possibility of intra-uterine transmission has been discussed. In leprosy it has been shown that a leprous woman whose tissues may teem with lepra bacilli can become pregnant and bear a child, and that lepra bacilli may be numerous in the placenta, may be found in the umbilical cord, and even occasionally in the tissues of the new-born child, but that, if the child is separated from the mother at birth, it does not develop leprosy. I believe that similar findings have been made in tuberculosis and it appears that neither of these two diseases can be transmitted *in utero*.

Another point in which the transmission of the two diseases is similar is in the ease with which they are transmitted to children and young people and the relative difficulty with which they are transmitted to adults. This is a matter about which I shall say more later.

Clinical and pathological findings

Clinically, there are of course very marked differences between leprosy and tuberculosis. Tuberculosis affects the internal organs very much more than leprosy, which affects chiefly the skin, nerves, and mucous membranes. Leprosy does affect some of the internal organs but only to a relatively mild degree. It is, however, interesting to note that the two internal organs most commonly affected in tuberculosis, namely, the lung and the intestinal tract, are rarely if ever affected in leprosy.

It is in the skin that leprosy may produce lesions very much like the lesions of skin tuberculosis or the tuberculide. These leprous lesions in the skin resemble so closely the skin lesions of tuberculosis that they have been called tuberculoid leprosy, or leprides. There are, however, certain clinical differences, the most important being that in the leprous lesions of this variety there is impairment of skin sensation and often thickening of cutaneous nerves supplying the patch. This nerve thickening may extend up to the nerve trunk. When you get a case of suspected tuberculide you should consider whether it may not be due to leprosy. Test the sensation of the skin of the patch and examine for thickening of nerves.

When leprous lesions of this variety were first described, some workers refused to believe that they were due to leprosy and thought that they were tuberculous lesions occurring in a leper. Now, it is generally accepted that they are due to leprosy. In some countries they are apparently much more commonly seen than in other countries, in India they are very common indeed.

Another way in which the clinical aspects of these two diseases show some resemblance is in the occurrence in both diseases of allergic reactions with a temporary increase in the signs and symptoms.

You will have heard about allergic reactions in tuberculosis and their significance, how they may produce a temporary increase in the clinical signs, and how the occurrence of these reactions is not necessarily a bad sign for in some cases (but perhaps not in all), allergy goes hand in hand with immunity, and allergic reaction may be followed by quiescence and arrest of the disease. Similar reactions are also seen in leprosy and they may produce alarming symptoms which, however, always subside in time without any special treatment. These reactions are not infrequently followed by long inactivity and sometimes by arrest of the disease. The failure to recognize allergic reactions in both leprosy and tuberculosis, and the failure to attribute to these reactions their proper significance, is one of the commonest causes of errors of clinical judgment in dealing with these two diseases.

Another way in which the two diseases resemble each other is by the occurrence in both of them of cold abscess. Tuberculous cold abscess and its features are well known to every one, but not many clinicians know that leprosy may cause cold abscess.

It is in the type of leprosy which has been called tuberculoid that cold abscess is sometimes seen. It occurs in nerves, sometimes in the cutaneous nerves supplying tuberculoid lesions

in the skin, and sometimes in nerve trunks. In cutaneous nerves it occurs in the form of small round or oval swellings which may be multiple. In nerve trunks it occurs first in the form of a fusiform swelling usually single, but as the process of caseation progresses the cold abscess may burst through the nerve sheath and form round or oval encapsulated swellings alongside a nerve which may attain the size of a hen's egg, or may track quite a long distance down a limb. Such abscesses sometimes discharge through the skin.

While I am discussing the clinical aspects of the two diseases I might emphasize the fact that leprosy itself rarely causes death, and that particularly in leprosy institutions, one of the commonest causes of death is tuberculosis of the lungs. Thus, you may have leprosy and tuberculosis in the same patient and this may cause a little difficulty in correct diagnosis. It should be remembered that in advanced cases of leprosy, lepra bacilli may be found in the sputum and in the fæces. They usually come not from the lungs but from the mucous membranes of the larynx, pharynx and the trachea. Thus, acid-fast bacilli in the secretions and excretions of a leper may be lepra bacilli or they may be tubercle bacilli. It is impossible to differentiate one from the other merely from the appearance of the individual bacillus, but the fact that lepra bacilli are usually more numerous and occur in masses may be of assistance. Also it is to be remembered that in such cases definite signs in the lung are usually caused by tuberculosis, for leprosy rarely if ever affects the lung.

