LEPROSY - A REVIEW, AND A PLAN OF CAMPAIGN FOR INDIA.

a Thesis

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by

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PREFACE.

"And it came to pass, when he was in a certain city, behold a man full of leprosy, who, seeing Jesus, fell on his face, and besought him, saying, Lord, if thou wilt, thou canst make me clean. And he put forth his hand, and touched him saying, I will, be thou clean. And immediately the leprosy departed from him."


And again -

"And behold, there came a leper and worshipped him, saying, Lord if thou wilt thou canst make me clean. And Jesus put forth his hand and touched him saying, I will, be thou clean. And immediately his leprosy was cleansed."

Matthew viii.2.3.

A few months ago, at an informal friendly meeting of medical colleagues in India, one of the company told, how a recently qualified doctor had examined twenty recruits amongst whom were two cases of leprosy.

These, the newcomer had diagnosed as "Ringworm"!
My mind leapt back across the years to the year 1924 when I arrived in the Malay States. Well do I remember my first case in Kuala Lumpur -- a young Chinese woman afflicted with nodular leprosy, which I had diagnosed as Syphilis, simply because the Wassermann reaction was positive.

My ideas concerning leprosy at that time were still coloured by my reading in lay and biblical writings, but two facts had been vividly impressed on my mind -- the need for urgency, and the desire to be of some practical use. "behold a man full of leprosy who, seeing Jesus, fell on his face and besought him".

That poor woman proved the incentive which was to widen my scanty knowledge of this "dread scourge" which lays its loathsome fingers so ruthlessly on the bodies of so many men and women of divers races, making them outcasts from their fellow men.

Long has been the road, weary the pilgrimage and great the sacrifices of the missionaries and the physicians who have been pioneers in this field. By trial and error the question of segregation has evolved in its true significance. Bacteriological and pathological knowledge of the disease have advanced. Modern teaching and propaganda are beginning to break/
break down the false assumption that leprosy per se, is incurable.

A great advance was made by Rogers in 1916 when he discovered the efficacy of the soluble sodium salts of the fatty acids of the Chaulmoogra and other oils, when these were injected intravenously and intramuscularly.

It is my humble desire to contribute even a little to the pool of knowledge which owes so much to those great men who have made leprosy a life study.

For 17 years in Malaya I have constantly had lepers under my care – Malays, Javanese, Boyanese, Siamese, Chinese and Indians. All my treasured case notes, photographs and X-Ray films, collected during this period, were unavoidably left to the tender mercies of the Japanese, when I escaped from the fallen city of Singapore. Consequently, I must crave indulgence for the omission of illustrative details, and must draw upon the deep wells of poignant memory!

In a small boat one escaped with one's life – that is all, and deemed oneself fortunate!

Three years as Administrative Medical Officer of 25 Indian States, have given me every opportunity to visit and inspect leper settlements and clinics in my area. It has been my privilege to discuss methods/
methods of treatment and control with the officers in charge.

A plan has gradually evolved from my present administrative work, and from practical experience gained in the Malay States, at first by many disappointments, but later and with fuller knowledge by many satisfying and encouraging results.

In this thesis, which is entirely my own work, I offer a review of leprosy and of my own practical experience of it. I also submit a plan of campaign as a helpful contribution to the control of the disease in India.

My warmest thanks are due to the very happy spirit of co-operation and support shown me by one and all of my colleagues in the States of Rajputana.
Leprosy (Lepra Arabum, Elephantiasis Graecorum) is a disease of low infectivity frequently referred to in writings from the most ancient times to the present day. The following outline of its history, although of doubtful accuracy, is an approximation of what is known.

Its origin seems to have been in Africa, for we first hear of its spread from thence to India and Egypt in pre-historic times.

In the Eber's papyrus circa 1500 B.C. descriptions of skin swellings correspond very closely to those of leprosy.

The Vedas record a very early reference to it about 1440 B.C.

In the Egyptian records circa 1350 B.C., in the reign of Ramesis II, there are indications of undoubted similar lesions in negro slaves brought from the Sudan. Throughout history, invasions and slave trade have proved important factors in the spread of leprosy from one country to another. Thus, from very early times, this disease was very common in Africa, India and Egypt, in which areas it is still common to the present day.

From Egypt, leprosy spread round the Eastern Mediterranean/
Mediterranean. The Jewish writings of the Bible contain many references to the infection.

"And many lepers were in Israel in the time of Elisha the prophet; and none of them was cleansed, saving Naaman the Syrian". Luke iv. 27.

It is possible that in the Hippocratic era no true leprosy existed in Greece, for Hippocrates did not describe it. But Aristotle, circa 345 B.C., made references to the disease in Asia Minor.

The conquests of Darius and Xerxes in 480 B.C. led to migrations of peoples and spread of leprosy.

Leprosy was unrecorded in Roman writings until it occurred in soldiers returning from the East. By the Romans it was introduced into other parts of Europe. Galen wrote of it in A.D. 180, and Virchow reported that by A.D. 600 there were hundreds of leper houses in Germany and Italy. The Romans also infected Spain about the fifth century.

From Spain it spread to France, and was probably introduced into England by the Romans. The Crusaders probably also aided its spread.

Leprosy reached its height in Western Europe about A.D. 1200, when some two thousand leper houses existed in France alone. It then declined in Europe, and by the seventeenth century had died out more or less, except in a few persistent foci in the Mediterranean area and in Northern Europe.

No satisfactory explanation has ever been given/
given for the dying out of leprosy in Western Europe, but, most probably, the isolation carried out in leper houses was in part largely responsible. Leprosy was introduced into America by invaders from Europe, and from Africa by imported slaves. Even within the last half century it has been introduced into uninfected countries, chiefly isolated islands;—for example, the South Sea Islands by Chinese settlers.

On the whole, the history of Leprosy is a history of the persistent endemicity of the disease in some parts of the world, for example, India and Africa, for thousands of years. Later, it has been introduced into other parts of the world where it may be seen in the form of long period epidemics.
DISTRIBUTION.

1. Present distribution of leprosy in the world.

Leprosy is now regarded as a tropical disease, and is found chiefly in tropical countries, although still existing in some countries in high latitudes. The present main foci are Africa, India, Burma, South China, Malaya, Philippine Islands, Hawaiian Islands and South America, although it still lingers in Iceland and Norway. Climate is not directly a determining factor in its distribution. It has been said very aptly that "leprosy may be determined by climate only so far as civilization is determined by climate."

2. Distribution of leprosy in India.

The highest incidence is along the East Coast from north to south, the most heavily infected areas being Western Bengal, South Bihar, Orissa and Madras. The Himalayan areas and Central India show a moderate incidence, while North and North-West India are relatively free from the disease. Among some aboriginal tribes it has been known for long, and is limited by tribal laws and customs; while among other tribes it was unknown until recently, when it was introduced by those who had left their villages to labour in cities and industrial areas, and, on returning home, infected their fellow tribesmen/
tribesmen. With the building of roads and railways, the industrialisation of the country, the gradual breaking down of the caste system, and therefore the freer mixing and greater movement of the population, areas which were formerly uninvolved have become infected.

The census of 1931 gives the total number of lepers in India as 147,911, or 42 per 100,000 of the population; but recent surveys carried out throughout India have shown that these figures are quite inaccurate, and even the census authorities admit, that the figures were arrived at on the reports of unqualified persons. The recent surveys undertaken by qualified observers report that there are at least one million persons suffering from leprosy in India. It is impossible to state accurately the number of sufferers from this disease in the whole world, but various authorities believe it to be in the neighbourhood of two to four million persons, and it is possible that this number is underestimated.

The 1931 census figures show, that the number of cases of leprosy in India increases rapidly from 0-1 year of age, the peak being reached between the ages of 35-40 years. After the age of 55 years the number of cases diminishes rapidly, and at the age of 70 years very few cases are seen.

To illustrate this I append a curtailed census table/
It has been found that the incidence of leprosy is higher in males than in females. A difference appears even in childhood, a difference which becomes more marked in adult life. The disease in females tends to take a milder form. The cause of these differences may be partly due to the fact that women in India are more or less secluded, and therefore less liable to infection. Lowe (2) considers it to be to a certain extent physiological.
ETIOLOGY.

There are three main theories which have held sway in the minds of men as to the origin and transmission of leprosy.

These are:

1. That the disease is an hereditary one.
2. That it is dietary in origin.
3. That infection mainly by prolonged and close contact is the outstanding cause.

As to the First theory:— In the years prior to 1871, when Hansen discovered the Lepra Bacillus (3), it was universally believed that leprosy was an hereditary disease.

The two famous Norwegian physicians Drs Danielssen and Boeck in their book published in 1848 (4) promulgated this theory. Danielssen inoculated himself and nine others with material from the lesions of nodular leprosy, but failed to produce the disease. He demonstrated to his own satisfaction, that leprosy was not an infective disease, and concluded that it was due to heredity.

This conclusion was also reached by the Commission sent to India from The Royal College of Physicians, London, in 1862 in order to investigate and report on Leprosy.

I found in Malaya the belief firmly ingrained and/
and established that the cause of leprosy was an hereditary one, - so much so, that no sooner had I informed the patient of my diagnosis, that I could be sure that he would quickly answer "Mana boleh, Tuan!" "(Impossible, Sir!)", adding, that not one of his family had ever had the disease. I have known patients become quite indignant at the presumed suggestion of such a possibility. I observed this particularly with patients who had no relations suffering from leprosy.

The belief in heredity, as the main cause of leprosy, I certainly found widespread, and firmly fixed in the minds of all races in the Malay States.

The Second theory was advanced by Sir Jonathan Hutchinson in 1861. He contended that leprosy was primarily due to the consumption of badly preserved and decomposed fish. He named the disease "Fish-eater's gout", and stated, that even years later the disease might develop.

No evidence has been so far forthcoming which points to the alimentary canal as a portal of entry for the lepra bacillus.

I am of the opinion that diet plays an important part as a predisposing factor. Not only does gross ignorance regarding the essentials and component parts of a balanced diet prevail among the poorer members/
members of the human race, but the driving necessities of hunger and want often force them to eat such articles as badly preserved and decomposed fish which are unfit for human consumption. I maintain that resistance is so lowered, the outbreak of skin diseases and open sores so prevalent, that these peoples present a soil ready for contagious infection.

Added to this, the insanitary conditions, the overcrowding, the unsatisfactory housing, the contamination of food and open sores by flies and insects, - are all factors which tend to lower still further the patient's defensive forces.

The Third theory, - that infection by contagion is the outstanding cause of leprosy, is now universally held by medical authorities to-day.

Sir Leonard Rogers has reported two cases of doctors who, when operating on patients suffering from leprosy, had accidentally cut their own fingers. Soon after, both doctors developed leprosy, the sores appearing first on the fingers which had been cut.

The discovery of the Lepra Bacillus in practically all active lesions of the disease was almost conclusive evidence of its infective nature; although up to date Koch's postulates have not yet been fulfilled. -
1. No evidence is forthcoming of the transmission of the disease experimentally to any animal.

2. I can find no reference in the literature that the successful culture of the lepra bacillus has been fully established. According to Rogers and Muir (5) fleas, bugs and lice, which had been fed on leprous patients, are found to harbour the bacilli; but there is no proof so far that they in turn inoculate leprosy into human beings. It is possible that the scratchings by the patient cause abrasions through which the lepra bacilli enter.

That it is a contagious infection is now accepted by informed medical opinion; but the portals of entry are not yet clear. Suffice it to say, that I have had many patients who undoubtedly acquired the disease by wearing the clothes of, or by sleeping over long periods in the beds of relations who were suffering from leprosy. Each of these patients had one or more abrasions of the skin.

Many a time have I seen leprous patches at the angles of the nose in worm-infested children. It is highly probable that picking the nose had caused skin abrasions through which bacilli could enter.

Children are very susceptible to the disease. It has been said that cases of leprosy are rare under five years of age, but Cochrane(6) states that most serious infections are contracted in childhood.
A study of leprosy in families has demonstrated the following three important facts which have been noted and reported by various Commissions to the East in recent times:—

1. A child born of an infective parent and removed immediately after birth, never develops leprosy. The disease is therefore not hereditary.

2. Children so born, but not separated from the infected parent, develop leprosy in a high proportion of cases. Some Authorities say that 50% is a fairly correct estimate.

3. An adult similarly exposed to infection, develops the disease comparatively rarely. It is the long continued close contact which is the most dangerous cause of infection; — the more direct and prolonged the contact the more likely is the disease to be acquired.

Sexual promiscuity has been an important factor in spreading the disease. It is well known that this occurs amongst certain semi-aboriginal tribes amongst whom leprosy is rife.

The fact, that at the present time leprosy is considered to be a tropical disease, whereas it was once common in Europe and even as far north as Iceland and Norway, proves that its disappearance from these parts must be largely due to the better sanitary conditions and stricter segregation adopted by/
by these countries; whereas, the humid atmosphere of the tropics encourages its continuation and spread. In India, the greatest incidence is along the East coast; in North-east India the incidence is low, and the proportion of infectious cases also is low – the reason being, that in the East coast areas humidity is very much higher and the standard of living is much poorer.