In some cases, however, guinea-pig inoculation of bacilli is necessary for accurate diagnosis, the guinea-pig not being susceptible to the leprosy bacillus.

Epidemiology and Control

I will now discuss certain points in the epidemiology of these two diseases. Some striking resemblances and some marked differences will be seen. Modern thought has emphasized the idea that all diseases tend to occur in epidemics. They are introduced into a community; they spread to begin with usually in a relatively severe form; the incidence rises to a peak and then declines, the diseases appearing in a mild form, and finally the epidemics die out, though further epidemics may occur later. The factors causing this decline of epidemics are very uncertain.

The acute diseases have short epidemic periods, perhaps only a few weeks or months, *e.g.*, cholera, plague. The more chronic diseases have longer epidemic periods. It is suggested that tuberculosis, a chronic disease, has a very long epidemic period, possibly two or three hundred years or more from start to finish. What evidence is there to support this view of the epidemic nature of tuberculosis ?

A study of tuberculosis in western Europe gives us the following ideas :—

(*a*) Tuberculosis has been declining in western Europe for many years. The mortality rate in England and Scotland has fallen 75 per cent in the last 50 years.

(*b*) It appears almost certain that the decline started long before the infectious nature and mode of transmission of the disease were generally realized, long before the bacillus was discovered, and long before any anti-tuberculosis work started. The decline started and continued during the period of rapid industrialization which would seem to favour the spread of tuberculosis.

(*c*) Although anti-tuberculosis work has not caused the decline, it has probably accelerated it markedly, and it is noteworthy that the temporary deterioration in environmental conditions and the temporary hold up of preventive work which occurred during the European war was accompanied by a temporary increase in the tuberculosis rate. This is a strong indication of the value of hygiene and anti-tuberculosis work, but the decline is due largely to other factors, some of which are beyond control.

The following factors have been suggested as tending to produce a decline in the tuberculosis incidence :—

(1) Increase in racial resistance caused by the elimination by death of susceptible stock, (2) the fall in the birth rate and the increased length of life with marked changes in the age distribution of the population, children forming a very much smaller proportion of the population than in the past. The morbidity and mortality rate of tuberculosis is always highest in young people but if the young people in the population are few, the tuberculosis rate falls correspondingly. (3) Marked improvement in social and hygienic conditions, nutrition, housing, etc.

These ideas have frequently been expressed about tuberculosis in western Europe. I now want to ask the question, 'Does leprosy occur in epidemics which decline spontaneously as the result of factors largely beyond control' ?

There is undoubtedly some evidence that it does. When we study the history of leprosy we find that Europe experienced an epidemic which lasted in all for over 1,000 years. In the thirteenth century, leprosy was probably about as common in England as it is in India to-day, but 400 years later it had almost disappeared. All kinds of reasons have been given for this strange phenomenon, but none of them is satisfactory and it seems probable that it was the natural decline of an epidemic possibly accelerated by segregation of lepers and by improved social and hygienic conditions and nutrition. Now let us consider India. Are these diseases occurring in the form of long-period epidemics and if so at what phase of the epidemic are we at present ?

When considering this question it should first be stated that the same disease may occur in

different forms in different parts of the world. In some places, plague, for example, appears in epidemic form and then disappears entirely, while in other places it is persistently endemic. The same may possibly be true of leprosy and tuberculosis; they may be epidemic in some parts and persistently endemic in other parts. What can cause these differences is not clear.

There are, however, some indications that these two diseases are occurring in India in the form of long-period epidemics, and there are certain things which suggest that the epidemic of leprosy may be past its height, while the epidemic of tuberculosis may be now on the up grade. The available evidence is based on information concerning the incidence and severity of the two diseases.