In Rajputana, humidity is low, and the people are wheat-eaters and have a far higher standard of living.

To summarise:—

1. Leprosy is an infectious disease.
   a. The younger the patient, the more susceptible he is.
   b. The more direct and prolonged the contact the more likely is the disease to be acquired.
   c. The following factors which diminish the defensive forces of the patient are predisposing causes:—
      (1) Insufficient and unsuitable food.
      (2) Overcrowding and insanitary surroundings.
      (3) The presence of other diseases such as, — Deficiency Diseases
          Syphilis
          Malaria
          Dysentery
          Ankylostomiasis etc.
   d. A high humidity.
BACTERIOLOGY.

The Bacillus of leprosy was discovered by a Norwegian, Dr G.A. Hansen in 1871 when engaged in research work among the lepers of Norway. It belongs to the Mycobacteria which occupy a position midway between bacilli and fungi.

They are differentiated from tubercle bacilli thus:

**Lepra Bacillus.**

1. Thin rods 6-8 μ in length.
2. Tapered at one or both extremities.
3. Less curved than tubercle bacilli.
4. Arranged in large clumps of small bundles of parallel bacilli.
5. When stained may break up like cocci.
6. Basic stains taken up readily. Acid-fast, but some strains are easily decolourised.
7. Has never been cultivated with certainty.
8. Causes no progressive disease when injected into guinea pigs.

**Tubercle Bacillus.**

1. 5-4 μ in length.
2. May be thickened at both ends.
3. More curved and slender.
4. Occurs singly or in groups of three, four or five.
5. Beading may occur.
6. Not so readily, on account of fatty capsule.
7. Cultivated readily.
8. Causes progressive disease.
PATHOLOGY.

Muir (7) states - "To understand the nature of Leprosy it is necessary to keep in mind its low toxicity and its extreme chronicity, in consequence of these the onset in the average case tends to be insidious, and often the disease becomes generalised without producing signs or symptoms sufficient to attract the patient's attention".

All organs of the body may be invaded but it manifests clinically in lesions of:—

- skin.
- mucosae.
- peripheral nerves.

The Lesion.

According to Black (8), the lesion contains the so-called "foam cells" or lepra cells. These are large monocytes which have taken up the acid-fast bacilli.

Taylor (9) states that a thinning out of the epidermis occurs owing to accumulation of these cells and other mononuclear elements. The papillae are obliterated, and the resulting appearance is of a tumour covered by thin smooth glossy skin.

There are two distinct types of leprosy:

1. The neural, or high resistant type.
2. The lepromatous, nodular, cutaneous or low resistant type.
1. The neural type verges towards that of tuberculosis, hence the name 'tuberculoid'.
According to McCarthy (10), this type presents a histopathological appearance of numerous sharply limited nodules of varying sizes, made up of lymphocytes and plasma cells, surrounding a central zone of epithelioid cells and Langhan's giant cells.

Muir (11) states, that the presence of Langhan's giant cells is a sign of strong local reaction to the lepra bacilli.

When necrosis occurs it causes nerve abscesses and ulcerations of the skin. It is the terminal nerves which are first affected, and later the nerve trunks:

There is no involvement of the skin as a whole, nor of mucous membranes, nor of the internal organs.

2. The Lepromatous type of leprosy.

In this type the reaction of the tissues is poor, and the number of lepra bacilli very great, - so much so that the protoplasam of the characteristic cell - the monocyte - shows fatty change, and vacuoles appear, crowded with lepra bacilli.

There are:-

No Langhan's giant cells.

Few epithelioid cells.

The absence of these two shows the poor nature of the defensive mechanism of the body, and there occurs/
occurs diffuse infiltration of –

Skin, causing corrugation from thickening.

Nerves.

Mucous membranes.

Lymphatic glands.

Bone marrow.

Internal organs - liver, spleen, lungs.

Eyes.

Prendergast (12), refers to the eyes as being especially affected in lepromatous leprosy.

I would emphasise the seriousness of lepromatous leprosy.

In this form of the disease the majority of infectious cases occur, and also, owing to the diffuse nature of the granulomatous tissue, many cases go unrecognised until the disease is well advanced, since clinical signs are not visible.

Sometimes leprosy begins as the neural type, and later changes to the lepromatous type. Such cases are called 'mixed leprosy'.
The pathological processes of the two main forms of the disease can be expressed as follows.

If Patient's resistance is high,
  \[\text{Tissue response to Leprae Bacilli is great.}\]
  \[\text{Multiplication of Bacilli is limited, resulting in Granulation tissue of a 'tuberculoid' type.}\]
  \[\text{Localised to certain parts of skin and PERIPHERAL NERVES.}\]
  \[\text{Clinical signs obvious.}\]
  \[\text{Neural leprosy}\]

If Patient's resistance is low,
  \[\text{Tissue response to Leprae Bacilli is slight.}\]
  \[\text{Leprae Bacilli multiply rapidly, producing Very diffuse granulation tissue.}\]
  \[\text{Widespread CUTANEOUS or nodular lesions.}\]
  \[\text{Neural signs milder.}\]
  \[\text{Clinical signs often slight or absent.}\]
  \[\text{Cutaneous or lepromatous leprosy.}\]
Incubation Period.

In the majority of cases of leprosy, the first recognised lesion is often the result of a generalised infection. The outward signs of early lesions may not be visible for a long time, especially in the dark skinned races. Added to this, the low toxicity of the lepra bacillus may cause the infection to remain latent, or unrecognised, by both patient and doctor for a considerable time. The incubation period is consequently of very variable duration, and is much longer than that of any other infection.

McCoy (13) states, "There are apparently well substantiated cases in which twenty-five years or more had apparently intervened, but usually the incubation period will be from 5-10 years".

Rogers and Muir (14), two highly skilled observers, conclude that the incubation period is from two to four years. They cite a case however, reported by Radcliffe-Crocker, of a girl developing leprosy seven years after returning to England from Ceylon. They state that - "slightly discoloured anaesthetic patches may be overlooked for years, and later/
later, exacerbations of the disease may be mistaken for its first appearance".

From the above it will be seen how difficult it is in any case of leprosy,

(a) to find the definite length of the incubation period.

(b) to recognise the starting point of the disease.

Leprosy manifests itself in two main types (see Page 22),

1. The Neural form - relatively mild, and found in the majority of cases.

2. The Lepromatous type - of serious import, and in which the greater number of infectious cases occurs.

In some cases the disease appears first in the neural form, and later may develop into the lepromatous type. It is necessary to state however, that there may be great variations in the merging of the two types. These cases are named "Mixed Leprosy."

Only a certain proportion of cases of leprosy can be regarded as infectious. Surveys in the villages of Eastern Bengal made in 1935 (15) showed, that on an average, one out of five cases were highly infectious.

More recent reports after a more accurate survey was/
was taken of the same district show that only one out of ten cases is highly infectious.

I found in Rajputana during the year 1943, after careful scrutiny of all the reports which came to my office from the 25 States in that Province - that only one out of twenty-two cases could be classed as highly infectious.

This significant difference I attribute to the greater resistance of the Rajputs, whose standard of living is higher and who are mainly wheat eaters. On the whole, they are much more free from associated diseases such as dysentery and ankylostomiasis - diseases which are more common in the hot humid climate of Bengal than in that of Rajputana. Certain it is that famines have played havoc from time immemorial amongst the inhabitants of Bengal.

No matter which type of disease the patient is suffering from I have found, on careful inquiry, that there is in most cases a history of prodromal symptoms. These consist of periodic attacks of malaise, headache and fever, lasting for a few days and recurring at intervals for many months. The patient hardly ever thinks of consulting a doctor for these, and, even if he did so, it is more than likely that the most experienced doctor would not always realise that he was dealing with another victim of leprosy.

I./
I. Neural Leprosy - Clinical appearances.

The two lesions of this type are the "macular" and the "anaesthetic".

It must be made clear that the term "macule" as used in leprosy must be differentiated from that used by dermatologists. The latter define the macule as "a spot which is not raised above the skin; it may be vascular or pigmentary. The term is usually applied to small lesions up to the size of a pea, a larger lesion being called a "plaque" or "a tache". Large sheets of redness are generally called an "erythema". (16).

So that there may be no confusion, it is necessary to point out that the leprosy worker uses the term 'macule' to describe any round, oval or irregular patch in the skin in which there may be a considerable amount of elevation, which may vary in size from one quarter inch to one foot or more in diameter, and in which one or more of the following changes may be found:–

1. LOSS OR PARTIAL LOSS IN CUTANEOUS SENSIBILITY.
2. Cutaneous nerves supplying the area may be thickened.
3. Loss, or partial loss of pigment.
4. Thickening and elevation chiefly round margin.
5. Erythema and sometimes ulceration in the centre.
6./
6. The skin appears dry, thin and glossy.
7. Owing to impairment of sweat glands, there is scaliness and falling out of hair.

Any of the above changes may be of any degree from slight to advanced.

The diminution in Cutaneous Sensibility varies.

In macules of the face it is, in the majority of cases, slight. It is more marked on the trunk, and most marked on the limbs.

Earliest sensations lost are those of Heat and Pain; later, sensation of Touch is affected.

These changes are usually accompanied by other subjective sensory changes - the patient may complain of chilliness and malaise,

mental depression,
formication,
hyperaesthesia,
pain and tingling on percussion.

The term 'tuberculoid' is often applied to the macules which show marked thickening and erythema because of the histopathological changes found in these lesions.
As the disease advances, and the peripheral nerve trunks are involved by spread from the cutaneous nerves, secondary signs and symptoms now appear in the area supplied by the affected nerve. These are:

1. Wasting and paralysis of muscles with consequent deformity.
2. Impairment of cutaneous sensibility, starting peripherally and extending up the limb.
3. Impairment of sweating, with dryness and scaliness of the skin of the affected area.
4. Trophic ulcers, and absorption of the bones of the hands and feet. Secondary infection of trophic ulcers is common.

The nerves most commonly involved are:

(a) Ulnar Nerve.— Usually affected just above elbow joint. Anaesthesia of fifth and ulnar half of fourth finger. Ulnar side of hand and forearm, later, paralysis of small muscles of hand. Development of typical 'claw-hand'.

(b)
A case of the neural or high resistant type of leprosy, with 7th nerve paralysis, and ectropion of the lower eyelids.
(b) Peroneal Nerve — Commonly affected in region of head of fibula.  
Anaesthesia of dorsum of foot, and outer half of front of leg.  
Toes flexed, drop foot, commonly inverted.  
Steppage gait.

(c) Post. Tibial Nerve. Anaesthesia of sole of foot, hyperkeratosis, trophic ulcers.

(d) Fifth and Seventh cranial nerves. Paralysis of facial and orbital muscles.  
Anaesthesia of cornea. Octropion, conjunctivitis, and corneal ulcer frequent, due to irritation of eyes from foreign bodies.

Bone lesions.

Sometimes bone absorption takes place in the hands and feet, but not where contractures occur. According to Cooney and Crosby (17) these bone lesions originate in degenerating changes in the tracts of Gall and Burdach, and are not pathognomonic of neural leprosy, as similar lesions occur in Tabes Dorsalis etc.
2. Lepromatous leprosy.

Here the poor victim is overwhelmed by the invasion of a vast army of Leprae Bacilli. His defensive forces are powerless; there is little or no tissue response. The leprae bacilli are found everywhere — so rapidly do they multiply and spread in the body. Prodromal symptoms are always marked. They include:

- Fever, rigors and sweating.
- Diarrhoea, alternating with constipation.
- Progressive weakness, epistaxis.
- The blood may show leucocytosis.
- Blood sedimentation rate always raised.

The lesions become diffuse and widespread, affecting any or every organ of the body.

Clinically, the chief lesions are found in the skin and mucous membranes.

1. Skin lesions.

The skin of the whole body may be affected. At first, many foci of infection in the superficial layers may appear and coalesce before the patient is aware that he is suffering from leprosy.

Certain areas of the skin are affected more than others — face, ears, backs of elbows and knees, and the buttocks.

As the disease progresses and penetrates more deeply,
deeply, the skin becomes stretched, shiny, and smooth to touch. On the face, where uneven stretching occurs, corrugations appear, which, combined with the enlarged nose, pendulous cheeks and thickened lobes of the ears, give the leonine appearance or "visage leonine" of the French writers. The hair of the face and of the eyebrows often falls out.

Some areas of skin show pigmentary changes, and have an indefinite margin. The surface is smoother than that of the macules of neural leprosy. They show no sensory changes, nor thickening of the nerves supplying the area. Leprae bacilli are easily found.

Later, nodules appear in the skin which may be as small as a pea or as large as a walnut. These nodules may break down and form ulcers.