Let us first consider leprosy. Statistics in India are very inadequate and unreliable, but such as they are, they do not suggest that the incidence of leprosy is increasing in India as a whole. The number of lepers reported in India in the census of 1871 was about the same as was reported in 1921 in spite of the very large increase in population during this period. (Recent census figures are nearly 50 per cent higher probably as the result of more accurate enumeration.) Another point is that work in recent years has shown, I think, conclusively, that the average case of leprosy seen in India is much milder than the average case seen in some other countries. These two facts, the mild form of the disease and the available statistical evidence, poor though it is, suggest that leprosy may be past the epidemic peak and may be on the downward grade. This does not mean that anti-leprosy work is not needed in India. On the other hand it may mean that conditions are favourable and that we may be able to accelerate any natural tendency in the decline in the leprosy rate.

Let us now consider tuberculosis, its incidence and severity. Here the evidence appears to point in a very different direction. Available statistics again are very poor but the indications are that tuberculosis is increasing, and increasing markedly in some parts of the country. Evidence regarding the severity of the disease in Indians as compared to other races is very limited. I can quote a very limited personal experience of tuberculosis in up-country people and in them I was astonished at the rapidity with which the disease developed and often proved fatal. Other doctors who have seen tuberculosis in India and in Europe have had similar experiences.

Benjamin (1938) has recently published an analysis of 2,158 cases in Indians. His conclusions are that tuberculosis in Indians 'is of a very serious type; it is acute, rapidly developing with little tendency to show a natural resistance and healing'. 'There is a severe exudative reaction, followed by a rapid breaking down of tuberculous tissue, with resulting cavity formation'. It should be stated that Frimodt-Möller reports that the results of treatment of tuberculosis in Indians are quite comparable to the results obtained in Europe.

This evidence, inadequate though it is, suggests that in India the tuberculosis epidemic may be on the upward grade. If so, it means that India is facing a very serious situation and that preventive work is a vital necessity.

I will now discuss briefly some of the factors which have apparently influenced the spread of leprosy in the past and which may now and in the future influence the spread of tuberculosis in India. One important factor which has undoubtedly influenced the spread of leprosy in various parts of the world has been the development of communications, with migration of large bodies of people from one area to the other or from one country to the other, the development of commerce and industry and the recruitment of labourers from distant areas. In this way leprosy has spread from one country to another and from one part to another of the same country. In modern times industrialization has had its influence and there are indications that this factor is operating in spreading leprosy in certain parts of India. People are migrating from areas where there is little or no leprosy, to industrial areas where they mingle with other people from areas where there is much leprosy. They get infected, develop leprosy and then return to their villages and introduce the disease there. Even when an industrial population is permanently resident in an area, the concentration of people and bad housing may help the disease to spread, but when the industrial population is largely migratory, as in India, the danger is much magnified.

These things apparently influence the spread of leprosy and I think that undoubtedly they are influencing the spread of tuberculosis also.

It may, however, be argued that in western Europe the period of intense industrialization was accompanied by a decline in tuberculosis. I would point out, however, that there are three great differences between India and Europe in this respect. Firstly, industry developed in Europe when tuberculosis was apparently already at its peak and possibly on the decline. This is not so in India. Secondly, the industrial population in Europe is not largely migratory as in India. Thirdly, in Europe the development of industry was accompanied by the development of reasonably effective public health work and social hygiene in its wide sense, and also by a rise in the standard of education. I fear that we cannot yet say the same of India. Therefore, I think that the influence of industrialization on the spread of tuberculosis may be much more marked in India than it has been in Europe.

I have mentioned above some points about leprosy and tuberculosis in communities and large groups of people. Now I will briefly discuss the question of leprosy and tuberculosis in

small groups of people, in other words in families.

A study of leprosy in families shows that of young children living in contact with open infectious cases, a high proportion, sometimes between 50 and 80 per cent or more, sooner or later develop signs of the disease, and the disease tends to be severe; whereas of adults living under similar conditions, only about 5 per cent develop the disease, and the disease is often in a mild form. These findings indicate that children are more susceptible to leprosy than adults and that most serious infections are acquired early in life. Even when the disease appears relatively late in life it is often the result of an infection acquired early in life, an infection which has long lain latent.

I believe that similar studies of tuberculosis in families have given somewhat similar results. It is found, for example, that if a mother is an open case of tuberculosis, she may infect child after child and the children often get severe tuberculosis, but the husband who is living with such a wife usually does not get the disease at all, or else gets it in a relatively mild form. These facts show the relatively high degree of immunity in adults. I have been interested to read that an increasing number of workers on tuberculosis is tending to regard adult tuberculosis as often being the late result of an infection acquired in childhood, although some workers think differently. At any rate it is clear that adults get the disease much less readily than children, although the difference may not be so marked in India as it is in Europe.