The mucous membranes of the nose, pharynx and larynx are often affected. Hypertrophy, nodulation and ulceration may cause hoarseness and dyspnoea. The ulcers formed from the necrosed nodules tend to spread both superficially and deeply, eating away all adjacent tissues; or, they may eventually heal, leaving dreadful deformities.

Hypertrophy of the mammary glands may be striking, and is thought by some observers to be due to failure of internal secretion from the testes which are often involved.

Leprous/
Leprous infiltration of the eyes may cause iridocyclitis and blindness.

The internal organs such as the liver and spleen, often show leprous lesions on post-mortem examination, but clinical symptoms are usually absent.

Absorption of bone may take place, fingers and toes may ulcerate and drop off. According to Paget and Mayoral, (18), bone lesions in nodular leprosy are due to the direct action of the Mycobacterium leprae which cause endarteritis, and later necrosis of bone.

A case of "Lepromatous", "Nodular", or low resistant type of leprosy.
A typical case of Gynecomastia, associated with atrophy of testes.
A case of subsided leprous infiltration shewing atrophy and wrinkling of the skin. Bandage on left arm covers an abscess of the left ulnar nerve close to the elbow joint.
**DIFFERENTIAL DIAGNOSIS.**

The following conditions must be carefully considered before forming a diagnosis.

**Group 1.**

One must differentiate from diseases which produce lesions in the skin resembling leprosy.

(a) **Yaws**

Treponemata found in serum from early lesions.

Absence of changes in sensation.

" " thickened nerves.

" " *Leprae* Bacilli.

Ready response to arsenic and bismuth.

(b) **Dermal Leishmaniasis**

History of Kala-Azar.

Leishman Donavan bodies may be found.

Macules small, with little tendency to spread.

Macules on face concentrated round mouth and nose.

(c) **Syphilis**

Absence of sensory changes.

Ready response to treatment.

Both diseases frequently present together.

A positive serological test does not exclude leprosy.

(d)/
(d) Tinae Versicolar  
Presence of fungus. Itching may be marked. No raised margin of macules. Absence of sensory changes.

(e) Psoriasis  
Lesions scaly from the start. Surface under removed scales studded with numerous small red points. Little pigmentation. Distribution – mainly extensor surfaces. No sensory changes nor thickened nerves. Depilation and anidrosis absent.

(f) Lichen Planus  
Some typical papules almost always seen. Lesions have characteristic lilac or purple colour. Flexor aspects of limbs most involved. Itching generally intense.

(g) Seborrhoeic Dermatitis  
Scales greasy. Patches spread by aggregation of follicular papules. Scalp, face, centre of chest and back chiefly affected.

(h) Leucoderma  
Loss of pigment complete, with no sensory changes.

(i) Pellagra  
Distribution of skin lesions on parts exposed to sun's rays or friction, are symmetrical.

Margin/
Margin sharply demarcated and pigmented. Glossitis, Stomatitis. History of dietary deficiency. In both of these there are— no sensory changes, no thickened nerves, no Leprae Bacilli.

Round or oval swellings usually confined to shin region of lower limbs, very tender, red or purple colour, bilateral, number variable, size up to two inches. "Erythema Nodosum must be regarded as the result of a non-specific reaction to a variety of infections and toxic agents, and it is not a specific disease". (Parry C.B. B.M.J. Dec. 30, 1944. 846.)

Group 2.

Leprosy must be differentiated from diseases producing loss of cutaneous sensibility.

(a) Beri-beri History of dietary deficiency. Both motor and sensory fibres affected, but former more pronounced. Muscles, especially calf/

(b) Syringomyelia

Dissociated anaesthesia, - loss of sensations of heat, cold, and pain, with retention of light touch.
Vasomotor and trophic changes.
Muscular atrophy and paralysis.
No thickened nerves.

(c) Morvan's Disease

In this form of Syringomyelia there is absolute loss of sensation in the hands and wrists, and often in feet and ankles. There are no thickened nerves, and the area of sensory loss is less definitely limited.
No skin lesions in other parts of body.

(d) Bernhardt's Syndrome

Neuritis of lateral femoral nerve. Numbness and absence of cutaneous sensibility in antero-lateral aspect of thigh.
No loss of hair, no anidrosis, nor thickened nerves.

(e) Cervical Ribs

Pain, paraesthesiae, or vasomotor disturbances in the upper limbs.
Wasting/
Wasting of small muscles of hand.
X-Ray examination of neck.
Difference between radial pulses.

Group 3.

There are certain diseases which produce deformities and lesions resembling those of leprosy.

(a) Diabetes Mellitus

Gangrene, or perforating ulcer may be the initial feature to attract attention. History, and a careful clinical examination will make the diagnosis clear. Arteriosclerosis is the essential factor.

(b) Buerger's Disease

History of intermittent claudication. Palpable thickening of arteries. Vasomotor changes. Pulsation in arteries of feet diminished or absent. Collateral circulation may be present. Progresses with intermissions.

(c) Raynaud's Disease

More common in females. Heredity. Cold is essential factor. Symmetrical and bilateral. Often paroxysmal. Gangrene dry, and final loss of tissue small, e.g. end of one finger.
In both Malaya and India I very frequently found patients with leprosy suffering from other skin lesions. The most common combination I found was leprosy and tinea versicolor, and a combination of leprosy and syphilide was also observed.
It does not require an expert knowledge of leprosy to diagnose an advanced case, since the lesions are so characteristic. Early cases present some difficulty, and certain factors stand in the way of early diagnosis. These are:

(a) A long incubation period which allows many infective cases to move about unrecognised.
(b) Ignorance of the patient, who often considers an early lesion of no significance.
(c) Apprehension that if the lesion be leprosy he may lose his job.
(d) Shame amongst the relatives lest it be known that there is leprosy in the family.
(e) Fear and dread on the part of many doctors of treating cases of leprosy.
(f) Lack of skill and experience on the part of the physician.
(g) Unreliable surveys of leprous patients.

The three important signs in diagnosis are:

1. Anaesthesia to light touch.
2. Thickening of nerves.
3. The finding of Leprae Bacilli.

In the neural type of leprosy bacteriological examination of the skin or nose rarely demonstrates the lepra bacillus.

Leprosy should never be diagnosed unless one of/
of these signs is present along with the clinical manifestations. The whole body should be examined in a good light for areas of loss of pigment or infiltration, which may be so slight that they are difficult to detect. Nerves should be palpated for signs of thickening. Skin sensation of the whole body should be tested. Loss of sensation to heat and cold, then to pain, then to touch, is the order in which the anaesthetic change takes place.

Loss of Sensation.

Anaesthesia is usually found in the macules, or in the distant parts of the limbs. It may be incomplete, and may be more definite in some lesions than in others.

To discover impairment of heat and cold sensation I use hot and cold test-tubes.

The sensation of pain is tested by means of pin pricks, one pin being held in each hand, and the patient is asked which pin he feels most.

When testing for loss of touch I ask the patient to close his eyes, and I draw a wisp of cotton wool lightly over the area to be tested. I then ask him to indicate with a finger the part touched. If he fails to respond there is failure of sensation.

Thickening/
Thickening of Nerves.

No nerve can be said to be thickened merely because it is felt, e.g. the ulnar nerve is often felt in normal persons.

For definite unilateral thickening comparison must be made with the nerve of the opposite side.

For bilateral thickening, the nerves should be compared with those of a normal person of the same age, sex and build.

Examination for Lepra bacilli is usually made to find out if a patient is infectious, or to judge of the progress of a case. It is wise to examine smears from both skin and nasal mucous membrane.

Method for skin examination.

Clip a small portion of skin about 3 mm. in length and 2 mm. in depth from the skin or lobe of the ear with a pair of sharp scissors curved on the flat. This section is pressed corium surface downwards on to a clean slide, a smear is made and dried.

Examination of mucous membrane.

A small piece of membrane from the septum of the nose should be removed with a tenotomy knife. If the surface is ulcerated scrapings should be taken. I deprecate the use of cotton swabs for making smears, as I have frequently found acid-fast organisms present/
present on the surface of the nasal mucosa, which might easily be mistaken for lepra bacilli.

Muir (19) advises "it is well to make smears of standard size and thickness so that the degree of infection may be at least roughly estimated".

Staining of smears.

The Ziehl-Neelsen method is used. Filter carbol-fuchsin on to the slide and let it remain for fifteen to twenty minutes. It is not necessary to heat the slide, if the basic fuchsin is well ground in a mortar beforehand, in order to be thoroughly dissolved in the alcohol. The smear is now decolourised. I have observed that a twenty per cent sulphuric acid is less apt to decolourise than a five per cent acid alcohol, because the lepra bacilli are less resistant to acid alcohol decolourisation than to that by aqueous acid solutions. The slide is now well washed with water, and the tissues regain a faint pink tint. If the colour is distinctly red the decolourisation has not been sufficient, and more acid must be applied. Counterstain the slide with a saturated watery solution of methylene blue for two minutes, then wash well with water and dry.

It must be noted that there is a great variation in the resistance to decolourisation of lepra bacilli,
bacilli, those from one case holding their colour almost as well as tubercle bacilli, while those from another case may decolourise very easily.

I shall now consider the special tests which have been employed:

(a) to confirm the diagnosis.
(b) to evaluate the progress of the disease.

1. **Histamin test.**

I have found this test useful in early cases where the diagnosis is obscure, because a normal reaction definitely excludes the diagnosis of leprosy. It is useless if the skin is too dark in colour. The test is carried out as follows:— One drop of 1:1000 histamine phosphate solution is placed on the affected skin, one drop on the normal skin, one drop in between: a prick is made through these drops without drawing blood, and the drops of histamine wiped off. In normal skin, erythema develops around the prick within 25-45 seconds. In an affected leprous area a wheal develops, but no erythema.

According to Pardo-Castello and Tiant (20), "a negative reaction is not entirely specific for the disease, since in some non-leprous neurologic conditions negative results have been elicited".

2./
2. **Anidrosis test.**

Sometimes there is considerable difficulty in differentiating early neural macules from Leucoderma and certain types of Ringworm e.g. Tinea Versicolor. In such cases the injection intradermally of five drops of Pilocarpine nitrate solution in sterile water (½ gr. tabloid dissolved in 1 cc. of sterile water), and a similar injection into a normal area of skin should be made. The appearance of minute sweat beads to approximately the same extent in both areas, is strong evidence that the lesion is not leprotic. This is a speedy and most useful test.

3. **Iodide test.**

The use of Potassium iodide in treatment was extensively used in India up to a few years ago. The reason for its use was to produce a mild reaction, and thus stimulate the development of immunity. Unfortunately, continuous repeated doses of this drug is frequently followed by disastrous results, such as the appearance of new cutaneous lesions, pain, tenderness and swelling of nerves, pyrexia, headache and malaise, and a rapid increase in the blood sedimentation rate (B.S.R.), - in fact, all the signs and symptoms of a lepro reaction. If, however, the general health of the patient is good, the B.S.R. nearly/
nearly normal, and several examinations for lepra bacilli have been negative, a dose of potassium iodide (20 grs in water) at bedtime on two successive nights, is most certainly of great diagnostic value. It produces a very mild reaction, and cases which had previously been found negative bacteriologically may now be shown to be positive.

4. Blood sedimentation rate (B.S.R.)

The estimation of the rate of fall of the erythrocytes is, in my opinion, of slight value in the diagnosis of leprosy; but is of great value in prognosis and as a guide to treatment. A rapid B.S.R. (other diseases having been excluded) shows that either the disease is in a serious form, or that the resistance of the patient is low. Both conditions, however, may be present. With the onset of leprotic fever the B.S.R. rises rapidly and during this period I immediately stop all active treatment. I have particularly observed that patients respond much better to treatment when the B.S.R. is nearly normal.

I use the well known Westergren method which is very simple.

I am in entire agreement with Henryk Dlugosz (21) who says "B.S. diagrams are very important for the observation of the clinical course of diseases, because there is a close parallelism between the B.S. diagrams/
diagrams and the condition of the patient".
I have been using B.S. diagrams for several years as a routine measure.

5. The Lepromin test.

This test is performed by the intradermal injection of 0.2 cc. of a suspension of killed lepra bacilli, obtained from the nodules of lepromatous cases. The results of the test indicate the power of reaction of the tissues to the lepra bacillus. In healthy persons showing the mild neural type of the disease, this test gives a positive result showing strong reactive power. Nodules appear at the sites of inoculation between the 1st and 3rd week and increase gradually in size. In young children, in adults with the lepromatous form of the disease, and in debilitated persons, the result of the test is weak or negative, showing absence of reacting power.

The test is of no value in diagnosis, but is of considerable value in prognosis, as it is a means of measuring the resistance of the patient.

Brilliant work is being carried out in this field by Dharnendra and Lowe (22). They have advanced the technique of this test by grinding up the bacilli in order to free their contents, whereby the injection produces much earlier distinct reactions, and greatly diminished late reactions.

Cochrane/
Cochrane et al (23) reporting on the lepromin test in children, conclude that the lepromin reaction tends more often to be negative in children in whom a history of contact with leprosy cases is maximal, but it is not significantly influenced by hereditary disposition. They state that continuous contact with an open case is the most important factor in breaking down resistance to infection.

6. The Kahn and Wassermann reactions.

In severe cases of lepromatous leprosy both Kahn and Wassermann reactions are frequently positive, even in the absence of syphilis or yaws.

When there is any doubt as to the result of a Wassermann or a Kahn reaction the Kahn verification test should be carried out, if practicable.

Beveridge (24), applied the Kahn verification test to 335 sera of syphilitic and non-syphilitic patients, to determine whether it was more sensitive than the Wassermann or Standard Kahn tests. He concluded that the verification test was valuable where normal serological tests were doubtful, or where the clinical condition of the patient did not justify a diagnosis of syphilis.

Kahn (25) states, "these tests do not offer a practical means for the detection of false positives in routine serological practice".

The/
The Kline reaction proves of value in differentiating between syphilitic and non-syphilitic leprous patients, as it never, to my knowledge, gives a positive response in the absence of syphilis as do the Wassermann and Kahn reactions. Windsor-McClean (26) has carried out its investigations on a large number of Nauruan lepers and non-lepers, and his conclusion is, that there is no evidence that active leprosy tends to produce a positive reaction to the Kline test.
Leprosy is often a mild disease. Many cases completely recover without treatment.

Certain factors must always be kept in mind before giving a considered opinion.

1. Children have a poor resistance to any type of leprosy. Even in mild cases prognosis must be guarded as they may change later into more severe forms. Muir (27) states that the lepronin test demonstrates that children's tissues give a poor response to the disease.

2. Adults. In neural leprosy the prognosis is good since resistance is high. In lepromatous or mixed leprosy, prognosis is of necessity very guarded. Long treatment is necessary. The lepromin test shows that the cellular defensive mechanism of the body gives a poor response.

3. Physical condition of patient. In my opinion malnutrition and the presence of other diseases makes prognosis doubtful in the neural type, and serious in lepromatous or mixed leprosy.

4. Climate. Prognosis is better for patients in a temperate climate than in the tropics, where humidity is high.

5. Race. I have frequently observed that leprosy in all its forms is more serious in Europeans and Eurasians than in the native population.
6. Duration. If the disease has been present in mild form for a long time, it is unlikely to develop into the severe form; but, if developing rapidly, the prognosis is more serious.
A REVIEW OF THE DRUGS ETC. WHICH HAVE BEEN USED IN THE TREATMENT OF LEPROSY.

No drug, so far as is known, has a specific action on the lepra bacillus. The ideal drug for the treatment of leprosy should satisfy the following points:

1. A drug which rapidly controls and eliminates the disease.
2. A drug which is cheap.
3. One which is painless and easy to give by injection.

It can be said at the outset that there is no drug in use at present which satisfies these criteria.

Various attempts have been made in the past to produce immunity by:

I. injections of autogenous vaccines,
   " " tuberculin,
   " " Deycke's nastin,
   " " sera,
   " " bee-sting venom,
   autohaemotherapy,
   various forms of protein shock,
   diphtheria toxoid,
   strychnine; arsenious acid,
   heavy metals such as - antimony, gold,
   mercury, copper.

II./
II. But the remedy which has been used more than any other are the Chaulmoogra and Hydnocarpus oils. These have been known for centuries and their popularity has waxed and waned throughout the years.

Tai Foong Chee is a treatment for leprosy widely used in Malaya amongst the Chinese. Chinese doctors informed me that it has been used in China for hundreds of years. Tai Foong Chee is the Chinese name for Hydnocarpus anthelmintica. It is given orally in the form of a powder, with a small amount of hempseed. (cannabis indica). I have never found it mentioned in the literature, nor could any Chinese doctor show me any reference to it in their medical books.

Chaulmoogra oil is an old Indian remedy for leprosy, and was introduced to European medicine by Dr Mouart of the Madras Medical Service. The drug was for long used orally, but owing to its nauseating properties and to the fact that it is a gastric irritant it proved neither popular nor very successful.

In 1849-95 Danielssen and Hansen used these oils, and in 1935 the Norwegian authority on leprosy Dr H.P. Lie (28), in reviewing their work, stated, that the oils "seemed to have no healing qualities; in fact Danielssen found it detrimental".

In/
In 1904, the nature of the acids peculiar to the Chaulmoogra and Hydnocarpus oils was discovered. It was not possible to utilise the results in medical practice until serious clinical research in leprosy was undertaken, especially in India and the Philippines. The value of esters of these acids was reported on, following clinical trials in Hawaii, India and Malaya, and their use has become widely adopted as the routine treatment of leprosy.

Hiser in 1914, introduced a mixture consisting of,

Chaulmoogra oil 60 ccs.
Camphor oil  60 ccs.
Resorcin  4 grms.

The injections, which were given intramuscularly, were very painful.

In 1916, Rogers (29) introduced sodium gynocardate for subcutaneous administration, and this proved an advance on the previous oral method. He also gave the preparation intravenously, and reported good results, much more rapid improvement taking place than when it was given subcutaneously.

Unfortunately, vein blocking frequently occurred after a series of injections.

In 1919 McDonald and Dean (30) in Hawaii reported success with the ethyl esters of Chaulmoogra oil by hypodermic injection and stated that 25% of patients/
patients were "apparently clinically and bacteriologically free from the disease".

In a later paper (31), the same authors reported that patients were automatically placed on this treatment on admission to the Leprosy Receiving Station in Hawaii, and claimed that now 50% of the patients so treated were improved. Unfortunately, these conclusions were drawn from clinical experiment only over a period of approximately 18 months.

During 1925, Muir of Calcutta (32), introduced the subcutaneous injection of pure Hydnocarpus Wightiana oil, expressed cold from the seeds of Hydnocarpus Wightiana fruit, and with 4% of creosote added as an antiseptic. He reported good results from this form of treatment.

In 1926 Messrs Burroughs Wellcome and Co. introduced "Alepol", - the sodium salts of a selected fraction of the lower melting point fatty acids of Hydnocarpus oil. This preparation can be used subcutaneously, intramuscularly or intravenously.

About the same time the above firm (33) introduced 'Moogral' to India. It is a mixture of the esters of the Chaulmoogric series, and is a clear almost colourless oil suitable for intramuscular injection (31).

In 1929 Wayson (34) reported the results of his study of the value of treatment in the Hawaii Leper Settlement.

"The/
"The use of Chaulmoogra oil and its derivatives in Hawaii for ten years, has not been attended by results which indicate that they have any specific therapeutic value, and any effect they may have remains to be determined".

Lara (35) believes that Chaulmoogra oil is of great value in suitable cases, but should be used only by those who have a long and close experience of leprosy; otherwise, disastrous results will assuredly follow.

Tomb (36) in 1930 stated, "There is no proof that general dietetic treatment, plus Chaulmoogra oil, yields better results than general dietetic treatment alone. No conclusive evidence exists of the efficiency of Chaulmoogra oil as such."

At the Leonard Wood Memorial Conference on leprosy, held in Manilla in 1931, strong recommendation was made for the use of the iodised esters particularly by the intradermal method. Iodised esters are prepared by adding 0.5 per cent iodine to the esters of the Chaumoogra series. The advantages claimed for this method are:-

1. It produces more rapid resolution of the superficial lesions.
2. It is relatively free from general or local reactions.
3./
3. It is particularly useful in the following types of lesions,
   raised macules,
   nodules,
   infiltration.

The disadvantages, on the other hand, are:

1. It is extremely painful to some patients.
2. The injections frequently produce a blue pigmentation, which may last for a long time at the site of injection.

Rogers and Muir (37), in discussing the chemotherapy of the Chaulmoogrates and Hydnocarpates and in advising their frequent use state that the action of these preparations cause:

(a) A dissolution of the fatty coats of the lepra bacilli.
(b) The setting free of antigens resulting in,
(c) Frequently repeated, mild lepra reactions
(d) whereby the defensive mechanism of the body is stimulated.

As late as November 1942 McCoy (38) writes "My own observations have led me to the conclusion that the oil and its derivatives are of little or no curative value, and that the unpleasant side effects probably outweigh any advantage of the patient that might possibly accrue from their use".

Frazier/
Frazier (39) states that "Long periods of latency alternating with unexplained relapses give rise to scepticism about the beneficence of hydnocarpus oil and other drugs".

Truly the popularity of the Chaulmoogra and hydnocarpus oils has waxed and waned for hundreds of years.

III. The Sulphonamides.

In the last few years Faget and Johanssen (40) have administered sulphanilamide to leprous patients with secondary lesions. These workers on a small group of cases concluded: "sulphanilamide therapy has proved effective in the treatment of secondary infections complicating leprosy, and is a help in the healing of secondarily infected leprous ulcerations.

Sulphanilamide cannot be regarded as a curative agent for leprous lesions, either of the macular or lepromatous type".

Later, a group of surgeons at the National Leprosarium, Carville, U.S.A. (41), began treatment of leprosy in 1941 with Promin, "the sodium salt of p.p. diaminodiphenylsulfone n.n. didextrose sulfonate".

They consider Promin superior to any of the other sulphonamide derivatives including sulphanilamide, sulphathiazole, sulphapyradine and sulphadoxine.

They/
They are greatly encouraged by the results of their experimental treatment. Their conclusions are:

1. "Promin appears capable of inhibiting the progress of leprosy in a considerable percentage of cases, although no case has become arrested under its influence.

2. Promin can be safely administered intravenously for prolonged periods, provided the blood and urine are examined frequently. When these precautions are taken, toxic manifestations are relatively rare and mild.

3. Promin can be considered to have opened a new avenue in the chemotherapy of the mycobacterial diseases. It is hoped, that further synthesis of the sulfa compounds may produce a substance, which will succeed in saving countless lives in this still dark field of medicine".

Muir (42) has used Promin at Trinidad and reports good results.

IV. Penicillin — Will this prove of value? as it has done in the case of Actinomycosis Bovis. Research has still to be carried out in this field.
To summarise, -

1. At the present time there is no specific drug for leprosy, although many workers claim success from the use of the "oils"; others consider these useless.

2. Research goes on, - the sulphonamides appear to provide a useful avenue of approach.

3. Workers of great experience are more and more forming the opinion, that the well-being of the patient plays a larger role than any drug in overcoming the disease.

4. My own view is that the Chaulmoogra and Hydnocarpus oils are most valuable if administered after the well-being of the patient has been carefully improved.
MALAYAN EXPERIENCES IN THE
TREATMENT OF LEPROSY.

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MALAYAN EXPERIENCES.

During the years 1924–1930 in the Malay States, I treated all my cases of leprosy on the orthodox lines laid down by the then modern authorities.

Gradually I began to realise, however, that certain patients were not progressing as well as I should have liked; that the oils of the Chaulmoogra and the Hydnocarpus group, if given too frequently and in large doses, desensitised many patients, with improvement of signs and symptoms up to a point, after which the oils had no beneficial effect. On the other hand, well-nourished patients continued to improve. It was most disappointing and discouraging.

I came to the conclusion that these oils were having a lowering effect on the defensive forces of some patients, and these were the poorly nourished ones!

At the same time I began to institute a careful inquiry into, and kept records for each patient of:

- weight,
- dietary habits, especially in respect of rice,
- type of disease, and
- response to treatment.
After a period of one year of this I was encouraged to believe that an adequate and balanced diet was of the greatest importance in the treatment of leprosy.

I had observed that patients with neural leprosy were invariably better nourished than the sufferers from the severer lepromatous type of the disease.

During the years 1932, 1933 and 1934, on investigating the diets of 145 cases of leprosy, I found that the diet of the low resistant type was invariably poor in—

- protein (generally obtained from rice),
- fat,
- fresh vegetables,
- vitamins.

It must be noted that the different Malayan races are not uniform in their choice of rice as a staple diet.

It is a well known fact in Malaya that the Tamils enjoy excellent health on a diet of Bengal rice and vegetables — the reason for this being that they use an unpolished or parboiled rice with a high percentage of protein and vitamin B1.

The Tamils in the Malay States suffer little from leprosy.

The Chinese, on the other hand, prefer a polished/
polished rice from which the huskings have been removed. This rice has a low protein value and has no vitamin B1.

The majority of lepers in Malaya are Chinese.

I decided that I was not paying sufficient attention to the preparation of the patient for treatment. I resolved that in future I would increase the patient's resistance to the highest point possible, before giving any direct treatment.

During the year 1935 I made the following experiment. From the 145 patients whose diet I had investigated I selected 30 of whom 15 were suffering from the neural type of Leprosy, and 15 from the early lepromatous type of leprosy. I divided them into three groups. All had been under treatment for from six months to one year, and all had reached a point when there was no evident improvement in their lesions. They were becoming discouraged, and some of them told me quite frankly that the injections (Moogral - a mixture of esters of acids of the Chaulmoogric series) which they were having, were not doing them the slightest bit of good. I agreed with them.