The relative immunity of adults to tuberculosis is usually attributed to repeated subliminal infections early in life. It is however very difficult to explain the relative immunity of adults to leprosy on this basis. It appears to be a common natural development with age.

These facts about leprosy and tuberculosis are being more widely recognized, and this is reflected in a strengthening of the emphasis laid on the prevention of infection of children.

In leprosy you may have your diagnostic and treatment centres, your leper colonies and so on, but unless provision is made for the isolation of open cases of leprosy from children, such measures are not likely to have much influence on the spread of the disease in the community, and may even do more harm than good. For example, a leper colony with married quarters and with no proper arrangements for separation of children, as is sometimes found, may become a breeding place for lepers.

I think that the same thing may be true of tuberculosis. You may develop your tuberculosis clinics, your sanatoria, and your educational and propaganda work in the homes of the patients and in the population at large, but as long as the prevention of infection of children and young people is not attempted it will be difficult to accomplish much in the control of tuberculosis in the community. I have seen an Indian mother with chronic tuberculosis, an open case, infect 5 children one after another. This is a very difficult matter to deal with properly. The average Indian house is too small and the average family too big and too poor to allow of reasonable arrangements to protect children and young people from infection by an open case in the house.

It seems to me that one great hope for the future lies in the direction of the development of some method of immunizing young people exposed to infection. There is of course the B.C.G. vaccine. Opinions regarding its efficacy are very divided. The development of work on the subject has been greatly handicapped by the terrible tragedy in Germany a few years ago when virulent cultures were accidently substituted for B.C.G. vaccines, with a high mortality in the vaccinated children. At any rate it is now clear that the vaccine does no harm, and numerous competent workers are convinced of its value. A summary of the findings made in studies of the use of B.C.G. vaccine by Nègre and Goyal (1938) have recently been published. It is worthy of note that some large industrial concerns in Europe who recruit Indians for training in Europe and for future work in India, insist on such workers being vaccinated with B.C.G. before going to Europe. If further work demonstrates clearly that B.C.G. vaccine or some modification of it does produce immunity, then I think that there should be a vast field for the application of this measure in India for the immunization of contacts and of workers in industry. I only wish that in leprosy there was any prospect or hope, however remote, of the development of some similar method of immunizing to leprosy those who are exposed to infection.

I have given you some of my ideas about these two diseases, the 'twin diseases' as they have been called, leprosy and tuberculosis. My knowledge of tuberculosis is very limited and some of my ideas about this disease may be wrong. I do hope, however, that I have been able to do one thing, namely, to show how tuberculosis and leprosy are linked together and to the whole realm of medicine and public health. What does this mean in practice? It means several things. It means, firstly, that those of us who are specialists in one subject should keep in touch with other subjects, particularly allied subjects, and as far as possible with the whole realm of public health work. It means secondly that anti-tuberculosis and anti-leprosy work and other similar activities should not develop entirely independently of public health activities in general. They may be started by special organizations but they should keep in touch with general public health work and in course of time they may be incorporated in them. Thirdly, it means that the public health system should ultimately include anti-leprosy and anti-tuberculosis work as an integral part of itself. There

is far too often a tendency on the part of medical and public health authorities to regard these two diseases, and particularly leprosy, as something apart from their general sphere. I hope that as time goes on this tendency will get less, that medical schools and colleges would really give good instruction in these subjects, that practising doctors in whatever sphere they happen to work, will have the necessary knowledge, and will regard the diagnosis and management of cases of these diseases as an essential part of their work, with of course advice from experts when necessary. I hope also that public health workers will regard anti-leprosy and anti-tuberculosis work in the same way. Real progress towards the solution of both these problems, leprosy and tuberculosis, can only be made when every doctor has become alive to their importance, and has the necessary knowledge to play his part, a most important part, in the work.

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Medical News

TUBERCULOSIS NEWS FOR THE MONTH OF MAY 1939

On account of the amalgamation of the King George Thanksgiving Fund into the Tuberculosis Association of India the Hassan Masud Suhrawardy Memorial Challenge Shield Competition which since 1932 was organized by the Fund will in future be held under the auspices of the Tuberculosis Association of India.