All these men were Chinese of the coolie class, accustomed to the same type of food. Their average age was 24 years. All had negative Wassermann and Kahn/
Kahn reactions, and, as far as I was able to ascertain, all were free from any other disease. Erythema, thickened macules and nerves, nodules and neuritis were present in varying degrees in each one.

I placed five lepromatous and five neural cases in each of the three groups.

My experiment lasted for two months. To each group was given treatment for ten days, rest for ten days, treatment for ten days, and so on until the end of the two months.

To Group I, I gave an intramuscular injection of 1 cc. Campalon every day for the first ten days. 2 ccs. " " " third " " 2 ccs. " " " fifth " "

Each patient of this group received 50 ccs. of Campalon in two months. I used liver empirically, purely as an adjuvant to diet. Later, and with more knowledge, I was to discover that there was far more in liver than protein and haemopoietic principles.

With Group 2, I followed the same procedure in spacing. To each patient I gave on each of the thirty days of treatment, an intramuscular injection of Sod. Cacodylatis gr.1, in 1 cc. saturated Acid Nucleici. These drugs were given for their tonic effects, and for the purpose of producing a leuco-cytosis.
To Group 3, which acted as a control, 1 cc. of sterile distilled water was injected intramuscularly on each of the treatment days.

The results of this experiment were most gratifying. All cases of Group 1 showed improvement of their lesions, some more than others. The erythema in some cases had entirely disappeared, and in several, thickened macules had become much thinner, and I remember that in one case the macules did disappear. I am sure that, at that time, even the most careful examination could not detect any evidence of active leprosy. This does not mean that these patients were cured.

All the cases of Group 2 said that they felt much better in health and that their appetite had improved; but I observed little change clinically in their lesions.

Even the members of Group 3, who were under the impression that they were having special treatment, said they felt better, and undoubtedly their spirits were raised. That also taught me a lesson!

I was so much encouraged with the above results that I decided to try the same experiment, using Campalon only, with cases who had had no previous active treatment for leprosy.

I collected 7 cases of advanced leprosy all of whom, as far as I could ascertain, had never been given/
given injections of any kind hitherto. It was impossible to eliminate the possibility of their having had treatment orally, in the form of the aforementioned Tai Foong Chee which is a very popular remedy for Leprosy with the Chinese. Four of this group of cases were of the advanced lepromatous type which is described by most authorities as unsuitable for active treatment. The remaining three cases were of the advanced neural type, with numerous thickened erythematous macules, and thickened inflamed nerves. All cases had had frequent attacks of leprotic fever.

Before commencing treatment, each case was carefully examined to exclude other diseases. A careful record of all leprous lesions was made on charts. Routine examination of blood, faeces and urine was carried out in each case. Lastly, on the first day of treatment an estimation of the sedimentation rate of the red blood corpuscles was made also. It was found to be much increased in all, but especially so in the lepromatous cases. Westergren's method was used, and the readings at the end of two hours varied between 45 mms. and 120 mms.

The 7 cases were divided into two groups. The two most advanced cases of each type were placed in Group 1, the other three formed the control Group 2.

Group 1 was treated as follows:—
On this occasion liver extract (Campalon) alone was given intramuscularly as before.

2 ccs. Campalon every day for the first ten days
3 ccs. " " " " third " 
3 ccs. " " " " fifth " 
No treatment was given during the second, fourth and sixth periods of ten days.
Group 2 was given injections of sterile distilled water as before.

The B.S.R. was estimated for each patient every fifth day during the whole period of two months. At the end of one month the B.S.R. was very slightly reduced in the two neural cases, and by the end of the second month there was also a slow continuous reduction in the lepromatous cases, and a further reduction in that of the neural cases.

I did not feel sure at that time whether this decrease in B.S.R. was due to an increase in the number of the red blood corpuscles, or was due to the specific action of the liver extract in supplying some deficiency in the patient's diet.

The four patients in this group appeared mentally brighter, and they admitted that they felt better. This was most encouraging, and when they were compared with the control group the improvement was more noticeable.

I now decided to carry on the experiment for another/
another three months. I was most interested to see if the sedimentation rate would fall still further, and if any decided improvement would appear in the patient's lesions.

Treatment was given at ten day intervals as before. Each injection of 2 ccs, of liver extract (Campalon) was given intramuscularly every second day. At the end of three months each patient had received 50 ccs.

E.S.R. estimations were made every fifth day and it was found, that the slight decrease in fall, observed during the first two months, became more rapid. At the end of the five months the E.S. rates in all cases had fallen to 15 mms. and below. (two hour reading).

There was no attack of leprotic fever during the five months in Group 1, whereas, amongst the members of Group 2 attacks occurred not infrequently.

There was a decided improvement in the lesions, erythema was less marked, and nodules did not appear to be so prominent. There was certainly a further improvement in the general condition, which was reflected in their mental attitude. All cases had increased in weight.

From the above experiments I formed the opinion, which has been verified by me on numerous occasions since then, that extract of liver definitely increases the/
the resistance of patients, even if they be advanced cases of lepromatous leprosy which are classified by most workers as unsuitable for direct treatment. I considered that an intensive preliminary treatment with liver to be of immense value, provided that all accompanying diseases were dealt with, and the patient's B.S.R. reduced to 15 mms. or less (two hour reading), before any direct attack against the leprosy was made.

It seemed to me that, as the staple diet of the Chinese is polished rice, which is deficient in protein and vitamin B1, the preliminary injections of extract of liver was supplying, to some extent, these deficiencies. Rosedale (43) 1939, has demonstrated the nutritive properties of polished and unpolished rice in regard to protein and Vitamin B1.

<table>
<thead>
<tr>
<th></th>
<th>Ozs.</th>
<th>Calories</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unpolished rice</td>
<td>21.4</td>
<td>2,200</td>
<td>53 grms.</td>
</tr>
<tr>
<td>Polished rice</td>
<td>&quot;</td>
<td>&quot;  &quot;</td>
<td>38 grms.</td>
</tr>
</tbody>
</table>

Polished rice contains no Vitamin B1.
Unpolished rice contains \( \frac{1}{10} \) Vit. B1 content of yeast.

It is clear, therefore, that the rice eaters of the poorer classes, (ninety per cent of whose diet consists of rice,) are not having an adequate or/
or balanced diet. Their diet is deficient in protein, fat, fresh vegetables, vitamins.

During the past years interest has been shown more and more by investigators in the close relationship between nutrition and leprosy.

Hutchison, when he formed the theory that leprosy was due entirely to the consumption of decomposed fish, may not have realised, and certainly failed to point out, that the people who ate this fish lived in famine areas and were already ill-nourished.

Lamb (44), reporting on his investigations into rat leprosy, states "upon using intracardia inoculations of rat leproma, diets deficient in the vitamin B complex, and somewhat low in protein produced an extensive increase in visceral lesions of rat leprosy".

Badger and Sebrell (45) in 1935, found, that the incubation period of rat leprosy in white rats on a vitamin Bi deficient diet was much shorter than in the rats fed on a well balanced diet.

Badger and Wolf (46) 1940, after various experiments on groups of rats, reported on the effects of malnutrition on rat leprosy.
Their findings are worth quoting.—

"The leproma, a granuloma, is believed to be formed by the infiltration of cells, and the development of fibrous tissue, and is an attempt by the animal organism to overcome the infection. The well-nourished animal is better able to attack the infective organism and build up a better defence, hence the larger lesions at the site of subcutaneous inoculation. In the malnourished animal the defence mechanism has been affected, and is less able to attack the infecting organism, hence the smaller lesion at the site of subcutaneous inoculation.

From these studies it appears, that the shorter incubation period and the smaller lepromata at the site of subcutaneous inoculations in the vitamin Bi deficient rats were due to an interference with the cellular defence mechanism brought about by a state of general malnutrition, and not specifically to vitamin Bi deficiency."

In 1939 Concepcion and Camara (47) reported, that they found a deficiency of ascorbic acid in the blood of 96 cases of leprosy. "It was reduced in the plasma in more or less direct proportion to the severity of the cases. The normal level was restored by the intramuscular injection of 50 milligrams."
confirm my own views most emphatically, in regard to the Chinese coolie class at any rate – that their food is plentiful in quantity but grievously inadequate in the essentials of a well-balanced diet.

I formed the following rules regarding my future treatment of leprosy, and to these I have strictly adhered to, with, I must say very successful results. I decided that the B.S.R. should be my best guide in future treatment, that occasional estimations are useless, and only a series of readings taken at regular and frequent intervals are of any value. These can easily be recorded in diagrammatic form on the record cards, and such a series provides a reliable record of progress.

The Rules I made were these:–

1. A careful history must be taken of each patient. He must be stripped and examined thoroughly; and the type, stage and lesions of the disease must be recorded and charted. A complete examination of the blood, including Wassermann and Kahn reactions, and of the urine and faeces must be carried out in all cases.

2. If the patient has an accompanying disease such as malaria, dysentery, hookworm or other worm infestation, or pyorrhoea, (the latter is very common and severe amongst the semi-aboriginal tribes of India), these must be treated and cured, as/
as far as possible, before beginning the routine treatment of leprosy. Also, if pulmonary tuberculosis is suspected the sputum must be examined repeatedly for tubercle bacilli, X-Ray photographs of the chest taken, and appropriate treatment instituted.

3. A preliminary course of treatment with intra-muscular injections of liver extract is to be given to all cases whether slight or severe. No direct treatment against leprosy is to be given if the B.S.R. is above 15 mms.

4. An estimation of the B.S.R. must be made before each weekly injection, during the direct treatment against leprosy. If the rate is found to be increased from that of the previous estimation, direct treatment must be stopped, and a course, or part of a course of preliminary treatment given, the length of the latter depending on the response of the B.S.R.

5. A very careful account of the patient's diet must always be obtained, and advice given in simple language as to value and choice of food-stuffs especially if any deficiency is found present.
THE DRUGS USED BY THE WRITER DIRECTLY AGAINST LEPROSY.

These are:-

1. A mixture of Esters of Acids of the Chaulmoogric series (Moogral).

2. Iodised Esters.

All drugs of the Chaulmoogric and Hydnocarpic series have a depressing effect. They produce a negative phase, which varies directly in length and degree with the size and frequency of the dose given.

In order to avoid a negative phase I never give any injection more frequently than once weekly, and never give the large doses recommended by most authorities. I have used the esters of the Chaulmoogric series, (following the preliminary treatment with liver extract,) for the past eleven years and I have been very satisfied with my results.

Administration.

All-glass syringes should be used, as they are easily cleaned, and I find that needles of a medium bore are the most suitable.

Many a medical practitioner gains a reputation by giving painless injections, and very often wins new patients because he pays attention to minor details. The secret of success is always to use sharp-pointed needles, and to give injections slowly.
I have, for many years, made a practice of examining all needles myself once weekly, under the low power of a microscope, and at the same time of sharpening those requiring attention. This process usually occupies a few minutes only, and the time spent is amply repaid by the thanks of a grateful patient. All needles and syringes should be sterilised immediately before use.

I give all injections intramuscularly into the gluteal region above a line joining the anterior superior iliac spine and the fold of the buttock, and, as the injections are given weekly, alternate sides are used. When a dose of 3-5 ccs. is being administered, I partly withdraw the needle when half the dose has been injected, and reinsert it at a different angle and give the second half of the dose. After the injection, the area should be massaged firmly, in order to facilitate absorption, and lessen any pain. Moogral rarely causes any pain if the injection is properly administered.

Esters, and Iodised Esters, are usually delivered in 100 cc. bottles. On receipt, I replaced the cork with a rubber cap which is secured firmly round the neck of the bottle. The bottles are then placed in an autoclave for half an hour at a temperature of 120°C. Esters, and particularly iodised esters, tend to/
to become irritant if exposed to moisture. It is, therefore, a good plan to draw a little ether into the syringe, and to discharge it before inserting the needle through the rubber cap, which had previously been cleaned with alcohol.

On injection days, the bottles containing the drugs for injection are placed in a basin of hot water. This makes the preparations less viscid, and therefore much easier to inject, also the warmed drug is less painful.

The weekly dose is increased progressively so long as the B.S.R. is satisfactory, and there is no fever and general disturbance. If the patient has any fever, or has any constitutional disturbance, and the B.S.R. is raised, no injection is given. If the patient misses one injection, he receives the same amount as his last dose on resumption of treatment; but, if he misses two or more injections, I find it safer to resume treatment at the beginning of his last stage.

The length of treatment varies in different cases, and on the duration of the disease before treatment. The rapidity with which active lesions disappear must also be taken into account. I carry on treatment, if possible, until all active signs of the disease have been absent for six months and repeated bacteriological examination of skin, mucous membranes/
membranes and lymph nodes on puncture have proved negative also for six months. After treatment is finished each patient is advised to return and be examined every three months for a period of two years.
ROUTINE COURSE OF TREATMENT OF LEPROSY.

Adults.

This consists of a course of injections given to all patients from 14 years of age upwards. The course is divided into four stages:—

Stage 1.

The first injection consists of 1 cc. of Ethyl Chaulmoograte (Moogral) given intramuscularly. Injections are given once weekly, and increased by \( \frac{1}{2} \) cc. each time until 5 ccs. are reached. This latter dose is repeated five times. At the end of each stage the patient is given two weeks' rest.

Stage 2.

The second stage commences with a 1 cc. injection of Moogral. This is increased by 1 cc. weekly until 5 ccs. are reached. This latter dose is given ten times.

Stages 3 and 4 are the same as Stage 2.

I must emphasise that the B.S.R. is estimated before each injection, and, if there is an increase in the rate of fall of erythrocytes from that of the previous reading, no injection is given, and a course of preliminary treatment is started.

It has been observed, that attacks of leprotic fever/
fever take place frequently following the administration of the Chaulmoogric and Hydnocarpus oils. I have found however, that severe attacks of leprotic fever are a rare occurrence if the patient is efficiently prepared beforehand. Mild attacks sometimes occur when the larger doses of Moogral are given, and these attacks are, in my opinion, distinctly beneficial, if not too frequent nor too prolonged.

Very occasionally, I substitute one or two doses of Iodised Moogral for the plain Moogral, if the patient has had no attack of leprotic fever, and is responding too slowly to treatment. This causes a very mild reaction and the patient's progress becomes noticeably more rapid.

A full course of treatment takes approximately one year to complete. Many of the milder neural cases, however, respond to treatment so quickly that by the end of the second or third stage all signs or symptoms of the disease have disappeared. The more severe lepromatous cases always require a full course, and often part of a second course before bacteriological examination becomes negative.

**Children.**

In children from 8 - 14 years of age, the routine course of treatment consists also of four stages, with a rest from treatment of two weeks between each stage/
stage. The injections are administered in the same way as to adults.

Stage 1.

The first injection is \(\frac{1}{3}\) cc. of Moogral and this is increased by \(\frac{1}{3}\) cc. weekly until 3 ccs. are reached. This latter dose is repeated five times.

Stages 2, 3 and 4.

Again the first injection is \(\frac{1}{3}\) cc. Moogral, and is increased by \(\frac{1}{3}\) cc. each week as before, but the last dose is repeated ten times.

In younger children from 4 - 7 years of age the same course of treatment is given, but the maximum dose of Moogral reached in any stage is 1\(\frac{1}{2}\) ccs.
THE LEpra REACTION AND ITS TREATMENT.

The Lepra reaction is a condition which should be familiar to, and easily recognised by all who treat leprosy. I think that it can quite rightly be called a complication of leprosy, because it gives rise at times to a considerable degree of fever and debility.

It occurs in both treated and untreated cases, but I have seen it in its most severe form in weak and undernourished patients.

A similar and almost indistinguishable reaction occurs in leprous patients under treatment with potassium iodide.

The nature of the phenomena of lepra reaction is not yet clearly understood. Some observers believe that it is due to allergy, and is similar to the sensitisation phenomena seen in a number of other diseases, while others consider that the reactions are exaggerated stages of the leprous process. I am of the opinion that the former theory is the correct one.

It is most important that this complication should be diagnosed correctly and quickly, because, if it is not, the active treatment against leprosy is continued, serious results will assuredly follow.

The signs and symptoms of Lepra Reaction are:

1. Fever, headache and joint pains.

2. Swelling and increasing erythema of lesions.

3./
3. Swelling of nerves and neuritis.
4. Appearance of fresh lesions.
5. The B.S.R. is always much raised during the reaction.

Mild attacks may last for 24 hours only, more severe attacks for four to five days or longer. They generally recur quite regularly every two or three weeks for long periods, and with each attack fresh lesions appear, and the untreated patient goes steadily downhill.

The most useful and reliable drug I find for the treatment of these reactions is Potassium Antimony Tartrate (P.A.T.). This drug, if given early, invariably brings down the fever and causes subsidence of fresh lesions in a very short time.

P.A.T. can be obtained in tablet form, each tablet containing 0.04 grammes. This enables a fresh solution to be made up for each injection. Usually two injections are sufficient, but occasionally, if the reaction is severe, four or five injections may have to be given. These are given every second day intravenously, and the dose is increased by half each time.

The following table shows the course; it also shows the amount of distilled water required for each injection.
<table>
<thead>
<tr>
<th>Day</th>
<th>P.A.T.</th>
<th>Distilled water</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>1 tablet</td>
<td>2 cos.</td>
</tr>
<tr>
<td>Third</td>
<td>1½ &quot;</td>
<td>3 ccs.</td>
</tr>
<tr>
<td>Fifth</td>
<td>2 &quot;</td>
<td>4 ccs.</td>
</tr>
<tr>
<td>Seventh</td>
<td>2½ &quot;</td>
<td>5 ccs.</td>
</tr>
<tr>
<td>Ninth</td>
<td>3 &quot;</td>
<td>6 ccs.</td>
</tr>
</tbody>
</table>

In neural cases, neuritis may be very severe. The subcutaneous injection of 0.5 cc. of adrenalin hydrochloride 1:1000 solution, often gives quick relief. Ephedrine sulphate gr ⅓ thride daily is very useful in obstinate cases, but sometimes one remedy after another has to be tried before the pain can be relieved.

The lepra reaction, even if untreated, will subside of its own accord, but it will leave the patient in a condition worse than he was before the attack. I cannot emphasise too strongly that appropriate treatment should be given early in the lepra reaction, as it will save the patient untold suffering.
THE TREATMENT OF SECONDARY LESIONS.

a. Perforating ulcers.

These usually occur in the hands and feet, most frequently in the latter, and are caused by the destruction of the nerve supply to the part. Perforating ulcers never discharge leprae bacilli and are not a source of infection.

The treatment depends upon the depth of the ulcer and the state of the nerve supply. If the nerve supply is much affected healing is very difficult. Any bone which is affected should be removed. The wound is then dissected out and the edges undercut and brought together with deep sutures. The patient is kept at rest with the part elevated. When healing of trophic ulcers of the feet has taken place the patient is allowed to take exercise on crutches, or a special padded shoe may be used so that no weight is put on the affected part.

(b) Lepromatous ulcers. These ulcers heal rapidly when dressed daily with shark liver oil which has a very high vitamin A content.

Muir (48) advises the painting of lepromatous ulcers with a one per cent solution of gentian violet or methyl violet in alcohol, then with a ten per cent aqueous solution of tannic acid three or four times on the first day, and later once a day. With this/
this treatment the ulcers rapidly heal, greatly to the comfort of the patients.

c. **Ulcers in the nose.**

These are caused by the breaking down of lepromatous nodules in the mucous membrane. Such ulcers discharge enormous numbers of lepra bacilli, and are serious sources of infection. The cartilaginous part of the septum is most often affected and frequently perforates. Contractures follow, leading to depression of the nose.

I have found that the application of a saturated solution of iodine in pure carbolic acid every third or fourth day, is most effective. The application is painless, and the lesions clear up rapidly. When the disease is arrested and depression of the nose is present, an operation may be necessary to improve the appearance.

d. **Thickening of nerves and nerve abscess.**

The ulnar nerve is the one most commonly affected. If nerve pain persists, following lepra reaction, and the nerves become much enlarged and an abscess develops, with resulting neuritis and paralysis, a local anaesthetic should be given, and a linear incision of the nerve sheath made for three or four inches over the enlarged part. This is frequently found to be just above the elbow joint. (See Illustration page 34)
The nerve must never be stretched. It is a useless and harmful procedure. An analgesic, given orally, is always necessary.

e. **Eye lesions.**

At the first indication of eye trouble all active anti-leprotic treatment must be stopped.

In the neural type of leprosy the eye lesions are due to involvement of the fifth and seventh cranial nerves with —

- anaesthesia of the cornea,
- paresis of the orbital muscles,
- derangement of the protective mechanism of the eye, rendering the eye susceptible to injury.

It is not surprising, therefore, that conjunctivitis and corneal ulcer are of frequent occurrence in these cases.

If protection only is required, a few drops of liquid paraffin should be dropped into the eye several times daily, and a piece of lint soaked in it should be tied over the eye at bedtime. During the day the patient should wear goggles with side screens for protection.

If a corneal ulcer or iritis are present, the following ointment will be found most beneficial. It diminishes pain, dilates the pupil, and has a tendency to clear up slight opacities.
It is prepared as follows –

**Dionin**

**Atropine Sulphate**

**Ungt. Hydrarg Ox. Flav.2%**

**Vaseline**

The patient should be given a good purge, and if necessary leeches may be applied to the temples.

Prendergast (49) reports a careful study of eye lesions of leprosy made at the U.S.A. Leprosarium. He states that Quinine bisulphate ointment and thyroxin used locally were fairly effective in corneal lesions, and protein shock therapy gave good results in acute cases.

Various operations have been devised by Gass (50) to join the outer part of the upper eyelid to that of the lower eyelid, and thus to protect the eyeball.

In lepromatous leprosy, the eye lesion is a leprous infiltration of the conjunctiva, cornea, and uveal tract causing irido-cyclitis. Symptoms are usually chronic, but, during an attack of lepromatous fever, they may become acute.

My experience of the local treatment of these lesions has been on the whole, not altogether encouraging, and any slight success I may have had has, I believe, resulted more from my attention to the general nutrition of the patient, than from local treatment.

Atropine/
Atropine must be used immediately a diagnosis is made, in order to prevent fixation of the pupil. Various antiseptics injected subconjunctivally have been used by different workers. I find that the best antiseptic is a solution (1:1000) of trypan blue in distilled water, and an amount sufficient to cause an appreciable swelling of the conjunctiva must be used. A course of Potassium Antimony Tartrate in the same dosage as that which I have advised for the treatment of the lepra reaction, is given at the same time.

If the general condition of the patient improves, the eye infection gradually recedes.

(f) Deformities.

Much can be done by operation to restore function to contracted limbs. It may be necessary to amputate a finger or a toe, in order to get a useful hand or foot. Greatly thickened or pendulous ear lobes can be trimmed to a normal size and shape with the aid of Muir's clamp, which has been specially designed for this purpose. An anaesthetic is not required, as, owing to the deep analgesia and the pressure of the clamp, no pain is felt.

Many crippling conditions, such as contracted fingers, can be avoided if massage and exercise are started early, and I find that ultra-violet radiation is most helpful in the healing of ulcers.
Conclusion.

The treatment which I have described in the foregoing pages and which has gradually evolved during 17 years' practical experience in Malaya, is, in my opinion, most efficacious.

Combined with the Plan of Campaign for control of leprosy in India which I am about to describe, I am confident that this terrible scourge could be almost completely eradicated in two to three generations.
Farewell to Malaya.

My work in Malaya came to an abrupt close in February 1942. It is with mingled feelings that I remember those patients who were at the time undergoing treatment, whose case histories and charts I so diligently recorded, who co-operated so heartily and so patiently in their own treatment, and whose whole outlook seemed to be coloured and brightened by their visits to the clinic.

Bitter as it was for me to lose those possessions, it was for them the deferment of Hope which maketh the Heart sick.
Plan of Campaign for India.

1. Problems.

2. Theoretical Solutions.
   A. Education of the Public and of the Medical Profession.
   B. Careful estimate of number of Lepers.
   C. Provision of free Treatment.

3. The Actual Ways and Means.
   The Past.
   The Present.
   The Future.
   a. Central Institution.
   b. Out-Patient Clinics.
   c. Complete Unit.
PLANNING A CAMPAIGN IN A COMMUNITY WHERE LEPROSY IS ENDEMIC.

The problems that present themselves in the diagnosis and treatment of leprosy are practically the same the world over. But, in the effort to stamp out and control the disease, difficulties arise which vary. These differences are principally topographical and racial. The methods and organisation which might be effective in thickly populated countries like India and Malaya, would be quite unsuitable to the widely separated communities of Africa, while the more backward the race the greater difficulty there is in persuading the victim to yield himself to treatment.

So much progress has been made in the investigation of leprosy in the different countries of the world during the past ten years, that it has become urgently necessary to co-operate leprosy teaching throughout the world. The old cry was "get rid of the leper", to-day the slogan is "get rid of leprosy".

It will be convenient to study the preliminary steps to be taken in a Campaign against Leprosy under three heads:—

1. The Problems.
2. The Theoretical solutions.
3. The actual ways and means.
1. **The Problems.**

The main difficulties which are found in India, for instance, may be taken as typical of most countries where leprosy is prevalent and presents a problem, are:

(1) Ignorance and fear on the part of the public, ignorance of the disease itself and fear as to the possible consequences of having contracted it. This is perhaps the most important difficulty to be overcome.

(2) Ignorance of many members of the medical profession regarding the early signs and symptoms of the disease, and the modern methods of treatment. Many doctors have had little opportunity of studying leprosy at college, while others have a prejudice regarding the curative powers of present day methods of treatment.

(3) Poverty of the people. The great majority of the people of India are exceedingly poor and can therefore afford but little in the way of medical treatment. It is also impossible for them to travel long distances for a treatment which may extend over a period of years.