The competition will be open to any corporation, municipal council, municipal committee or district organization in British India or an Indian State doing anti-tuberculosis work, or any other organization, association or committee, not being larger than a district organization, which is doing anti-tuberculosis work in any of the said territories, which the central committee of the Association may from time to time admit to be eligible.

His Excellency the Governor of the N.-W. F. P. laid the foundation of a tuberculosis sanatorium at Daddar on the 11th May. The sanatorium provides for 70 beds and includes 10 beds for females. It is understood the institution is being modelled on the lines of the Union Mission Tuberculosis Sanatorium at Madanapalle.

The Bombay Tuberculosis Association has been formed with H. E. the Governor as Patron and the Hon. the Prime Minister as President of the Association.

Bombay collected Rs. 7,01,548 for the King-Emperor's Anti-Tuberculosis Fund while Ahmedabad organized a separate appeal for anti-tuberculosis work and collected Rs. 1,82,000.

In allocating funds for future work, the Association has decided to invest Rs. 2,00,000 as permanent endowment for the Provincial Association. Rs. 2,00,000 have been invested in the name of the Bel-air Sanatorium, Panchagani, and the interest thereon will be paid to this institution so long as it continues as a charitable institution and admits free patients. The Bombay Municipality on agreeing to enlarge the accommodation in the Turner Sanatorium from 40 to 80 beds, has been given Rs. 60,000 to meet one-half the cost on capital expenditure. Rs. 60,000 have been earmarked for building and equipment of clinics near the teaching hospital and the municipality and the governments have been approached on the subject. Rs. 98,190 have been allocated for clinics in the districts.

The Italian Fascist National Federation against tuberculosis has placed at the disposal of the International Union against tuberculosis six scholarships at the 'Carlo Forlanini' Institute in Rome. The Tuberculosis Association of India, who is a member of the International Union, has been invited to recommend names of young physicians already familiar with tuberculosis problems.

These scholarships, of a value of 2,000 lira respectively, *plus* board and lodging will be tenable for one academic year (from 15th November to 15th July) including the usual holiday periods.

The kind of work undertaken at the institute will be subject to an agreement between the director of the institute and the candidate.

The Executive Committee of the International Union against Tuberculosis on which India is represented through the Tuberculosis Association of India places at the disposal of the Governments and Associations belonging to the Union a biennial prize of a value of 2,500 French francs, in memory of the late Professor Leon Bernard, who was the Founder and, for fourteen years, the Secretary-General of the Union.

This prize will be awarded for the second time in the year 1940 to the author of an original essay on 'Conjugal Tuberculosis', in French or in English.

The essays must be typewritten or printed and must not exceed 10,000 words. They must be forwarded to the Secretary, The Tuberculosis Association of India, 20, Talkatora Road, New Delhi, not later than the 1st March, 1940. Essays approved by the Association will later be forwarded to the Executive Committee of the Union.

Should the Executive Committee decide that no essay submitted is of sufficient merit, the prize will not be awarded in 1940, but will be offered again in the following year.

ASSAM MEDICAL COUNCIL

An Ordinary General Meeting of the Assam Medical Council was held on Monday, 8th May, 1939, in the Office of the Inspector-General of Civil Hospitals, Assam, Shillong.

On taking the Chair, the President addressed the Council as follows:—

'Gentlemen, I welcome you once more to the deliberations of the Assam Medical Council, and I take this opportunity of making a few general observations on the agenda which is before you.

During the past year, since the last session of the Council, one important matter has remained over for consideration and another has presented itself for the first time. The former refers to the question of "covering", regarding which you will remember that the council came to no final decision at its last session. Since then I have examined the matter, to the best of my ability, from every point of view, and I shall ask you to-day to come to a final decision on the matter. As the question is an important one, it will perhaps be desirable to place before you what appeared to me to be the underlying facts and arguments which, in due course, I shall ask you to consider.

The most important point at issue is evidently the implication of the word "covering", and in interpreting this word it is, I think, essential to consider the different conditions which prevail in the United Kingdom, where the use of the word originated, and in Assam, where the use of the word has been copied, as in most of the