(4) Lack of sufficient centres for treatment. This follows from 3, where the distances to be travelled, in a country the size of India, are often enormous.

(5)/
(5) Inadequate knowledge of the number and distribution of the lepers requiring treatment. Surveys made recently by doctors in different states in India show that there are numerous early cases which could only be detected by medical men with experience of leprosy; whereas it has been proved that in the various censuses taken in India, only those cases which could be detected by unskilled workers were enumerated, and those were the most advanced cases obvious to anyone.

2. The Theoretical Solutions.

These may be considered under the following headings:

A. (1) Education of the public.
   (2) Education of the medical profession.

B. Organised investigation as to the extent of the disease in affected areas.

C. Provision of ample facilities for free treatment.

A. Education of the public.

Until comparatively recent times leprosy was not considered a disease worthy of co-ordinated scientific investigation, but, with the advancement of our knowledge, old ideas of it are fast losing ground.

Now,
Now, in any scheme for combating leprosy, it is essential that the public should be educated to an intelligent understanding of the disease. This can be done in the two following ways:

a. By education of the public at home.

b. By education of the public in areas where leprosy is endemic.

a. The general public of the British Isles must be made passionately interested in this problem by intensive propaganda methods, - by radio, films, lectures, newspaper articles etc., etc. They must be made to feel the urgent need for cleansing the British Empire of so grave a disease, and must be made to shoulder some responsibility for its two millions, probably more, sufferers from leprosy.

Appeals for money must be made. There is always money to be had for a good cause. Money is desperately needed. What cause could be worthier or more urgent than this one?

The public at home must be made leprosy-conscious by every possible means.

b. Education of the public in areas where leprosy is endemic, as in India.

The question arises, what should the public be taught?
1. We must make it known that leprosy is no longer incurable. If diagnosed in the early stages and treated efficiently, leprosy can be cured.

Even when the disease has been present for some time, considerable improvement, and, in many instances, a cure is possible.

This knowledge would not only modify the ignorance so prevalent with regard to the disease, but would go a long way to eradicating the feelings of shame and fear which, hitherto, have proved such strong deterrents to early presentation for treatment.

2. It must be impressed upon the public, that it is not always those who are obviously leprous who are the greatest danger to the community. (In many of these cases the disease may have run its course, leaving them mutilated but not infectious. These are commonly termed 'burnt-out' cases).

The greatest risk of infection is found in the early and middle stage cases in whom the doctor can detect little or no evidence of leprosy.

3. The Indians generally, as well as the victims themselves, should be taught emphatically that, as regards infection, Leprosy is of two types, - infectious and non-infectious.

Firstly, the knowledge of this fact would to a great extent, hinder people from concealing possibly/
possibly infectious lepers in their homes. On the other hand, it might also prevent relatives from casting out one who showed signs of the disease, and who, on investigation, might be found to be suffering from the non-infectious type of leprosy.

Their understanding of the simple facts, and of the ways of obtaining relief, would guide them to the appropriate quarters, where diagnosis could be made and treatment instituted.

Secondly, this knowledge might weigh with employers (51) who are disinclined to employ lepers. The knowledge that the case was a non-infectious one would go far to allay his scruples.

How easy it is for a man, unemployed, ill-fed, suffering from a disease which brands him an outcast from society, gradually to sink until he reaches the beggar class! It is then that he becomes in all probability, an infectious case and a danger to others!

4. The housing problem must be tackled. Leprosy flourishes in a diseased atmosphere.

The people of India must be taught the elementary laws of cleanliness and sanitation. It cannot be sufficiently emphasised, that no real progress can be made in a campaign for the eradication of leprosy, until the fundamental conditions for public health have been improved.

Such/
Such measures would go far to hasten the elimination of co-existing diseases such as tuberculosis, dysentery and ankylostomiasis. These diseases, when associated with leprosy, tend to lower gravely the resistance of the patient. This has been my own experience in Malaya, again and again.

5. The people should be taught the importance of diet. Simple facts about the value of food stuffs in their own areas and the necessity of including certain articles in their diet, should be explained to them.

6. Lastly, and even of more vital importance than the foregoing, are the facts which should be taught concerning the children.

   a. Leprosy is not an hereditary disease.
   b. The Indian people must be impressed with the simple and compelling fact, that, if the children of infective leprous patients are early removed from them, the probability of these children acquiring leprosy is a remote one.
   c. The welfare of these children should be safeguarded as that of our own children has been at home during this war.
If steps were taken to improve the health and build up the resistance of all children in endemic areas, by diet, good housing conditions etc., what an enormous stride forward would have been taken in the stamping out of leprosy.

The tragedy of the children could thus be averted!

Propaganda must be so designed as to reach every caste and creed. This could be done by advice in clinics, radio talks, films, pamphlets, posters – simple, attractive and direct, with slogans such as,

'Health comes first!

'Health is yours for the asking!

'Seek health at the Clinic!

All these should be carried out in the appropriate language or dialects, and should be controlled by a propaganda headquarters.

A2. Education of the medical profession.

There is little use carrying on extensive propaganda if we have not a sufficient number of doctors capable of making a diagnosis, and of giving efficient treatment. Propaganda must be brought to bear on the medical profession at home. It is not surprising how little the ordinary practitioner knows about leprosy, since, as a medical student, he is not trained to diagnose and treat the disease. That is impossible in this country!
There is a vast field of enterprise for enthusiastic students. They should be taught:—

1. That leprosy is remediable.
2. That it is not a hideous and fearsome disease, but one full of interest and enormous possibilities for cure.
3. That it opens out for them a great vocation.
4. That the fundamentals for the eradication of leprosy (viz. the recognising of the early signs and symptoms of the disease) can only be taught at the source.

Therefore I would strongly advocate the founding of one or more yearly scholarships of value at each home University, which would enable enthusiastic graduates to live in comfort, and attend such famous centres as the School of Tropical Diseases, Calcutta. There, a thorough training would be provided in the latest methods of diagnosis and treatment. A year, thereafter, at one of the leper settlements would add much to their knowledge and experience.

In this way, an army of leprosy specialists would be built up. Some of these would eventually become research workers, some would have more interest in the clinical side, while others would take up administrative work.

It is my considered opinion that for the winning of such a scholarship six to twelve months' training in Public Health would be essential.
The training of Indian students at their own Universities should be most thorough in the recognition of the early signs and symptoms of leprosy. Their opportunities for seeing many and varied manifestations of the disease are numerous.

B. Organised investigation as to the extent of the disease in affected areas.

We require an accurate census of lepers. This is no easy matter. No ordinary census officer can recognise an early case of leprosy. Skilled observers usually make a count four to five times greater than those of an ordinary census. Unfortunately, owing to the war, survey parties are few and far between.

There is a great need for trained leprosy officers, and I feel strongly, that here is a great opportunity for employing greater numbers of Indian medical graduates in the work of detecting and enumerating lepers. This can only be done, so far as I can see, by raising the present inadequate salaries to an attractive level, and by making the posts pensionable.

These survey officers could do much in the way of propaganda among the people, and encourage them to attend regularly for free treatment at the out-patient clinics.

C./
C. Provision of ample facilities for free treatment.

Increased knowledge as to the nature of Leprosy during the past twenty years has changed our views, regarding the measures to be taken against it.

If we are to stamp this disease out, it is absolutely essential that every leper should be given the opportunity of free treatment. The majority of the people of India are so poor and so ignorant, that the treatment clinic must become an important and popular institution available to all, and within reach of the furthest village. Local treatment clinics must be multiplied many times.

3. The Actual ways and means.

The Past.

We have travelled far from the days when the medical profession was comfortably blinded to its own responsibilities in the problem of leprosy. To us, it was not first and foremost a medical problem. Segregation of all known lepers was looked upon as the only routine measure; and, since the disease was considered to be incurable, and as no remedy was known, little progress was made.

The care of the leper in India was left in the hands of Christian men and women, who were inspired by their own high purpose to minister to all spiritual and physical needs.
To these high-souled and unselfish pioneers we owe an immeasurable debt, for they did so much to awaken the social conscience of our people at home.

Ignorance of the disease, in its infectious and non-infectious manifestations, and the belief that cure was impossible, led, more often than not, to the segregation of non-infectious cases.

In the last century, and early part of this, acts have been passed in India for the compulsory segregation of beggar lepers, but the acts were not practicable and were seldom carried out to any great extent.

Compulsory segregation by law simply tends to encourage concealment, and so far has proved a failure.

The basis of voluntary segregation has been the one on which the greater part of leper work has been maintained; although, one must note, voluntary segregation has usually meant that the disease was well advanced, but not always infectious.

The Present.

Since 1924 magnificent help has been given by the British Empire Leprosy Relief Association, and more recently this help has been increased by the passing of the Colonial Development and Welfare Act. They have given money generously, for the relief/
relief of suffering in different parts of our Empire.

The outlook of the medical profession has now entirely changed. Realisation of the importance of certain fundamental requirements:

1. Early diagnosis.
2. Segregation of infectious cases only.
3. Separation of children from infective parents.
4. The importance of caring for the people's welfare

is influencing our present and future policy.

To-day, much is being done the world over to make the advanced case of leprosy an impossibility.

Let us hope that leprosy's frank manifestations - the horrible ulcerations and the hideous deformities will have utterly disappeared in a future generation!

Much can be learnt from the brilliant and earnest work now being carried on in other parts of the world.

The Fiji Government (52), has given an informative account of the Central Leper Hospital, Makokai. "The Fijian patients have increased from 352 in 1919 to 444 in 1943, but it is satisfactory to note that this is due to earlier admissions, for the number of early neural cases has increased from 0 to 27, and the advanced lepromatous ones have fallen from 32 to 0............."
The following facts are quoted (53) regarding the campaign against leprosy in Brazil. "The leper population of Brazil is approximately 45,000 (100/100,000 of the population). Approximately one third are in leprosariums, and one third are under medical care, but one third are unknown to health authorities. In the state of Rio de Janeiro 1,017 lepers are known; a large majority of these have the disease in contagious form. Effort is being directed toward rounding up all infective persons, and toward education of doctors in diagnosis, treatment and control of leprosy."

Sloan (54), discusses early leprosy as seen in Hawaii with respect to incidence, presenting symptoms and technique of diagnosis. Segregation reduced the number of known lepers from 1,175 in 1890 to 390 in June 1943. He states that leprosy occurs primarily in Hawaiian school children or young adults in lower living conditions, who have been in contact with known lepers or are members of their families. In most cases, the incubation period is probably 3-10 years. Not every case of leprosy can be diagnosed by a single examination, and every suspicious case should be reported to the local health officer and carefully followed.

I quote the British Medical Journal's report (55) on Muir's recent work in the Trinidad Leper Settlement.

"Good/"
"Good progress had been made since Trinidad had modified its stringent leper segregation act to allow early cases of a mild type to be treated in special out-patient clinics, in accordance with British Empire Leprosy Relief Association principles.

In India and Africa admission of lepers to the modern type of agricultural colonies was now regarded as a privilege; no compulsion was required. Conditions were much more difficult under the compulsory segregation system in Trinidad, but patient work had improved the atmosphere of that settlement."

To sum up, it can clearly be seen how widespread is the recognition by the medical profession to-day, that, for the successful treatment and control of leprosy, a specialised training is essential on the one hand, and, on the other, the co-operation and well being of the patient must be attained.

In India, the evolution of the type of refuge for the leper has been adapted to the progress made in our knowledge of the disease.

When the leper was regarded as an outcast from society the lazar house was provided. Later, a more Christian attitude was adopted, and leper homes came into being, where the unfortunate victims of the disease were given care and attention.

When it was recognised that leprosy would yield to treatment, leper hospitals were founded; from these/
these were evolved the leper colonies, which could provide work on the land and other occupational therapy, and where segregation could still take place.

These leper colonies are unfortunately too few in number, and cannot possibly fulfil the requirements of so vast a country as India.

Muir (56) in 1927 advocated the Propaganda-Treatment-Survey clinic, maintaining that the out-patient clinics were to be the chief means of tackling the leprosy problem in the future.

I agree wholeheartedly that by this means a greater number of early cases can be treated, and more lepers can be brought under control. In a country like India however, certain disadvantages appear.—

(a) Small out-patient clinics, widely separated, are very difficult to organise and run efficiently. There is at present no local central control.

(b) Many patients require special treatment e.g. operative treatment, X-Ray investigation, in the intervals between their attendance at the clinics. At present they cannot obtain this.

(c) The segregation of infective out-patient cases in their own homes is very rarely carried out efficiently, and no provision is made for these locally.
The Future:

Therefore, I consider that Muir's plan is insufficient. I firmly maintain that a strong and influential 'Central Institution for Leprosy' is needed, similar to the Malaria Institute of India, together with branches scattered throughout India, but concentrated more thickly in the great endemic areas of Bengal, Orissa, Bihar, and Madras. Each of these branches should have control over ten to twelve or more out-patient clinics, according to the density of the population, and should in itself be a 'Complete unit'.

I. The Central Institution for Leprosy would be a controlling body which would,

1. Administer all funds.
2. Control propaganda.
3. Select and appoint the necessary personnel for each complete unit.

II. My ideal Out-patient clinic.

A. The Purpose of the out-patient Clinic would be:-

1. To welcome within its doors every sufferer from leprosy in the district, and to gain the co-operation and confidence of each one.
2. To give free treatment to all leprous patients within the scope of an out-patient department.
3. To act as a receiving station for the 'Complete Unit', and advise, encourage and persuade/
persuade seriously ill and infective patients to enter the Complete Unit for investigation and treatment.

4. To keep full records of all patients treated, and to provide each patient with a small card bearing his name, clinic number, last day of treatment, and result of treatment already given, so that, if the patient goes to another clinic, his record card can be obtained.

5. To make regular house to house surveys in its own district and to keep statistical records.

6. To teach the simple facts of diet and hygiene.

B. The Personnel attached to the out-patient clinic.

1. A resident specialist practitioner – an Indian graduate who speaks and understands the local dialects.

2. An assistant trained in dispensing and in giving injections.

3. A highly trained nurse with two assistants, one who would assist with dressings etc. the other to go out in the district and act as a welfare worker.

C. Buildings of the ideal Out-patient clinic.

(a) Good accommodation for the staff.

(b) Two small male and female wards to take in three to four patients each for emergency treatment.
(c) Consulting room and two waiting rooms, one for males, the other for females (the reason for this is that 'purdah' is still adhered to in certain parts of India).

(d) A block containing laboratory, dispensary, office for records, and a small theatre for minor operations.

(e) Modern sanitary arrangements should be made, where practicable.

Each Out-patient clinic should be placed as near as possible to the centre of a group of villages.

I have observed that in India there has always been a tendency to concentrate on one scheme, whether for treatment or for control of leprosy. I feel very strongly, that it is only by a combination of schemes and methods of control that the attack on leprosy can be effectively carried out.

At present, patients come for treatment to out-patient clinics, and contacts are examined for signs of the disease. The villagers are taught some facts about leprosy, and are asked to isolate all infectious cases; but this is where the present system fails. The people are usually too poor, or have not sufficient and suitable accommodation to isolate an infectious case efficiently, or they do so for a short time only. It is often almost impossible to make/
make the villagers realise that a strong able-bodied and apparently healthy man can be a highly infectious case of leprosy, and a public danger!

To-day, segregation in homes, hospitals and settlements is possible only for a limited number of cases, on account of the lack of accommodation, and expense.

The greatest difficulty which I experience in the treatment of leprosy in India is to persuade poor patients to attend regularly for treatment. In the case of the rich private patient there is usually no trouble; he understands, when the nature of the disease is explained to him, that he has to be under treatment for a long period, perhaps years. But the poor patient is a problem, as he may be of the beggar class, and therefore seldom remains long in one district. The next doctor he consults has no record of his resistance nor of his previous treatment, and any treatment he may give, might possibly do more harm than good.

I am trying to institute a system of duplicate record cards in the states of Rajputana, one of which is to be kept permanently by the patient, so that if he migrates, the next doctor he consults will know what treatment he has received and the results of that treatment.

I maintain that the Out-Patient Clinics do not
in themselves provide the full answer to the control of Leprosy in India.

I have already suggested that there should be a Central Institute for Leprosy in India which would control branch institutions (hereafter referred to as Complete Units). These would resemble the Lady Willingdon Leper Settlement in South India, but on a smaller scale. I should like to see a great number of these Complete units scattered throughout India, situated mostly in those areas where leprosy is most prevalent, and each should control one dozen or more treatment clinics.

The Complete Unit should be situated if possible on high ground, free from malaria, in the centre of an area of arable land, of which one hundred acres or more could be acquired for the institution. There must be a good water supply, and the sanitary arrangements must be modern.

It must be easy of access for, and keep close in touch with its out-patient clinics.

These institutions should be so planned that the patients lead as normal a life as possible. They should be kept busy with suitable occupation and exercise to prevent home sickness and depression.

To ensure the segregation of the greatest number of infectious patients from the particular area it is essential to supply:—

1./
1. Good food.
2. Homely and happy surroundings.
4. Some prospect of recovery.
5. Above all, we must banish fear, and must gain the confidence and cooperation of the patient who will respond with loyalty and goodwill.

In this way I am confident that ninety per cent of patients, nay more, would become willing inmates.

The complete isolation of all infectious cases would, if it were possible, no doubt rapidly control leprosy in India.

The Purpose of the ideal Complete Unit is, therefore, to provide:

a. Segregation and treatment for all infective leprous patients.
b. Investigation and special treatment for any leper.
c. Accommodation and treatment for infected maternity cases.
d. Accommodation and treatment for children taken from infected surroundings.
e. Education of children, and a training centre for those patients who recover, and who may be of use in the future in the general campaign to control leprosy.
I find that patients respond more rapidly to treatment if they lead an active life, and therefore daily work should be provided according to the capacity of each one. Road-making, building, draining, carpentry work, handicrafts, fruit and vegetable growing, with time set aside daily for games, would all help the well-being of the patient.

At first, subsidies would be required from the government, but gradually the institution should be able to grow all the food required for the inmates. I am afraid that it could never hope to become completely self-supporting, since it is obvious that articles made by lepers can never be popular with the public.

The Complete Unit would have accommodation in accordance with the needs of the population of its area. Huts similar to those used in the district could quite easily be erected by ambulatory patients for their own use. They could, at low cost, also erect a school, workshop and other necessary out-buildings.

The central group of buildings should be well constructed and provide accommodation for the staff, male and female and children's wards for special treatments, laboratory, dispensary, surgical theatre and kitchens. It should be fully equipped with portable X-Ray apparatus etc., etc.
The Staff.—

The officer in charge should be a doctor with administrative experience. He could take charge of propaganda work, make yearly surveys of the area under his control and organise all occupational therapy.

In addition, there should be two male and two female assistant medical officers. All of these officers should have had special training in leprosy and should be able to speak the common language or dialect of the district.

One male officer would pay periodic visits to the out-patient clinics and be responsible for the admission of new patients; another would take charge of all treatment. One female doctor would look after the women, and the second be responsible for the welfare of the children.

There should be an adequate staff of nurses and compounders. (In India compounders are trained to dispense, give injections and anaesthetics, and act as male nurses). There should be a matron on the staff of each Complete Unit to organise the running of the hospital and the training of nurses etc. (these could be found amongst the more intelligent of the non-infectious patients).

The male doctors could train 'compounders' in the same category to be useful in laboratory, ward and/
and theatre.

It is most important that a nurse thoroughly trained in dietetics should be on the staff.

The Children.

As children have a very high susceptibility to the disease, they must be separated from infected parents at birth, and kept free from contact with infection.

The success of any campaign against leprosy, depends to a very great extent on the care taken to prevent children from contracting the disease; in fact, I would go as far as to say that it will be realised more, and still more, that the key to the whole problem of eradicating leprosy lies in three words, "Protect the Child".

The great majority of severe cases of leprosy are those who were infected in childhood.

Many children have to undergo treatment for a prolonged period, consequently every Complete Unit would have to be so arranged and equipped that it gave facilities for a happy home life, — plenty of fun and laughter and games for the little ones; a school for teaching handicrafts, hygiene, cooking and domestic training for the older ones, nourishing food in plenty for body and mind so that, when the time came, these children would go forth as messengers of/
of a healthier mode of life, because they would be equipped with "mens sana in sano corpore".

I have already described the preliminary treatment and routine treatments of leprosy and its complications. These are the treatments which I have found successful, and would carry out in my Plan of campaign.
SUMMARY AND CONCLUSIONS.

I have dealt with the theories concerning the transmission of leprosy, and have referred briefly to its history and distribution. I have then shown the distinguishing characteristics of the lepra bacillus and of the tubercle bacillus which closely resemble one another, and must be differentiated.

The pathological processes have been described, and the types of leprosy which arise from them.

The uncertainty as to the length of the "incubation period" has been pointed out. There is considerable evidence in favour of a period of two to three years, but authentic cases have been described in the literature by different writers with incubation periods of seven years and even of twenty-five years. The resulting danger arising from undiagnosed infectious cases of leprosy has been emphasised.

Attention has been drawn to the word 'macule' as used in leprology, which is entirely different in significance from its use by dermatologists.

The characteristic lesions of the neural type and of the lepromatous type of leprosy have been discussed in detail; and the need for skill and experience on the part of the physician, in accurately diagnosing early cases of the latter type, has/
has been considered to be of primary importance.

Various tests have been described which are of value as aids to diagnosis and prognosis. The importance of a series of estimations of the blood sedimentation rate as a guide to treatment is believed to be essential, and is also of importance in recording the patient's resistance.

The fact, that the Wassermann and Kahn standard reactions may be positive in advanced cases of leprosy not associated with syphilis, has been pointed out; and a brief reference has been made to the undoubted value of the Kline reaction in differentiating such cases.

It must be noted that prodromal symptoms of headache, malaise, pyrexia, may be present for weeks or months before clinical signs appear; the patient may not consult a doctor, and may go for a long period unrecognised as a case of leprosy.

The factors which stand in the way of early diagnosis have been carefully enumerated. Stress has been laid on the three important signs viz. -

1. Anaesthesia to light touch.
2. Thickening of nerves.
3./
3. The finding of lepraë bacilli. without any one of which no case should be diagnosed as leprosy.

The methods of examination for lepraë bacilli in skin and mucous membrane have been described in detail.

The differential diagnosis has been discussed under three headings,

1. Lesions of the skin resembling leprosy.
2. Lesions producing loss of cutaneous sensibility.
3. Lesions causing deformities and ulcers.

The factors influencing prognosis have been weighed with care, emphasis being laid on the poor resistance to leprosy of the tissues of children, and of all malnourished persons.

It is most important to realise that there is so far no specific drug against leprosy, — the ideal drug being one which satisfies the following points,

1. It must rapidly control and eliminate the disease.
2. It must be cheap.
3. It must cause little discomfort when given by injection.

An account has been given of the Chaulmoogral and Hydnocarpus group of drugs, and it has been shown that/
that some leprologists have complete confidence in their efficacy, while other workers consider their action to be without value.

A survey has been made of the treatment of leprosy as carried out by myself during the years 1924-1942, and it is my considered opinion that these oils are of immense value if a preliminary treatment of liver extract has been given beforehand to raise the patient's cellular defensive mechanism to the highest level, as I have found that otherwise these oils have a most depressing effect.

The present position of the sulphonamides, especially Promin, has been briefly referred to, and it is suggested that Penicillin may be the answer to the quest for an ideal drug against leprosy.

The disastrous results which frequently follow the administration of potassium iodide to leprous patients have been described.

It has been pointed out that in Malaya the severer form of leprosy is found most frequently in malnourished persons, especially those who exist on a diet of polished rice which is deficient in protein and Vitamin B1.
In planning a campaign against leprosy certain outstanding problems require a satisfactory solution. These problems have been discussed at length in the text, and a number of practical suggestions are put forward which are the result of my own experience in the treatment of leprosy since 1924.

1. An intensive propaganda campaign to be launched to raise funds, and to stimulate public and medical interest.

2. The whole control of leprosy in India to be in the hands of clinicians, research workers and administrators who have specialised in leprosy; and it is suggested that leprosy scholarships be endowed for this purpose, so that in time to come an ever increasing army of specialists be raised.

3. The people of India to be educated by every simple and direct means,
   a. as to the value and the proper cooking of the food the country provides, so that malnutrition may be eradicated,
   b. to co-operate in the voluntary segregation of all infectious cases of leprosy,
   c. to expect that children of infectious parents can be safeguarded by early separation.
d. to realise that leprosy is not an incurable disease, especially if treated in the early stages.

I feel most strongly that, by patient effort, the present devastating and prejudiced ignorance of the people of India could be turned into a powerful impetus for guarding the welfare of their own families, and thus promoting the health of future generations.

4. To have yearly surveys in each area undertaken by reliable observers.

5. Suggestions have been made that all future leprosy work in India should be under the control of a 'Central Institution', with branch institutes (which I have named Complete Units) each to control a number of out-patient clinics. In time, Complete Units with their subsidiary Out-Patient Clinics would spread like a network over the length and breadth of India, so that no district of the land would remain unserved, and no leper would remain unknown nor uncared for.

Truly do I believe that this Plan of mine is no mere daydream, but one that can help to provide a practical answer to the sorrows of this great country. Our purpose and our courage must remain steadfast – to obliterate the stain of leprosy for ever from the British Empire.
strong in will
To strive, to seek, to find, and not to yield.

Say not, the struggle nought availeth,
The labour and the wounds are vain,
The enemy faints not, nor faileth,
And as things have been, things remain.

For while the tired waves, vainly breaking,
Seem here no painful inch to gain,
Far back, through creeks and inlets making,
Comes silent, flooding in, the main.
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